PROSTATE CANCER IN CALIFORNIA

Special Report

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PROSTATE CANCER IN CALIFORNIA

Special Report

Editor:
Paul K. Mills, Ph.D.

June 2000

This publication was prepared by:
Public Health Institute
2001 Addison Street, 2nd Floor
Berkeley, CA 94704-1103
(510) 644-8200

Suggested citation: Mills PK, (ed). Prostate Cancer in California: A Special Report. Berkeley, CA: Public Health Institute, June 2000.

Design: Maggie Burgos, Public Health Institute

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ACKNOWLEDGMENTS

The preparation of a report such as this is made possible by the contributions of many people. First and foremost, the hospitals, cancer registrars and physicians throughout the state of California who identify and collect critical information on cancer patients provide the very basic data upon which rates of disease are calculated.

The staff of the regional registries throughout the state who collate and manage the data at the regional level are also of utmost importance in the operation of the statewide registry and the production of reports. The regional directors and the epidemiologists of the regional registries include Hoda Anton-Culver, Ph.D., Leslie Bernstein, Ph.D., Judy Boone, Jane Breuss, C.T.R., Deborah Bringman, M.P.H., Vincent Caggiano, M.D., Sharan L. Campleman, Ph.D., M.P.H., Becky Cassady, R.R.A., C.T.R., Christina Clarke, M.P.H., Wendy Cozen, D.O., Rosemary D. Cress, Dr.P.H., Cynthia Creech, C.T.R., Kathleen Davidson-Allen, C.T.R., Dennis Deapen, Dr.P.H., Mignon Dryden, C.T.R., Alan R. King, M.D., Tom Kurosaki, M.S., Lihua Liu, Ph.D., Paul K. Mills, Ph.D., John W. Morgan, Dr.P.H., Kiumarss Nasseri, D.V.M., Ph.D., Lilia O'Connor, M.B.A., C.T.R., Angela Prehn, Ph.D., Ronald Ross, M.D., Sidney Saltzstein, M.D., M.P.H., Tom Taylor, Ph.D., and Dee W. West, Ph.D.

Thanks also to the staff at the California Cancer Registry in Sacramento who perform invaluable tasks in the overall function of the statewide registry. Without their assistance this report would not be feasible. The staff include Cyllene R. Morris, D.V.M., Ph.D., Richard Cohen, M.S., Carin I. Perkins, M.S., Mark Allen, M.S., Sandy L. Kwong, M.P.H., Robert Schlag, M. Sc., and William E. Wright, Ph.D.

Funding for this report has been made possible in part through the National Program of Cancer Registries at the Center for Disease Control and Prevention (Grant No. U75-CCU010677).

OVERVIEW

One in five American men will be diagnosed with prostate cancer at some point in his lifetime. Since prostate cancer, more than any other form of cancer, is a disease of aging, it will continue to be a source of morbidity and mortality among American men for many years to come. Despite its importance as a public health issue, little is known about the cause of this disease except its tendency to run in families, to afflict black men more than men of other racial groups and, possibly, to be associated with the primary male sex steroid hormone, testosterone.

In this monograph, several aspects of prostate cancer epidemiology and treatment in California are discussed. The descriptive epidemiology of the disease (i.e. how it is distributed in the population by characteristics of person, place, and time) is discussed, as is the analytic epidemiology concerning known or suspected risk and/or protective factors for the disease. In addition, survival data are presented, along with recommendations for screening. The purpose of the report, however, is to focus our thinking on future efforts on the primary prevention of this disease which will hopefully lessen the terrible impact it is currently taking on the health of the American male.

Mills and Cohen describe the dramatic variation in prostate cancer by age, race/ethnicity, and calendar time in Chapter 1. More than any other cancer, prostate cancer shows a dramatic relationship with age in that rates rise exponentially after the age of 40 and are 80 times higher in males 85+ years of age than in men 40-45 years of age. Blacks are shown to experience the highest age-adjusted incidence of any race/ethnicity group and experience incidence rates twice as high as Asian men. One of the most salient aspects of prostate cancer was the rapid rise in prostate cancer incidence rates between 1988 and 1992 followed by a decline in rates between 1993 and 1995. Incidence rates rose 70 percent during this time period, and the decline most likely reflects an exhaustion of the pool of susceptibles. This pattern is undoubtedly associated with the introduction of screening tests for prostate cancer in the late 1980s, particularly screening with the Prostate-Specific Antigen screening test (PSA).

The dramatic increase and decrease in prostate cancer incidence rates between 1988 and 1995 has not been observed with prostate cancer mortality rates, however. In Chapter 2, Nasseri presents data on mortality rates from prostate cancer in California between 1970 and 1996. Mortality rates increased between 1970 and 1991 by an estimated 0.82 percent per year in California and since have declined by an annual rate of 3.42 percent.

In addition to age, race/ethnicity, and calendar time, there are other risk factors for prostate cancer that are discussed by Cozen and Liu in Chapter 3. Like most cancers, prostate cancer is multifactorial in etiology, and a combination of family history, diet and specific genetic factors appears to be implicated for this cancer. Having one first degree relative with prostate cancer increases a man's risk for the disease two to three times, while having two or more relatives with prostate cancer increases risk further. In general, the genetic form of the disease appears at younger ages and may be responsible for approximately 9 percent of all prostate cancer. Diets high in fat content may be associated with elevated risk of prostate cancer because high fat diets appear to increase levels of testosterone, while low fat diets tend to reduce levels. Testosterone, the primary male sex hormone, regulates prostate growth and may play a role in the development of prostate cancer; especially when it is converted to dihydrotestosterone by the enzyme 5-alpha-reductase.

In California there is considerable geographic variation in prostate cancer incidence and mortality, and this variation is discussed by Cress and Morgan in Chapter 4. The inter-county variation, they note, may be as strongly related to screening and access to

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health care issues as it is to true biological differences in risk. In support of this, they note that at the county level, overall risk of being diagnosed with prostate cancer was not related to risk of dying from prostate cancer. Geographic variation in incidence of prostate cancer in California can be attributed largely to difference in screening practices rather than variation in prevalence of risk factors for prostate cancer.

Since data on survival with prostate cancer are not available on a statewide basis, Prehn, Lin, and Darbinian restrict their attention to survival data from the San Francisco Bay Area for the years 1973 to 1994. Overall, the prostate cancer five-year relative survival improved from 71.2 percent in 1973 to 93.5 percent in 1990. This remarkable improvement, however, was not experienced by men of different ages, races or with different stage and grade of disease at initial diagnosis. Men with distant disease or poorly differentiated disease continue to have a poor prognosis. The greatest improvements in survival have affected men with regional disease at diagnosis. In addition, black men and Filipino men are less likely to survive their disease than men of other race/ethnicity regardless of stage or grade of disease at diagnosis.

It is likely that the rapid increase in the number of prostate cancers diagnosed in California (and elsewhere) in the early 1990s was due to the introduction and widespread adoption of screening for this disease using the PSA screening test. In Chapter 6 Morgan,Vang, and Wong describe the recommendations of four national organizations (American Urological Association, American Cancer Society (ACS), U.S. Preventive Services Task Force, and the National Cancer Institute) and correlate these recommendations with several outcome measures of prostate cancer morbidity and mortality. When the financial cost of following the recommendations of at least one of these agencies is considered, the continued use of the PSA test must be balanced against expected benefits (e.g. ACS recommendation of annual screening of men between 50 and 75 years would cost the U.S. $12.7 billion annually). An assessment of the impact of screening on mortality rates must be made as well as the risk attendant to not screening. The risk of waiting may lead to disease progression and hence poorer prognosis.

In Chapter 7 Morris presents data on cumulative and interval risk of being diagnosed with prostate cancer. As with incidence and mortality, black men continue to experience the highest lifetime risk (18.1 percent), as well as the highest interval risk regardless of current age. Even at the relatively young age of 50 years, black men have a 10.4 percent risk of being diagnosed with prostate cancer in the next 20 years, whereas for white men this risk is 7.2 percent.

The surgical treatment of 34,756 men in California diagnosed with prostate cancer in 1994 and 1995 is discussed by Kwong in Chapter 8. Prostatectomy was performed most frequently (32.7 percent) while transurethral resection was performed less frequently (11.9 percent). Surgery was performed most frequently on men with regional disease at diagnosis although more than half of all men did not receive surgery as their first course of treatment. Black males were less likely to receive surgery of any type.

Stage of disease at diagnosis is a major prognostic indicator, and in Chapter 9 Boyer-Chammard, Taylor, and Anton-Culver describe patterns of stage at diagnosis by age, race/ethnicity, geographic residence and year of diagnosis. Between 1988 and 1995, 59.5 percent of California men with prostate cancer were diagnosed at the local stage, 16.2 percent with regional disease and 9.7 percent with distant disease. The remainder was of unknown stage or unknown race/ethnicity. Rates of local disease decreased with increasing age and whites had the highest proportion of local disease (61.2 percent).

Blacks had the lowest proportion of local disease (54.9 percent). Rates of local disease tended to be lowest in the more rural areas, in particular in the Central Valley and in Northern California.

Social class, a measure of affluence and access to health care services, is also of interest in prostate cancer epidemiology. Liu and Cozen in Chapter 10 show marked variation in incidence rates of prostate cancer in Los Angeles County by social class, especially for the more recent time period during the "PSA era." During the years 1988-1995, the incidence of prostate cancer was highest in the highest social classes of Los Angeles County, regardless of race/ethnicity. This disparity was not evident in the "pre-PSA era" and undoubtedly reflects differences in screening among the different social classes in Los Angeles County. This gradient was most pronounced for local disease.

PAUL K. MILLS , PH.D.

The Incidence of Prostate Cancer in California, 1988-1995

Paul K. Mills, Ph.D.[1] and Richard Cohen, M.S.[2]

[1] Cancer Registry of Central California, Fresno, CA
[2] Human Population Laboratory, Berkeley, CA

INTRODUCTION

In 1995, approximately 17,000 men in California were diagnosed with prostate cancer.[1] This makes prostate cancer the most frequently diagnosed invasive cancer among men in California and accounts for more than a quarter of all newly-diagnosed cancers among males in the state. Although incidence rates are now leveling off after experiencing a peak in 1992, the disease will continue to be a major public health burden since prostate cancer, perhaps more than any other human cancer, is closely related to aging. The median age at diagnosis is 71 years;[2] and as the population continues to age, the absolute numbers of prostate cancers diagnosed among men will increase.

In this chapter the descriptive epidemiology of prostate cancer is presented, including data on the incidence of the disease by calendar time, age, and race/ethnicity. These descriptive features of prostate cancer can provide clues regarding the biology and etiology of this burdensome disease.

RACE/ETHNICITY

There is considerable racial variation in the incidence of prostate cancer in California and elsewhere. Black men experience by far the highest age-adjusted incidence rates of the disease. In 1995 black men in California had an incidence rate of 203.7/100,000 population. This was about twice as high as the rate experienced by Hispanic and white, non-Hispanic men in the same year (99.0 and 116.8/100,000 respectively) and nearly three times higher than the rate experienced by Asian/Pacific Islanders (68.8/100,000) (Table I-1 and Figure I-1).

The high risk of prostate cancer seen in black men in California is not unique to the state. Data from the Surveillance, Epidemiology, and End Results (SEER) program of the National Cancer Institute, for example, show that black men throughout the United States experience the highest incidence rates of any racial ethnic group.[3] Between 1991 and 1995 the incidence rates of prostate cancer in black men in the U.S. was 241.2/100,000. These elevated rates in blacks are not an artifact produced by differences in detection but rather suggest a real increase in risk. Interestingly, rates of prostate cancer among black men in Africa are not elevated. In Zimbabwe, for example, between 1990 and 1992 the age-standardized prostate cancer incidence rate was only 29.2/100,000 compared to 127.6/100,000 among black men in the San Francisco Bay Area during the same time

Table I-1: Invasive Prostate Cancer Incidence Counts and Age-Adjusted Rates[1] Per 100,000 Population by Year of Diagnosis and Race/Ethnicity[2], California, 1988-1995

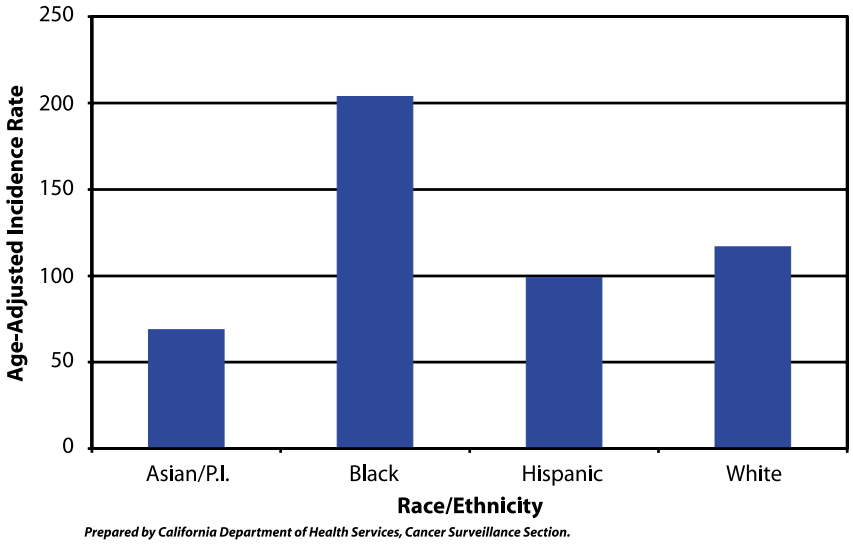
	All Races		Asian/P.I.		Black		Hispanic		White	
Year of Diagnosis	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate
1988	12,123	101.6	356	50.0	876	148.7	861	72.0	9,856	105.3
1989	13,005	106.5	398	52.0	917	153.7	1,004	79.7	10,499	110.4
1990	15,499	121.9	465	55.4	1,087	174.9	1,161	86.0	12,486	127.3
1991	19,969	152.0	585	63.0	1,327	208.4	1,437	99.3	16,163	161.4
1992	23,206	172.2	800	79.7	1,604	242.0	1,828	117.3	18,185	179.3
1993	21,772	159.3	916	85.4	1,714	252.0	2,018	120.6	15,864	156.8
1994	18,533	134.3	880	77.3	1,567	225.7	1,928	108.1	12,982	129.3
1995	16,993	121.5	825	68.8	1,446	203.7	1,863	99.0	11,712	116.8

[1] Rates are age-adjusted to the 1970 U.S. population. Data are from the California Cancer Registry (01/98).
[2] Race/ethnicity groups are mutually exclusive. Persons of Hispanic ethnicity are identified by medical record and/or surname, and may be of any race. Men of unknown race/ethnicity are excluded from race-specific data.

Prepared by California Department of Health Services, Cancer Surveillance Section.

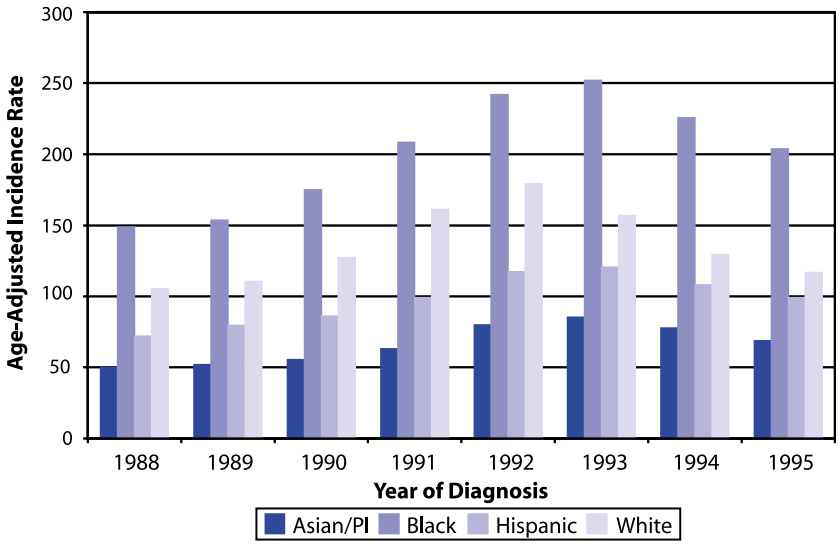
RACE/ETHNICITY

Figure I–1: Age-Adjusted Incidence Rate Per 100,000 Population of Invasive Prostate Cancer by Race/Ethnicity, California, 1995



Prepared by California Department of Health Services, Cancer Surveillance Section.

Figure I–2: Age-Adjusted Incidence Rate Per 100,000 Population of Invasive Prostate Cancer by Race/Ethnicity and Year of Diagnosis, California, 1988-1995



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RACE/ETHNICITY

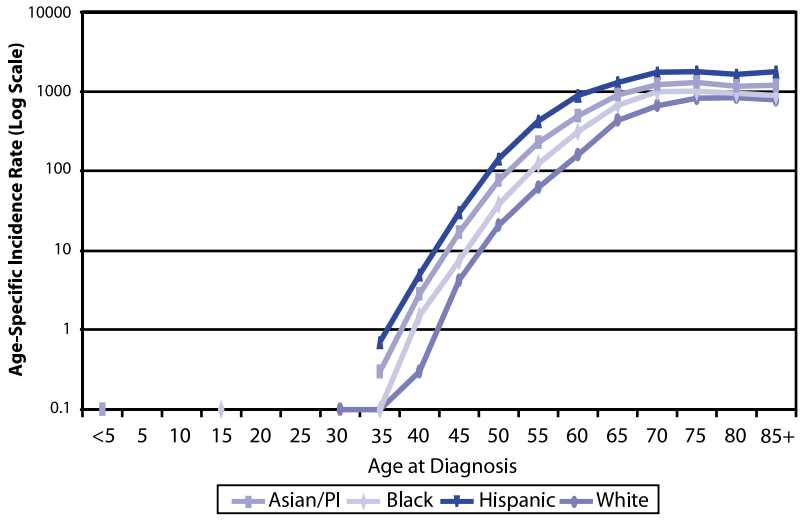
period.[4] This suggests that some environmental factor(s) unique to the U.S. accounting for the high rates. Although the exact mechanism explaining this higher risk is unknown, a complex interrelationship between diet (probably dietary fat), steroid hormones (testosterone and its metabolites) and genetic factors may contribute to the elevated risk in black men.[4]

CALENDAR TIME

Between the inception of statewide reporting of cancer in California in 1988 and the early 1990s, the incidence rate of prostate cancer increased dramatically. In all four race/ethnicity subgroups, there was a 70 percent increase in the age-adjusted incidence rate of prostate cancer in that time period (Figure I-2). Since 1992, the incidence rates have fallen, and by 1995, the rate in the white, non-Hispanics was 116.8/100,000 which was only about 11 percent higher than in 1988. For the other race/ethnicity subgroups, (including Hispanic, Black and Asian/other), however, rates in 1995 were still about 37 percent higher than they were in 1988. This may reflect the point at which incidence rates peaked in the various race/ethnicity subgroups. In whites, this was in 1992, while in the other race groups, incidence rates peaked about a year later.

This pattern of a dramatic increase in incidence followed by a gradual decline is consistent with the introduction of a new screening test for the disease and has been observed in other population-based cancer registries throughout the U.S.[6] Prostate cancer is detectable via transrectal ultrasonography (TRUS) which was introduced in the mid-1980s and which is thought to be responsible for the initial rise in prostate cancer incidence. Prostate cancer is also detectable by use of a serum marker called prostate-specific antigen (PSA). PSA is a prostate-associated glycoprotein that is shed into the bloodstream and is detected by immunologic techniques. It was introduced in the U.S. in the late-1980s and contributed to the continued rise in incidence rates. It is likely that the PSA screening associated epidemic of prostate cancer has passed and that incidence rates will return to a more gradual rise with calendar time reflecting a true biological increase of the disease in the population.

Figure I–3: Average Annual Age-Specific Incidence Rate Per 100,000 Population of Invasive Prostate Cancer by Age at Diagnosis and Race/Ethnicity, California, 1991-1995



Prepared by California Department of Health Services, Cancer Surveillance Section.

Table I-2: Five-year Prostate Cancer Incidence and Average Annual Age-Specific and Crude Rates[1] Per 100,000 Population by Age at Diagnosis and Race/Ethnicity[2], California, 1991-1995

	All Races		Asian/P.I		Black		Hispanic		White	
Age at Diagnosis	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate
00-04	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
05-09	1	0.0	0	0.0	0	0.0	0	0.0	1	0.0
15-19	3	0.1	0	0.0	0	0.0	2	0.1	1	0.0
20-24	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
25-29	1	0.0	0	0.0	0	0.0	1	0.0	0	0.0
30-34	3	0.0	1	0.1	0	0.0	0	0.0	1	0.0
35-39	18	0.3	1	0.1	3	0.7	2	0.1	12	0.3
40-44	144	2.4	2	0.3	19	5.0	20	1.5	100	2.8
45-49	700	15.0	19	4.2	84	30.2	67	7.4	512	17.0
50-54	2,508	70.1	70	20.6	318	142.1	246	38.4	1,780	75.7
55-59	6,108	211.2	165	61.8	803	430.8	612	123.8	4,344	225.5
60-64	12,326	474.0	350	159.0	1,292	889.9	1,262	309.3	9,041	498.9
65-69	21,065	893.3	839	441.0	1,595	1311.0	2,189	682.9	15,659	913.6
70-74	24,271	1236.0	991	662.1	1,611	1738.0	2,151	991.7	18,421	1231.0
75-79	17,700	1337.0	781	814.4	1,033	1798.0	1,256	1017.0	13,441	1290.0
80-84	9,631	1229.0	489	839.2	543	1641.0	730	955.6	7,136	1165.0
85+	5,992	1225.0	298	786.8	357	1787.0	536	902.0	4,455	1206.0
All Ages/ Crude	100,473	128.0	4,006	52.4	7,658	141.0	9,074	40.5	74,906	175.0

[1] Data are from the California Cancer Registry (01/98).
[2] Race/ethnicity groups are mutually exclusive. Persons of Hispanic ethnicity are identified by medical record and/or surname, and may be of any race. Men of unknown race/ethnicity are excluded from race-specific data.

Prepared by California Department of Health Services, Cancer Surveillance Section.

Table I-3: Invasive Prostate Cancer Incidence Counts and Age-Adjusted Rates[1] Per 100,000 Population by Year and Age at Diagnosis, California, 1988-1995

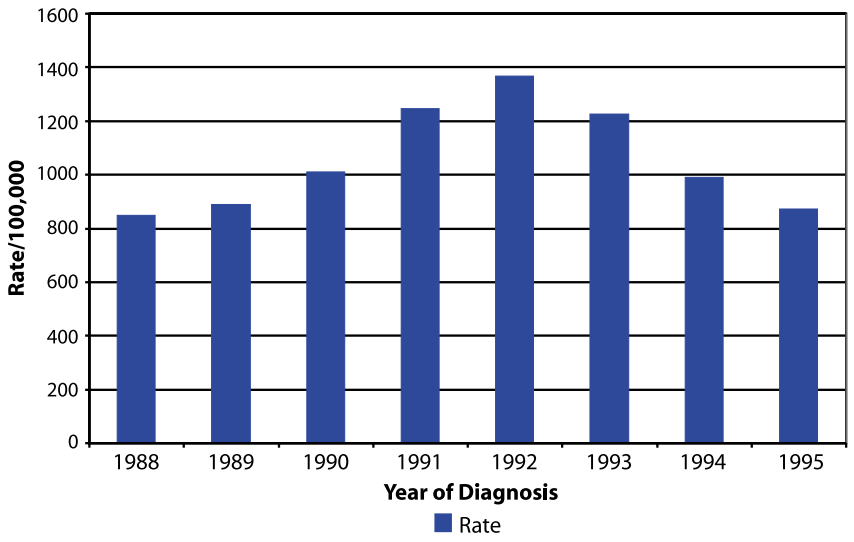
	Age Under 65		Age 65 and Over	
Year of Diagnosis	Cases	Annual Rate	Cases	Annual Rate
1988	2,067	19.7	10,056	848.9
1989	2,172	20.6	10,833	890.4
1990	2,630	24.5	12,869	1011.0
1991	3,499	32.2	16,470	1246.0
1992	4,539	41.2	18,667	1367.0
1993	4,719	42.5	17,053	1226.0
1994	4,579	40.7	13,954	989.0
1995	4,478	39.2	12,515	872.8

[1] Data are from the California Cancer Registry (01/98).

Prepared by California Department of Health Services, Cancer Surveillance Section.

AGE

Figure I–4: Age-Adjusted Prostate Cancer Incidence Rates In Men Greater Than 65 Years, California, 1988-1995,

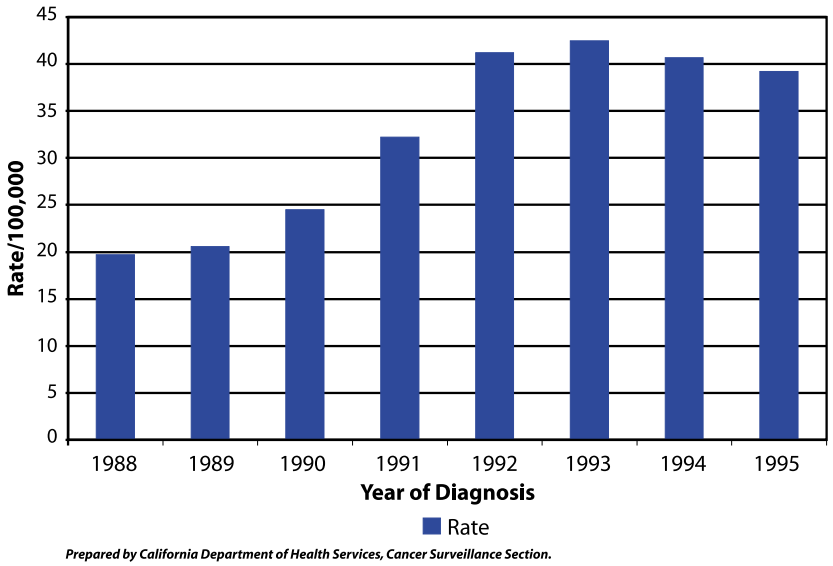


The incidence of prostate cancer is a direct function of age. The disease is almost non-existent before the age of 40, and after that the incidence rate rises exponentially with age (Figure I-3). The slope of the age-incidence curve is steeper for prostate cancer than it is for any other form of human cancer. Rates in men 80-85 years of age, for example, are 80 times higher than in men 40-45 years of age. This pattern is apparent in all four race/ethnicity subgroups (Table I-2). The preponderance of prostate cancer in black men is apparent in every age group. Even among the youngest age groups the incidence rates are twice as high in the blacks as in the other race/ethnicity subgroups (e.g. 30/100,000 in black men 40-45 years of age versus 17/100,000 in non-Hispanic whites of the same age).

Almost three-quarters of all prostate cancer diagnosed in California in 1995 occurred in men 65 years of age or older (Table I-3). However, incidence rates in those younger than 65 increased by 215 percent between 1988 and 1993 while rates in older men (greater than 65) increased 61 percent in the same time period (Figures I-4 and I-5). This suggests a differential impact of screening on the population. Among men less than 65, the age-adjusted incidence rate of prostate cancer was 99 percent higher in 1995 than it was in 1988. However, in older men, the incidence rate in 1995 was only 3 percent greater than it was in 1988.

AGE

Figure I–5: Age-Adjusted Prostate Cancer Incidence Rate In Men Less Than 65, California, 1988-1995



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With the exception of the childhood cancers, all major forms of human cancer of epithelial origin (i.e., breast, lung, prostate, and colon) show strong organ-specific relationships with age. This is particularly so for prostate gland cancer where incidence rates are in excess of 1,000 per 100,000 population per year in the elderly while the disease is rare in younger aged men. This pattern may be explained by cumulative, life-long exposure to some commonly occurring carcinogen(s) which requires a prolonged period between initial exposure and disease occurrence.

Currently, only 12 percent of the U.S. population is age 65 years or older. However, as the population continues to age, this proportion will steadily increase. Given the age-dependence of prostate cancer, the absolute numbers of prostate cancers diagnosed in American men also will increase even as therapeutic advances continue.

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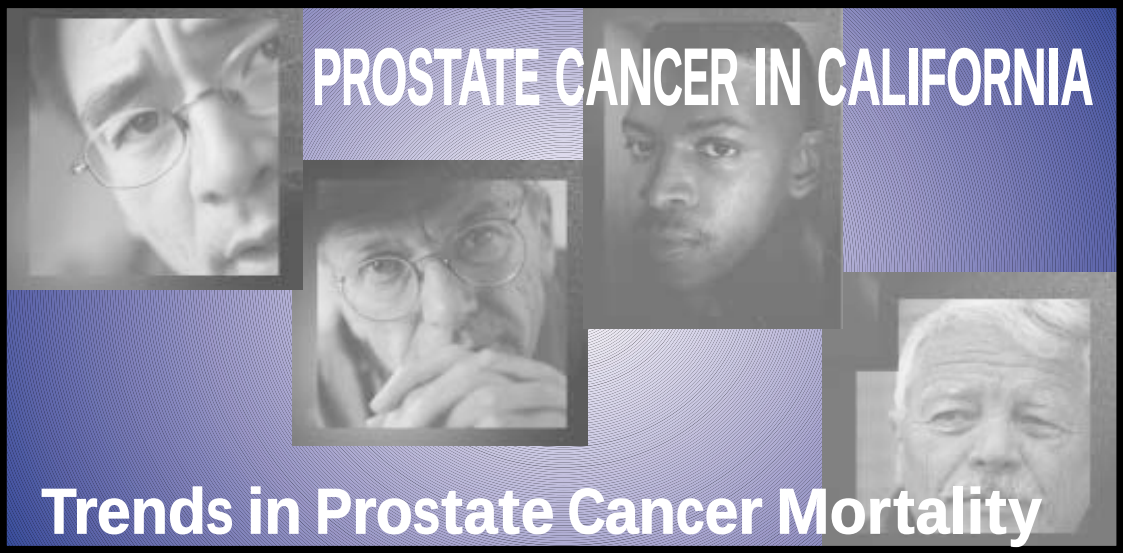


Trends in Prostate Cancer Mortality in California, 1970-1996

Kiumarss Nasseri, D.V.M., M.P.H., Ph.D.[1]

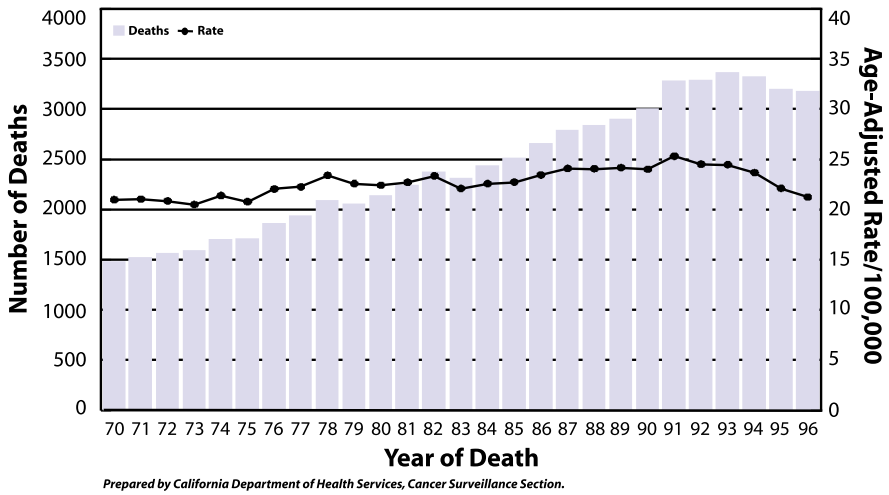
[1] Tri-Counties Cancer Registry, Santa Barbara, CA

INTRODUCTION

Cancer is the second leading cause of death in California. Among California men, prostate cancer is the second most common cause of cancer deaths, accounting for 12.1 percent of cancer deaths.[1] Nonetheless, compared to many other sites, cancer of the prostate gland often progresses slowly, and survival is relatively high. According to recent data, five-year relative survival from prostate cancer is 93 percent.[2] Although relative survival continues to decline at ten, fifteen, and twenty years after diagnosis, only one-third of men who die after being diagnosed with prostate cancer actually die of the disease itself. This chapter demonstrates that despite the surge in prostate cancer diagnoses in the early 1990s, prostate cancer mortality has not increased, and, in fact, has decreased significantly since 1991. Although this temporal pattern may reflect improvement in survival due to early detection and the diagnosis of some clinically insignificant cancers, it may also reflect changes in the demographics of the population and the wider use of radiation therapy and prostatectomy to treat this disease.[3]

This chapter evaluates characteristics of 65,238 deaths from prostate cancer recorded in California from 1970 to 1996. This information is based on data abstracted from death certificates by the California Department of Health Services' Office of Vital Records and Statistics. The California Cancer Registry (CCR) began statewide data collection in 1988; and long-term data on prostate cancer incidence is not available for California except for the San Francisco Bay Area and Los Angeles County where incidence data collection began in 1973 and 1972, respectively. In both areas, the incidence and mortality patterns for prostate cancer are very similar to national data collected by the Surveillance, Epidemiology, and End Results (SEER) program of the National Cancer Institute that has monitored cancer incidence and mortality in a 10 percent representative sample of the US population since 1973. Data from SEER program show that incidence rates peaked in 1992 and mortality rates peaked in 1991.[2] Between 1973 and the peak years incidence rates increased 177.5 percent and mortality rates increased only 18.2 percent.

Figure II–1: Prostate Cancer Deaths and Age-Adjusted Mortality Rate, All Races Combined, California, 1970-1996



Prepared by California Department of Health Services, Cancer Surveillance Section.

Table II-1: Number of Deaths and Age-Adjusted Prostate Cancer Mortality Rates, California, 1970-1996

	Race/Ethnicity									
	All Races Combined		Non-Hispanic White		Non-Hispanic Black		Hispanic		Asian/Other	
Year	Deaths	Rate	Deaths	Rate	Deaths	Rate	Deaths	Rate	Deaths	Rate
1970	1475	21.0	1289	21.3	99	37.0	67	16.0	20	7.7
1971	1519	21.0	1319	21.2	98	34.3	83	18.8	19	7.0
1972	1560	21.0	1333	20.8	131	44.8	79	16.8	17	6.7
1973	1586	20.5	1365	21.0	133	40.4	75	15.3	13	4.0
1974	1697	21.4	1452	21.4	123	37.4	96	18.0	26	8.0
1975	1706	21.0	1470	21.1	141	40.2	73	13.0	22	7.0
1976	1858	22.0	1572	22.0	149	40.0	106	18.0	31	9.1
1977	1935	22.2	1638	22.3	159	41.0	104	17.0	34	9.2
1978	2086	23.3	1733	23.2	198	50.0	122	18.4	33	9.0
1979	2054	23.0	1727	22.8	201	50.0	93	13.2	33	9.0
1980	2135	22.3	1775	22.5	203	46.3	125	16.3	32	7.7
1981	2236	23.0	1859	22.9	220	48.0	119	15.0	38	9.0
1982	2368	23.3	1974	23.7	206	43.1	137	16.0	51	10.7
1983	2311	22.0	1922	22.5	208	42.4	132	14.4	49	10.1
1984	2434	23.0	1935	22.1	284	54.5	152	16.0	63	11.4
1985	2508	23.0	2053	23.0	244	46.8	141	14.0	68	12.0
1986	2652	23.4	2145	24.0	273	50.3	159	15.2	74	12.0
1987	2785	24.1	2245	24.3	261	47.6	199	18.2	77	11.3
1988	2833	24.0	2266	24.2	288	51.4	197	17.2	82	12.0
1989	2894	24.2	2349	24.9	279	49.5	166	14.0	97	13.0
1990	2999	24.0	2395	24.5	311	53.2	193	15.2	99	12.0
1991	3275	25.3	2587	26.0	330	54.0	249	18.3	108	12.0
1992	3286	24.5	2622	25.4	309	50.0	241	16.5	113	11.0
1993	3360	24.4	2646	25.3	333	53.0	250	16.1	131	12.0
1994	3321	24.0	2624	25.0	328	50.3	271	16.4	98	8.3
1995	3191	22.0	2466	23.0	333	51.0	267	15.4	123	10.0
1996	3170	21.2	2471	22.4	320	47.0	256	13.4	122	9.0

Rates are adjusted to the 1970 U.S. Standard population and presented per 100,000 population.
Source: California Department of Health Services, Death Certificate Master Files, 1970-1996

Prepared by California Department of Health Services, Cancer Surveillance Section.

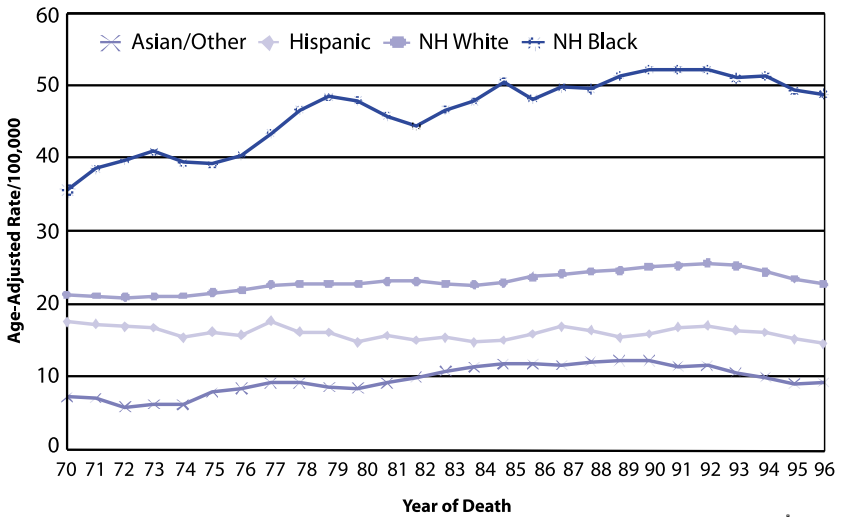
Time & Race/Ethnicity

Table II-1 presents the total number of prostate cancer deaths by race/ethnicity and the annual age-adjusted rates for each year from 1970 to 1996. The number of deaths increased steadily from 1,475 in 1970 to a peak of 3,360 in 1993 and then decreased to 3,170 in 1996. The peak mortality rate of 25.3 per 100,000, however, occurred in 1991.

Figure II-1 presents the long-term trends in the number of deaths and the age-adjusted mortality rates from prostate cancer for all races combined. A decreasing trend is clearly visible, after a peak in 1991, for the mortality rates. Trends of prostate cancer mortality rates in individual race groups are presented in Figure II-2. The magnitude of rate differentials by race/ethnicity is remarkable. The mortality rate is highest in non-Hispanic

TRENDS IN CALIFORNIA

Figure II–2: Prostate Cancer Mortality, by Race/Ethnicity and Year,* California, 1970-1996



*Three Year Moving Average

Prepared by California Department of Health Services, Cancer Surveillance Section.

black and lowest in Asian/other groups. The black/white rate ratio of 1.72:1 in 1970 has increased to 2.1:1 in 1996. The gap between Hispanics and non-Hispanic whites is widening and has changed from a Hispanic/white rate ratio of 0.75:1 in 1970 to 0.60:1 in 1996. Trends in all race groups follow a similar pattern of a gradual increase up to 1991 and a sustained decrease thereafter, except for Hispanics whose rate shows a decreasing pattern from 1970-1996. It is worth noting that Hispanic ethnicity in this mortality report is based solely on identification by surname and is uniform for all years in this study. (Note: Number of deaths and age-adjusted mortality rates reported for Hispanics in this study are about 10 percent less than similar statistics reported by CCR[1] which use other Hispanic identifiers in addition to the surname. This discrepancy is entirely limited to the distinction between Hispanics and non-Hispanic whites; and because of the large number of deaths in the latter group, it has no appreciable impact on rates for non-Hispanic whites).

Table II-2 shows the percentage of the absolute change as well as the estimated annual percent change (EAPC) for prostate cancer mortality by race/ethnicity and time period in California and for all races combined in the US. The EAPC for non-Hispanic blacks is about twice that of the non-Hispanic whites between 1970 and 1991, while the EAPC for Asian/others is twice that of the non-Hispanic blacks. The prostate cancer mortality rate in Hispanics has practically remained level during this period. Changes in the age-adjusted rates after 1991 are remarkable. Rates for non-Hispanic whites and Hispanics have been dropping by the significant rate of 3 and 5 percent, respectively, per year since 1991. Rates for non-Hispanic blacks and Asian/other groups are also dropping but are not statistically significant.

AGE

Table II-3 presents the total number of prostate cancer deaths and the truncated age-adjusted rates for two major age categories partitioned at age 65. This table reveals

Table II-2: Absolute and Estimated Annual Percent Change (EAPC) in Prostate Cancer Mortality Rate by Race/Ethnicity and Period, California, 1970-1996

	1970-1991			1991-1996		
Race	Absolute	EAPC	p-value	Absolute	EAPC	p-value
All Races Combined	18.6	0.82	0.000	-13.0	-3.42	0.002
Non-Hispanic White	20.5	0.87	0.000	-11.6	-2.93	0.007
Non-Hispanic Black	42.3	1.68	0.000	-5.7	-1.92	0.096
Hispanic	7.8	-0.22	0.569	-17.5	-4.95	0.010
Asian/Other	51.7	3.65	0.000	-19.2	-6.14	0.071
SEER, All Races, 1973-95	18.2	1.07	0.000	-0.97	-1.65	0.028

p-value indicates the significance of the difference between EAPC and zero (0).

Prepared by California Department of Health Services, Cancer Surveillance Section.

Table II-3: Number of Deaths Under Age 65 and Truncated Age-Adjusted Prostate Cancer Mortality Rates, California, 1970-1996

Under age 65	Race/Ethnicity									
	All Races Combined		Non-Hispanic Black		Non-Hispanic White		Hispanic		Asian/Other	
Year	Deaths	Rate	Deaths	Rate	Deaths	Rate	Deaths	Rate	Deaths	Rate
1970	193	2.5	21	5.3	160	2.4	9	2.0	3	1.1
1971	176	2.2	14	3.3	149	2.2	11	2.1	2	1,0
1972	192	2.4	21	5.0	159	2.4	10	2.0	2	1.0
1973	219	3.0	43	10.0	169	2.5	6	1.0	1	0.3
1974	207	2.5	22	5.0	170	2.4	13	2.0	2	1.0
1975	226	3.0	31	6.3	177	3.0	15	2.2	3	1.0
1976	237	3.0	36	8.0	184	3.0	16	2.0	1	0.2
1977	232	3.0	35	7.0	180	2.4	13	2.0	4	1.0
1978	250	3.0	33	6.3	200	3.0	13	1.4	4	1.0
1979	228	2.3	31	6.0	181	2.3	16	2.0	0	0.00
1980	274	3.0	37	7.0	213	3.0	21	2.2	3	1.0
1981	289	3.0	35	6.1	234	3.0	20	2.0	0	0.00
1982	273	3.0	36	6.2	215	3.0	17	1.4	5	1.0
1983	269	3.0	32	5.4	213	3.0	23	2.0	1	0.1
1984	324	3.1	64	11.0	224	3.0	31	2.4	5	1.0
1985	289	3.0	45	8.0	216	3.0	24	2.0	4	1.0
1986	273	3.0	38	6.3	202	3.0	24	2.0	9	1.2
1987	286	3.0	43	7.2	212	3.0	26	2.0	4	1.0
1988	290	3.0	46	8.0	212	3.0	25	2.0	7	1.0
1989	301	3.0	47	8.0	218	3.0	31	2.0	5	1.0
1990	270	3.0	42	7.0	193	3.0	29	2.0	6	1.0
1991	307	3.0	52	8.1	220	3.0	29	2.0	6	1.0
1992	291	3.0	49	8.0	203	3.0	32	2.0	7	1.0
1993	267	2.4	45	7.0	182	2.4	34	2.0	6	1.0
1994	296	3.0	50	8.0	207	3.0	32	2.0	7	1.0
1995	257	2.2	38	6.0	176	2.3	34	2.0	9	1.0
1996	221	2.0	48	7.0	139	2.0	27	1.3	7	1.0

Rates are adjusted to the 1970 U.S. Standard population and presented per 100,000 population.
Source: California Department of Health Services, Death Certificate Master Files, 1970-1996.

Prepared by California Department of Health Services, Cancer Surveillance Section.

AGE

Table II-4: Number of Deaths 65 Years and Over and Truncated Age-Adjusted Prostate Cancer Mortality Rates, California, 1970-1996

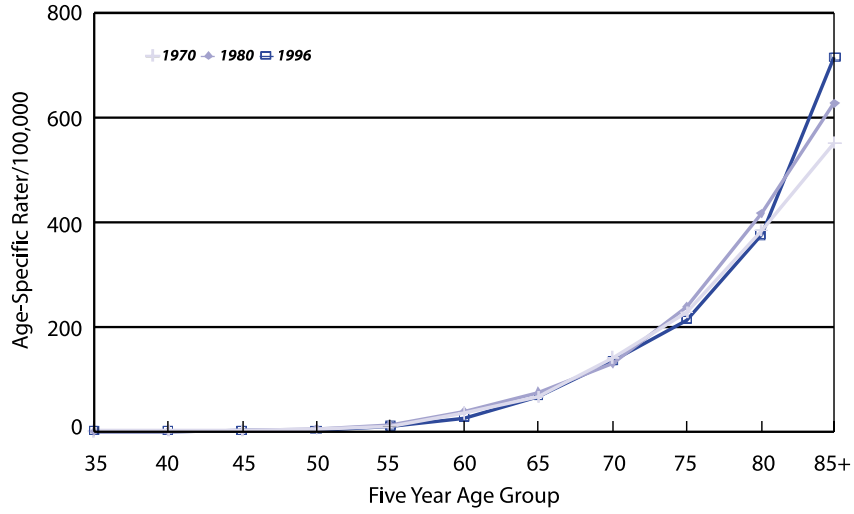
65years and over	Race/Ethnicity									
	All Races Combined		Non-Hispanic Black		Non-Hispanic White		Hispanic		Asian/Other	
Year	Deaths	Rate	Deaths	Rate	Deaths	Rate	Deaths	Rate	Deaths	Rate
1970	1282	188.0	78	322.4	1129	192.0	58	147.0	17	68.0
1971	1343	191.0	84	314.0	1170	193.0	72	170.0	17	60.0
1972	1368	188.1	110	405.0	1174	187.4	69	152.0	15	62.0
1973	1367	181.4	90	318.0	1196	185.0	69	144.2	12	37.0
1974	1490	193.0	101	332.0	1282	194.0	83	164.0	24	74.0
1975	1480	185.0	110	345.0	1293	190.0	58	109.0	19	58.0
1976	1621	198.0	113	334.0	1388	199.0	90	158.3	30	90.0
1977	1703	200.2	124	348.3	1458	202.4	91	154.0	30	84
1978	1836	211.0	165	442.2	1533	209.0	109	171.4	29	81.4
1979	1826	205.1	170	446.2	1546	207.0	77	116.4	33	91.0
1980	1861	200.0	166	406.0	1562	201.0	104	144.0	29	71.0
1981	1947	202.0	185	427.0	1625	203.0	99	128.0	38	87.0
1982	2095	210.0	170	377.1	1759	213.3	120	148.0	46	99.0
1983	2042	198.1	176	377.2	1709	201.4	109	128.0	48	99.0
1984	2110	198.0	220	451.0	1711	196.0	121	135.0	58	107.4
1985	2219	203.2	199	401.0	1837	206.3	117	124.4	64	114.4
1986	2379	212.0	235	449.0	1943	214.0	135	136.3	65	106.0
1987	2499	217.0	218	414.3	2033	219.0	173	166.0	73	110.0
1988	2543	216.0	242	446.4	2054	218.0	172	158.0	75	108.1
1989	2593	217.0	232	427.0	2131	223.3	135	120.3	92	124.2
1090	2729	217.4	269	471.0	2202	222.2	164	135.3	93	110.1
1991	2968	228.0	278	469.0	2367	232.2	220	169.0	102	111.0
1992	2995	221.3	260	429.0	2419	231.0	209	149.0	106	104.1
1993	3093	223.0	288	468.1	2464	231.2	216	146.0	125	113.3
1994	3025	213.0	278	437.4	2417	224.0	239	149.0	91	77.1
1995	2934	200.3	295	457.2	2290	208.0	233	139.0	114	89.0
1996	2949	195.0	272	409.0	2332	207.0	229	123.0	115	82.4

Rates are adjusted to the 1970 U.S. Standard population and presented per 100,000 population.
Source: California Department of Health Services, Death Certificate Master Files, 1970-1996.

Prepared by California Department of Health Services, Cancer Surveillance Section.

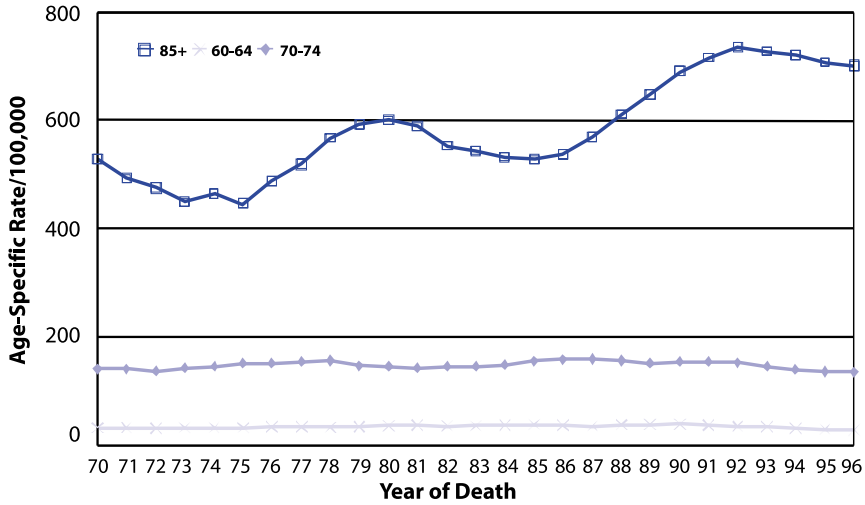
that the majority of prostate cancer deaths occur in males over the age of 65. In 1970, 15 percent of all deaths were under 65 years of age. In 1996 this is 7.5 percent. Age-specific mortality rates in 1970, 1980, and 1996 for men over 35 years of age are presented in Figure II-3. Except for minor fluctuations for men 85 years of age and older, the trend of age-specific mortality for these years is identical. Figure II-4 shows the patterns of age-specific mortality rates by selected age groups. The contrast between the rates over the study period for the younger age groups (60-64 and 70-74) with age group 85 and over is noteworthy. Since 20 percent of all prostate cancer death during this period has occurred among men over 85 years of age, even limited fluctuations in this group may result in significant deviations in the overall pattern.

Figure II–3: Prostate Cancer Mortality, All Races Combined by Selected Years of Death, California, 1970-1996



Prepared by California Department of Health Services, Cancer Surveillance Section.

Figure II–4: Prostate Cancer Mortality, All Races Combined, Selected Age Groups,* California, 1970-1996



** Three year moving average*

Prepared by California Department of Health Services, Cancer Surveillance Section.

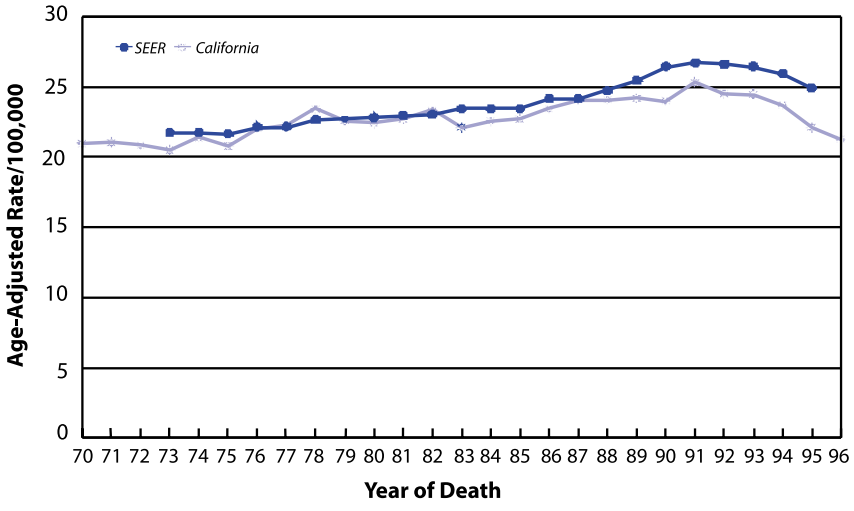
AGE

Studies of prostate cancer mortality in non-white US residents,[4] and of prostate cancer mortality trends in Australia, England and Wales,[5] suggest that the mortality rate from prostate cancer was decreasing for those born after 1900. Based on these and similar observations, it was suggested that prostate cancer mortality in the US may follow a similar pattern and begin to decrease soon after 1991.[6] In California, the median age of prostate cancer deaths for all races combined increased from 76 years in 1970-1991 to 78 years in 1991-1996. The median age for non-Hispanic blacks changed from 74 to 75 years during the same periods. These results seem to support the suggestion that the observed decrease in the prostate cancer mortality rates in California since 1991 may represent a birth cohort effect, although the data included in this study do not extend far enough back in time to allow for such evaluation.

COMPARISON WITH THE UNITED STATES

Prostate cancer mortality rates in California have followed a pattern similar to the rates in the United States as reported by SEER. However, the magnitude is different. The overall prostate cancer mortality rate in California has increased at an estimated annual rate of 0.82 percent, whereas in the US it has increased by a rate of 1.07 percent before the peak in 1991. Since then, however, the US rate has decreased by an annual rate of 1.65 percent, and California rate has decreased by a rate of 3.42 percent. As shown in Figure II-5, there is a noticeable separation between prostate mortality rates in California and the US beginning in 1987 and continuing to 1996. This may be a function of the race/ethnicity composition of California versus the United States.

Figure II–5: Prostate Cancer Mortality, All Races Combined, California and SEER, 1970-1996



*SEER: Surveillance, Epidemiology and End Results Program of the National Cancer Institute.

Prepared by California Department of Health Services, Cancer Surveillance Section.

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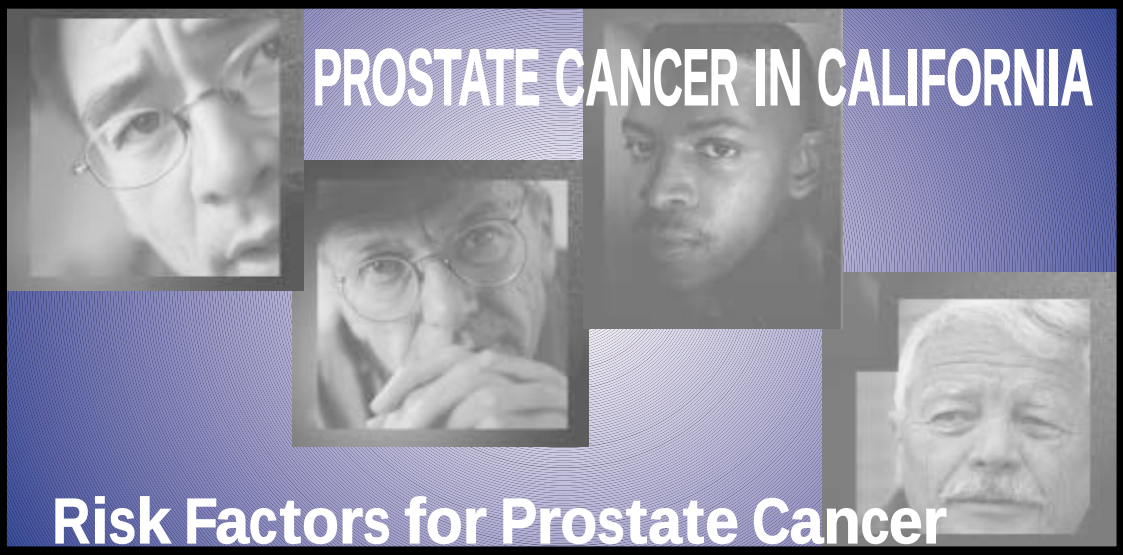

Risk Factors for Prostate Cancer

Wendy Cozen, D.O.[1] and Lihua Liu, Ph.D.[1]

[1] Cancer Surveillance Program, University of Southern California, Los Angeles CA

GENERAL/DEMOGRAPHIC

Prostate cancer is currently the most common cancer diagnosed among men in industrialized countries. The single biggest risk factor for prostate cancer is age; the disease increases after age 40 in a logarithmic fashion throughout life.[1] Worldwide, black men experience the highest rates of prostate cancer, followed by whites of European origin, especially Northern Europeans.[2] Asian men have the lowest risk of all ethnicities. This includes Asian men born in Asia and elsewhere.[2] Migrants from lower risk areas (e.g., Poland and Japan) who moved to the U.S. have an incidence of prostate cancer that is intermediate between their country of origin and that observed in U.S. whites, [3, 4] suggesting that environmental factors play a role in prostate cancer risk. Of course, an alternative explanation of the increased incidence observed in migrants to the U.S. is increased screening.

There is a slight increase in risk among men living in urban versus rural areas,[5] but this could be attributed to greater access to medical care, and thus, diagnosis.

Prostate cancer incidence varies by religious group: Mormons in Utah and Jews in Los Angeles have a 10-15 percent increased incidence,[6, 7] but Seventh Day Adventists, who advocate a vegetarian diet, have a decreased incidence.[8, 9] Among white men, married men have a higher risk of prostate cancer than non-married men,[10] but this, again, could be due to increased use of medical services resulting in more frequent diagnosis.

Prior to the introduction of the prostate-specific antigen (PSA) blood test for diagnosis of prostate cancer, there was no difference in incidence between men of high and low social class as measured by income and education (see Chapter 10, Socioeconomic Status and Prostate Cancer). However, after 1987, coinciding with the introduction of this test, a well-defined social class gradient became evident, with men of higher social classes experiencing higher incidence through 1992, compared to men of lower social class. This time-dependent gradient suggests that men of high social class have had more access to the PSA test, and thus are at a higher risk of getting diagnosed compared to men without access to a PSA test. Whether the PSA test is contributing to the detection of clinically relevant disease, i.e., disease that will progress to cause mortality, is still under evaluation.

DIET

International comparisons of prostate cancer incidence and mortality show a positive correlation between estimates of *per capita* fat intake and prostate cancer incidence and mortality.[11] Since then, at least 41 studies have been conducted in men to evaluate the relationship between fat consumption and prostate cancer.[12] Overall, it appears that consumption of meat and dairy products, the major sources of fat in Western countries, is associated with an increase in prostate cancer risk. However, it is not clear that this increased risk is due to the fat content of these foods.[12] Other substances contained in these animal foods could be responsible, such as zinc or calcium, or it could be that diets high in animal products are low in plant-derived foods, and this deficit is what is responsible. Methods of cooking meat, such as grilling, frying, or barbecuing, produce carcinogens which could be the actual culprits. Finally, diets high in fat might increase prostate cancer risk indirectly through hormonal mechanisms; high fat diets appear to increase testosterone[13] and lowfat diets reduce them.[14]

Red meat consumption has also been specifically linked to an increased risk of advanced prostate cancer, implying that it accelerates the disease.[15] Animal studies support the role of certain types of fat in accelerating the growth of prostate cancers once started, while other types of fat inhibit growth.[12] The preponderance of evidence suggests that at least some types of fat are involved in prostate cancer risk and/or progression, but the relationship is complex and further work needs to be done to clarify this issue.

DIET

Other dietary factors that have been examined include Vitamin A and beta carotene, Vitamin D, phytoestrogens (soy products), and most recently, lycopene, a carotinoid substance found in tomatoes. There have been a large number of studies evaluating the effect of consumption of green–yellow vegetables, Vitamin A and beta carotene, and there is no consistent evidence that these substances protect against the development of prostate cancer.[10] There are limited data on Vitamin D and prostate cancer risk, but initial studies suggest that high serum Vitamin D levels may protect against prostate cancer risk[16, 17] and high calcium intake may increase risk.[17]

Phytoestrogens, literally "plant estrogens," are substances with weak estrogen-like properties found in foods like soy beans. Because Asians have low incidence rates of breast and prostate cancer, and consume high levels of soy-containing foods, it has been postulated that these foods may play a protective role against the development of hormone-related cancer.[18] With respect to prostate cancer, these compounds may act by binding to androgen receptors and thus interfering with the action of testosterone on the prostate.[19]

Several prospective dietary studies have found incidentally that consumption of tomatoes and tomato-based products are associated with lower prostate cancer risks;[9, 20] however, serum-based studies evaluating lycopene (the carotinoid in tomatoes that makes them red) have not replicated these findings.[21, 22] At present, the conclusions regarding the protective effect of tomatoes remain tentative and more research may resolve the issue.

ALCOHOL

There was early speculation that excessive alcohol use may actually decrease prostate cancer risk because of its inhibitory effect on the production of male hormones, which stimulate the prostate gland. An autopsy series of men with alcohol-induced liver cirrhosis reported a large deficit of prostate cancer in these men.[23] Subsequent studies designed to evaluate the issue have not demonstrated much of an effect of alcohol, either positive or negative, on prostate cancer risk. However, most of these studies examined low to moderate levels of drinking, and the role of excessive alcohol intake has not been adequately studied.[10, 24]

TOBACCO

Results regarding tobacco use and prostate cancer risk are conflicting and confusing, and seem to depend on the population being studied. Early studies found that smoking either decreased prostate cancer risk or had no effect.[10] In a study of relatively wealthy retirement home residents, current smokers had a slightly reduced risk of prostate cancer, while long-term smokers had a significantly reduced risk.[25] Other studies have found the opposite for current smokers- from a 2- to 3- fold increase in risk among heavy smokers.[21, 26, 27] It is possible that sociocultural determinants of smoking that vary from population to population are actually responsible for the apparent increase in risk in some populations and that the relationship between smoking and prostate cancer is not causal.[10]

SEXUAL ACTIVITY AND SEXUALLY TRANSMITTED DISEASES

A number of studies have been published that evaluate the relationship between sexual activity and prostate cancer risk. The results have been inconsistent, with some studies showing a positive association with a variety of factors (age at first intercourse, frequency of intercourse and number of sexual partners) and some studies showing no association. One reason for the inconsistent results may be the difficulty in obtaining accurate histories from patients and controls. However, most studies show a 2 to 3-fold increase in the risk of prostate cancer linked to a history of sexually transmitted diseases, particularly gonorrhea. There are no plausible biological theories to explain

SEXUAL ACTIVITY & SEXUALLY ...

prostate cancer on the basis of infection, and the underlying hypothesis is that a history of sexually transmitted diseases is a more accurate marker of sexual activity and thus for higher androgen levels (see below, *Hormones*).

VASECTOMY

The preponderance of evidence from a handful of studies supports a modest increase (about 50 percent) in prostate cancer risk among vasectomized men which increases with time since vasectomy. One large study recently conducted among men of different racial and ethnic groups shows no such association.[28] As in sexually transmitted diseases, the relationship, if present, is probably indirect. For example, there is some evidence that vasectomized men have higher testosterone levels compared to men of similar age and race who have not undergone this procedure.[28, 29]

HORMONES

Because male sex hormones, such as testosterone, regulate prostate growth, it is reasonable that they might play a role in the development of prostate cancer. Surgical or chemical castration is a treatment for prostate cancer, demonstrating that the tumors are dependent on male sex hormones for progression. It is difficult to study hormone levels in prostate cancer patients directly because the disease itself may alter the levels. However, testosterone levels are higher among black young men compared to white young men, correlating with their increased risk of the disease.[29] Asian men do not have lower levels of testosterone compared to white men, however, they do have evidence of a lower rate of conversion of testosterone to its active form, dihydrotestosterone, by the enzyme 5-alpha-reductase.[30, 31] This indirect evidence supports the hypothesis that higher levels of testosterone, or its active form, dihydrotestosterone, may contribute to increased prostate cancer risk.

FAMILY HISTORY/ GENETICS

There is strong evidence that prostate cancer is familial. Men who have a first-degree relative diagnosed with prostate cancer have a 2- to 3- fold increased risk of developing the disease[10]. This increased risk increases to 5-fold among men with 2 affected first-degree relatives, and 10-fold among men with more than 2 affected first-degree relatives.[32] It is important, however, to distinguish between "genetic or inherited" factors and "familial" factors, which imply either an inherited factor or environmental factors that the families share in common, such as diet. Hereditary prostate cancer implies that there is a single gene passed through families that is the cause of prostate cancer in these families. Typically, prostate cancer occurs at younger ages in such families and most likely accounts for no more than about 9 percent of all prostate cancer cases.[33]

The majority of cases of prostate cancer among families are probably multifactorial, due to both genetic and environmental factors. Ross and colleagues have hypothesized that variations in the genes controlling male hormone metabolism might play a role in the development of prostate cancer.[19] The frequencies with which these genetic variations occur in blacks, white Americans and Asian Americans, correlate with the ethnic-specific differences in prostate cancer incidence,[34, 35] and could explain ethnic differences in risk.[36] Specific variations in two of these genes, the androgen receptor gene[37] and the 5-alpha-reductase gene,[38] have been linked to the development of prostate cancer. A third gene that is being evaluated is the gene that codes the Vitamin D receptor, since high Vitamin D levels may be protective for prostate cancer. Variations in the Vitamin D receptor gene may affect the way that Vitamin D binds to its receptor and thereby its subsequent action on the target cells. Initial studies suggest that certain forms (polymorphisms) of the Vitamin D receptor gene are strongly associated with prostate cancer,[39] and larger studies are now being conducted.

OCCUPATION

Several specific occupational exposures, such as cadmium, farming, and rubber and textile manufacturing, have been reported to be associated with an increased risk of prostate cancer, however, none of the findings have consistently held up in subsequent studies.[10] Because some elevation of prostate cancer risk has been noted in farmers, concerns were raised that pesticides might contribute to risk. There have not yet been many reports but one in Swedish pesticide applicators showed a slight (13 percent), but significant, increased risk of prostate cancer among the workers.[40] Based on studies conducted to date, it is not likely that occupational exposures explain a significant number of prostate cancer cases.

In summary, the causes of prostate cancer are multiple but the strongest risk factors appear to be positive family history, black ethnicity, animal fat consumption and possibly specific genetic factors.

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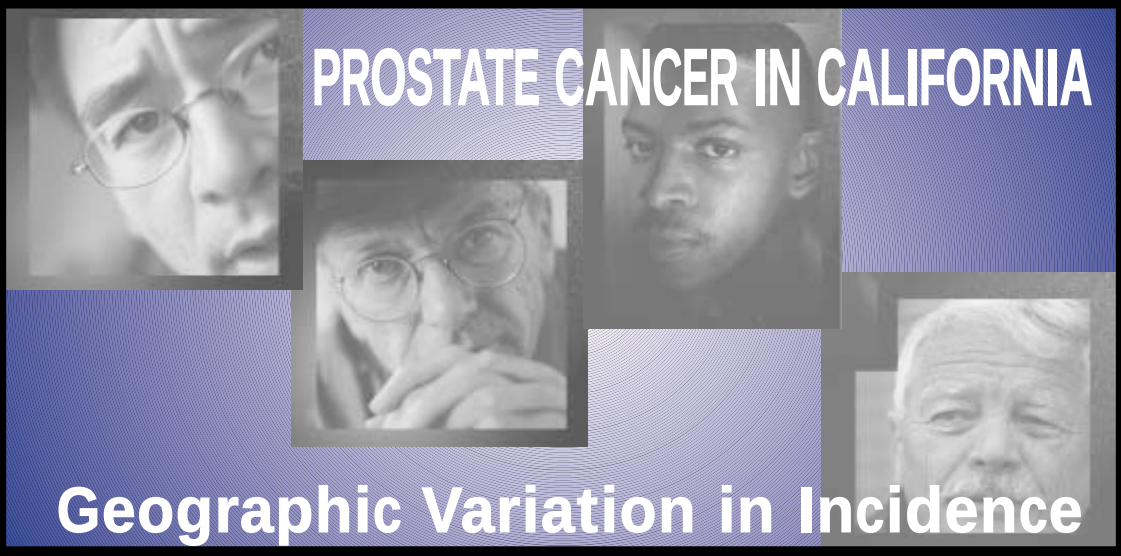

Geographic Variation in Incidence and Mortality of Prostate Cancer in California, 1991-1995

Rosemary D. Cress, Dr.P.H.[1] and John W. Morgan, Dr.P.H.[2,3]

[1] Cancer Surveillance Program, Sacramento, CA
[2] Desert Sierra Cancer Surveillance Program, Loma Linda, CA
[3] Loma Linda University School of Public Health

CHAPTER IV

INTRODUCTION

Geographic variation in rates of certain cancers can suggest directions for research into factors that might influence the risk of developing or dying from these cancers. Prostate cancer is a good example. Its incidence varies widely among different countries, with the lowest rates in Asian countries and the highest rates in the United States. The incidence of prostate cancer increases in Asian men who emigrate to the United States. Geographic variation in cancer risk is generally agreed to reflect the effects of environmental differences, with environment defined in its broadest sense as any factor that affects the residents of a geographic area.[1] Such factors may include cultural effects on lifestyle and behaviors such as consumption of different foods, and diet has been shown to influence risk of prostate cancer. Analysis of geographic variation in prostate cancer incidence (rate of new cases) and mortality (rate of death) is complicated by the effect of screening on both incidence and mortality. Screening may increase the number of cancers detected at an early stage, and decrease the number of people who die from this cancer. This chapter describes incidence and mortality of prostate cancer by county for California.

METHODS

Because risk for prostate cancer varies by race, rates were calculated for each of four major race/ethnicity groups: non-Hispanic whites, non-Hispanic blacks, Hispanics, and non-Hispanic Asian/Pacific Islanders. Rates were calculated for counties with five-year race-specific populations of at least 100,000 men and with at least 15 new cases or deaths between 1991 and 1995. Rates were age-adjusted to the 1970 US population, and age-adjusted race-specific rates that differed significantly from the California rate are indicated with statistical significance set at a level of 0.01.

RESULTS

Tables IV-1 and IV-2 display the average annual age-adjusted incidence and mortality rates of prostate cancer by county for all races combined and for each race/ethnicity group for 1991-1995. Figures IV-1 and IV-2 illustrate these rates in graphical form, with shading to discriminate counties with rates that were statistically significantly higher, lower, or not significantly different from the state rate.

There was substantial variation in incidence by county. For example, among non-Hispanic white men risk of being diagnosed with prostate cancer was almost twice as high in Marin County (188.8 cases per 100,000 population) as in Kings County (94.8 cases per 100,000 population). In contrast, there was little variation in mortality by county.

For non-Hispanic white men, seven counties (Contra Costa, El Dorado, Los Angeles, Marin, Sacramento, San Diego, and Ventura) had age-adjusted incidence rates that were statistically significantly higher than the average rate for California. Thirteen counties had rates that were significantly lower than the state average: Butte, Humboldt, Kern, Kings, Mendocino, Nevada, Riverside, San Bernadino, San Joaquin, Sonoma, Stanislaus, Sutter, and Tehama. Two counties had mortality rates for non-Hispanic white men that were statistically significantly higher than the state average (Humboldt and Orange) and one had a significantly lower rate (Riverside).

Hispanic men in Orange and San Diego Counties had incidence rates that were significantly higher than the average rate for Hispanics in California. Those who lived in San Joaquin and Monterey Counties had significantly lower mortality rates than the state average. Only San Bernadino County had a mortality rate significantly higher than the state average for Hispanic men. For Asian men, Solano and San Diego Counties had incidence rates substantially higher than the average age-adjusted incidence rate, while rates for Asians in San Joaquin County were significantly lower than the state average.

RESULTS

Only in Solano County did Asian men have a mortality rate significantly higher than the state average. For non-Hispanic black men, incidence rates were significantly higher than the state average in Sacramento County and were significantly lower in Alameda County. In none of the ten counties where mortality rates were calculated for black men did mortality differ significantly from the state average.

Table IV-1: Five-Year Prostate Cancer Incidence Counts[1] and Average Annual Age-Adjusted Rates[2] Per 100,000 Population by County of Residence and Race/Ethnicity[3], California, 1991-1995

	All Races		Asian/P.I.		Black		Hispanic		White	
County	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate
California	100,473	147.4	4,006	74.8	7,658	226.1	9,074	108.5	74,906	148.4
Alameda	4,011	143.4	267	70	822	200.9	* 271	101.2	2,511	145.5
Alpine	1	-	-	-	-	-	-	-	-	-
Amador	215	-	-	-	1	-	6	-	208	-
Butte	1,009	141.9	4	-	11	-	35	-	893	134.0*
Calaveras	211	-	1	-	2	-	7	-	193	-
Colusa	55	-	-	-	1	-	6	-	45	-
Contra Costa	3,127	159.0*	95	73.2	279	231.7	136	113.2	2,485	157.0*
Del Norte	120	-	2	-	-	-	2	-	112	-
El Dorado	673	174.8*	5	-	4	-	17	-	643	176.2*
Fresno	2,297	149.7	66	64	129	-	319	109.7	1,611	152.2
Glenn	87	-	-	-	1	-	5	-	80	-
Humboldt	367	117.8*	3	-	2	-	7	-	350	119.9*
Imperial	374	129.3	6	-	15	-	154	123	178	-
Inyo	88	-	1	-	-	-	2	-	83	-
Kern	1,573	123.9*	31	-	84	-	148	92	1,130	114.3*
Kings	204	109.6*	7	-	16	-	43	109.7	126	94.8*
Lake	338	135.5	-	-	13	-	11	-	294	128.6
Lassen	31	-	-	-	-	-	-	-	29	-
Los Angeles	27,290	152.3*	1,351	73.3	3,807	233.6	3,498	105.5	17,350	158.0*
Madera	432	165.3	2	-	23	-	53	-	327	158.9
Marin	1,177	185.9*	17	-	15	-	15	-	1,098	188.1*
Mariposa	85	-	-	-	1	-	1	-	83	-
Mendocino	327	142.2	2	-	4	-	7	-	269	125.9*
Merced	564	148.2	8	-	38	-	93	141.5	376	136.3
Modoc	16	-	-	-	-	-	1	-	14	-
Mono	10	-	-	-	-	-	1	-	8	-
Monterey	958	128.9*	64	-	49	-	97	82.9*	718	134.7
Napa	525	136.9	11	-	5	-	20	-	484	138.7
Nevada	403	117.3*	-	-	-	-	11	-	390	116.7*

[1] Cases for counties with five-year race-specific population sums of less than 100,000 are not shown.
[2] Rates are age-adjusted to the 1970 U.S. population. Rates based on fewer than 15 cases are not shown. Data are from the California Cancer Registry (01/98).
[3] Race/ethnicity are mutually exclusive. Persons of Hispanic ethnicity are identified by death certificate and/or surname, and may be of any race. Men of unknown race/ethnicity are excluded from race-specific data.
* Rate is significantly different from comparable race-specific statewide rate, 1991-1995 ($p<0.01$).

Prepared by California Department of Health Services, Cancer Surveillance Section.

RESULTS

continued

Table IV-1: Five-Year Prostate Cancer Incidence Counts[1] and Average Annual Age-Adjusted Rates[2] Per 100,000 Population by County of Residence and Race/Ethnicity[3], California, 1991-1995

	All Races		Asian/P.I.		Black		Hispanic		White	
County	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate
Orange	7,325	150.2	237	70.3	83	260.2	563	121.9*	6,140	153
Placer	782	155.9	11	-	4	-	33	-	720	157.5
Plumas	63	-	-	-	-	-	-	-	61	-
Riverside	5,366	147.2	45	72.6	213	207.4	397	110.7	4,321	140.0*
Sacramento	3,950	163.1*	154	79.7	351	267.8	* 199	120.5	3,167	165.3*
San Benito	88	95.8*	1	-	1	-	22	-	61	-
San Bernardino	3,923	140.6*	53	79.4	261	224.8	449	113.9	2,918	133.2*
San Diego	9,428	162.4*	277	100.9*	326	207.1	685	121.8*	7,671	161.2*
San Francisco	2,858	134.5*	470	70.1	455	249.9	202	105.3	1,611	149.2
San Joaquin	1,316	111.1*	73	45.5*	69	-	121	76.0*	1,024	123.8*
San Luis Obispo	1,113	167.9*	2	-	20	-	37	-	932	153.8
San Mateo	2,394	138.5*	167	88.1	120	-	153	113.6	1,866	140.5
Santa Barbara	1,455	155.3	23	-	22	-	142	119	1,184	154.2
Santa Clara	4,159	140.7*	325	76.8	120	234.6	399	108.4	3,146	149.3
Santa Cruz	757	146.2	26	-	9	-	67	142.5	591	134.4
Shasta	747	157.7	2	-	3	-	17	-	695	153.8
Sierra	12	-	-	-	-	-	-	-	12	-
Siskiyou	176	114.2*	1	-	2	-	4	-	158	-
Solano	930	143.3	83	121.6*	144	228.2	56	97.2	631	136.9
Sonoma	1,280	120.7*	4	-	10	-	40	82.7	1,185	121.1*
Stanislaus	1,130	128.4*	13	-	25	-	73	83	904	122.7*
Sutter	211	114.9*	13	-	5	-	15	-	176	114.9*
Tehama	207	110.8*	1	-	2	-	6	-	192	109.2*
Trinity	82	-	-	-	-	-	-	-	78	-
Tulare	964	129.1*	14	-	21	-	137	100.2	770	139.6
Tuolumne	274	143.6	2	-	1	-	10	-	244	135.3
Ventura	2,310	160.6*	55	-	47	-	229	116	1,849	160.5*
Yolo	422	140.4	10	-	10	-	40	-	355	148.5
Yuba	183	129.6	1	-	12	-	12	-	156	133.1

[1] Cases for counties with five-year race-specific population sums of less than 100,000 are not shown.
[2] Rates are age-adjusted to the 1970 U.S. population. Rates based on fewer than 15 cases are not shown. Data are from the California Cancer Registry (01/98).
[3] Race/ethnicity are mutually exclusive. Persons of Hispanic ethnicity are identified by death certificate and/or surname, and may be of any race. Men of unknown race/ethnicity are excluded from race-specific data.
* Rate is significantly different from comparable race-specific statewide rate, 1991-1995 ($p<0.01$).

Prepared by California Department of Health Services, Cancer Surveillance Section.

Table IV-2: Five-Year Prostate Cancer Mortality and Average Annual Age-Adjusted Rates[1,2] Per 100,000 Population by County of Residence and Race/Ethnicity[3], California, 1991-1995

	All Races		Asian/P.I		Black		Hispanic		White	
County of Residence	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate
Alameda	790	28.1*	33	8.8	229	56.3	54	21.2	473	26.8
Alpine	0	-	0	-	0	-	0	-	0	-
Amador	34	-	0	-	1	-	0	-	33	-
Butte	180	22.3	0	-	4	-	3	-	173	22.8
Calaveras	28	-	0	-	0	-	1	-	27	-
Colusa	6	-	0	-	0	-	0	-	6	-
Contra Costa	499	26.1	15	12.5	51	45.5	14	-	419	27
Del Norte	12	-	0	-	0	-	0	-	12	-
El Dorado	90	24.2	1	-	1	-	2	-	86	24.2
Fresno	393	24.4	9	-	22	-	51	18.4	310	27
Glenn	12	-	0	-	2	-	1	-	9	-
Humboldt	114	34.6*	0	-	0	-	2	-	111	35.8*
Imperial	69	23.4	4	-	4	-	28	24.8	33	-
Inyo	18	-	0	-	0	-	0	-	18	-
Kern	294	22.9	5	-	31	-	29	19.7	229	22.7
Kings	32	16.7	0	-	2	-	7	-	23	16.7
Lake	69	25.3	0	-	5	-	7	-	57	22.3
Lassen	16	-	0	-	0	-	0	-	16	-
Los Angeles	4,204	23.7	163	9.5	782	51.4	538	17.3	2,717	24
Madera	54	20.2	1	-	7	-	4	-	42	19.9
Marin	164	26.3	2	-	2	-	3	-	157	27
Mariposa	18	-	0	-	1	-	1	-	16	-
Mendocino	70	28.5	1	-	0	-	2	-	67	29.2
Merced	85	22.1	1	-	5	-	8	-	71	24.8
Modoc	8	-	0	-	0	-	0	-	7	-
Mono	6	-	0	-	0	-	2	-	4	-
Monterey	172	23	10	-	12	-	14	-	136	25.1
Napa	105	24.5	1	-	0	-	6	-	98	25.1
Nevada	71	20.8	0	-	0	-	2	-	69	20.5

1 Cases for counties with five-year race-specific population sums of less than 100,000 are not shown.

2 Rates are age-adjusted to the 1970 U.S. population. Rates based on fewer than 15 cases are not shown. Data are from the CDHS Center for Health Statatistics Death Master Files, 1991-1995.

3 Race/ethnicity are mutually exclusive. Persons of Hispanic ethnicity are identified by death certificate and/or surname, and may be of any race. Men of unknown race/ethnicity are excluded from race-specific data.

* Rate is significantly different from comparable race-specific statewide rate, 1991-1995 ($p<0.01$).

Prepared by California Department of Health Services, Cancer Surveillance Section.

RESULTS

continued

Table IV-2: Five-Year Prostate Cancer Mortality and Average Annual Age-Adjusted Rates[1,2] Per 100,000 Population by County of Residence and Race/Ethnicity[3], California, 1991-1995

	All Races		Asian/P.I		Black		Hispanic		White	
County of Residence	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate	Cases	Annual Rate
Orange	1,166	25.5	25	8.2	13	-	81	20.8	1,046	27.2*
Placer	121	24	1	-	0	-	3	-	117	25.6
Plumas	10	-	0	-	0	-	0	-	9	-
Riverside	794	20.8*	3	-	41	42.6	67	21.1	683	20.7*
Sacramento	569	24.2	24	12.3	63	53.9	26	16.6	454	24.2
San Benito	20	21.3	1	-	0	-	3	-	16	-
San Bernardino	721	26.3	9	-	49	47	97	26.6*	564	25.6
San Diego	1,520	25.3	33	13.4	75	54.9	82	15.5	1,328	26.1
San Francisco	486	21.2*	56	8.1	95	51.4	38	18.6	295	24.2
San Joaquin	296	23.2	23	10.7	20	-	33	22.4	219	24.5
San Luis Obispo	164	23.2	4	-	2	-	5	-	153	23.6
San Mateo	393	23	22	13	33	-	23	18.4	314	23.1
Santa Barbara	222	21.9	12	-	6	-	13	-	190	22.3
Santa Clara	622	22.7	45	11.1	12	33.9	62	18.6	501	25.7
Santa Cruz	134	23.5	7	-	1	-	7	-	119	24.4
Shasta	129	26.5	0	-	3	-	1	-	125	26.9
Sierra	4	-	0	-	0	-	0	-	4	-
Siskiyou	54	33.2	0	-	1	-	0	-	53	-
Solano	158	25.9	15	21.1*	32	62.6	8	-	103	23.8
Sonoma	281	24.2	3	-	2	-	10	-	265	24.5
Stanislaus	222	24.3	2	-	2	-	19	23.3	199	25.4
Sutter	49	25.9	0	-	0	-	2	-	47	29.4
Tehama	53	27.3	1	-	0	-	1	-	50	27.2
Trinity	14	-	0	-	0	-	0	-	14	-
Tulare	192	24.2	4	-	9	-	16	12.9	162	27.3
Tuolumne	37	18.8	0	-	0	-	2	-	35	18.5
Ventura	282	20.0 *	7	-	6	-	20	10.9	249	21.5
Yolo	65	20.9	4	-	4	-	1	-	56	22.4
Yuba	42	28.9	0	-	1	-	2	-	39	32.5

1 Cases for counties with five-year race-specific population sums of less than 100,000 are not shown.
2 Rates are age-adjusted to the 1970 U.S. population. Rates based on fewer than 15 cases are not shown. Data are from the CDHS Center for Health Statatistics Death Master Files, 1991-1995.
3 Race/ethnicity are mutually exclusive. Persons of Hispanic ethnicity are identified by death certificate and/or surname, and may be of any race. Men of unknown race/ethnicity are excluded from race-specific data.
* Rate is significantly different from comparable race-specific statewide rate, 1991-1995 ($p<0.01$).

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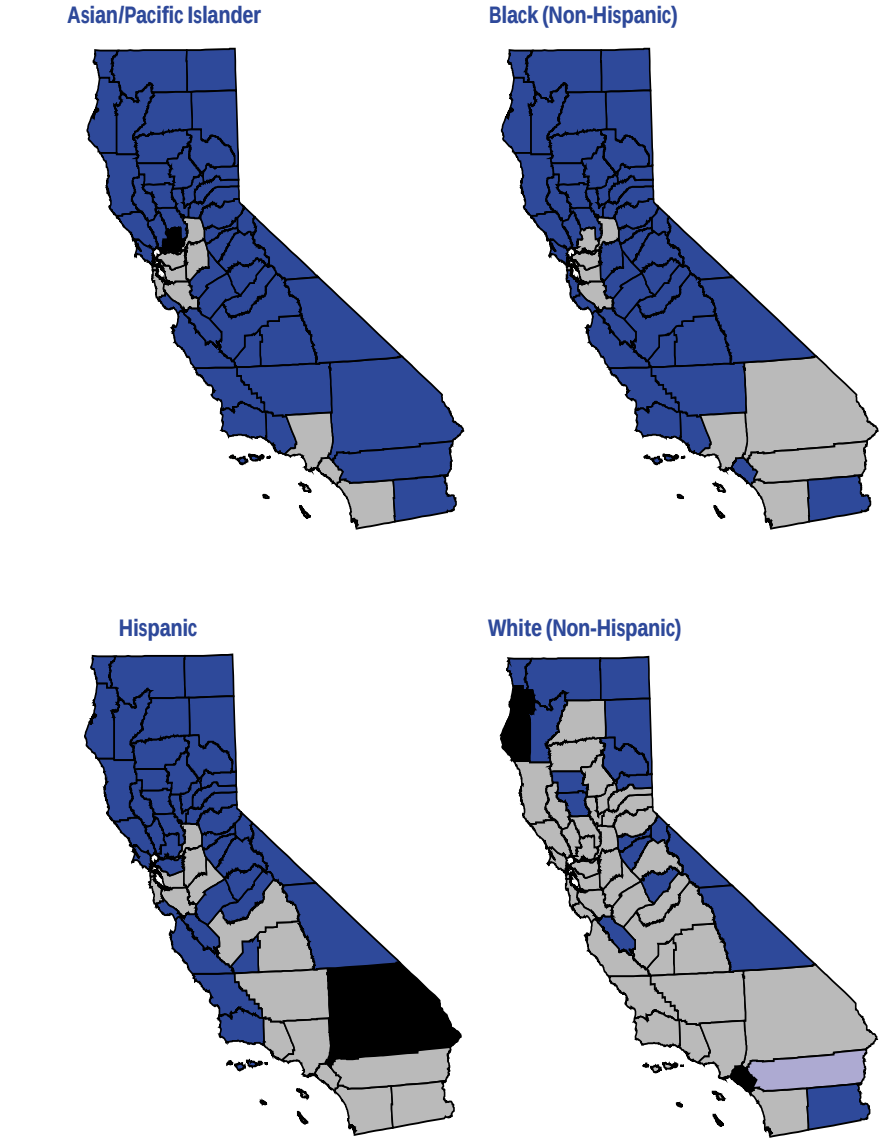
Figure IV–1

Age-Adjusted (US 1970) Prostate Cancer Mortality Rates by County and Race/Ethnicity, California, 1991-1995



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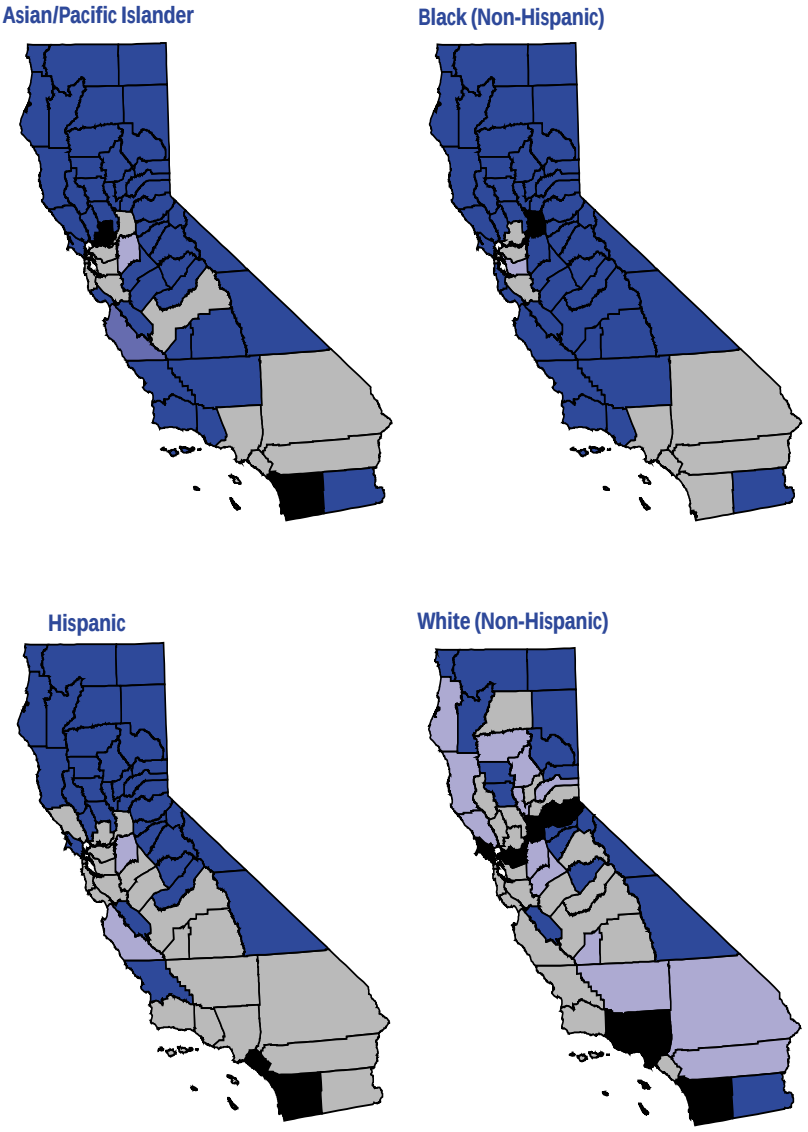
RESULTS

Figure IV–2

Age-Adjusted (US 1970) Prostate Cancer Incidence Rates by County and Race/Ethnicity, California, 1991—1995



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DISCUSSION

Screening for prostate cancer has been shown to increase detection of early stage cancer, and the popularity of prostate-specific antigen (PSA) testing has been credited with the unprecedented rise in prostate cancer incidence in the United States during the early 1990s, and with the later decline in incidence.[2-5] This effect of PSA screening on incidence also has been observed in California.[6] Because the 1991-1995 incidence rates presented in this report represent a time period when prostate cancer incidence rates peaked in California, especially among non-Hispanic whites, the observed variation in incidence by county reflects to some undetermined extent different patterns of prostate cancer screening. Because of the controversy about whether PSA screening leads to improved survival and lower mortality,[2,6] there has been wide geographic variation in utilization of this screening technique.[2] The geographic variation in incidence of prostate cancer observed in these data is consistent with variation by county in socioeconomic status that likely resulted in differences in access to health care, including PSA screening and use of other diagnostic procedures. In a preliminary statistical analysis, there was a substantial correlation between the average income for non-Hispanic white families by county (according to the 1990 US Census) and the average annual age-adjusted incidence rates for prostate cancer by county among non-Hispanic white men for 1991- 1995. The correlation was positive but less pronounced for other race/ethnicity groups. (See chapter VIII for further discussion of the relationship between socioeconomic status and prostate cancer).

Mortality rates by county in California showed a considerably different pattern from incidence rates. In none of the counties where incidence rates for non-Hispanic white men were significantly higher than average were the mortality rates also significantly higher. Although there is controversy about whether PSA screening contributes to reduced mortality from prostate cancer,[5] these results suggest that counties with high incidence rates had an increased utilization of screening that led to earlier diagnosis and better survival. Incidence rates for Asian, Hispanic, and non-Hispanic white men all were significantly higher than the state average in San Diego County, while mortality was similar to the state. Men in Humboldt County had a significantly lower incidence rate but had the highest mortality from prostate cancer. This suggests a deficit in prostate cancer screening and early diagnosis for men in this county. Similarly, incidence rates for Asian, Hispanic, and non-Hispanic white men were significantly lower than the state average in San Joaquin county, while mortality rates for all race/ethnicity groups were similar to the state average. Only among Asian men in Solano County were both incidence and mortality higher than the state average.

In conclusion, this preliminary analysis of geographic variation in race-specific incidence and mortality from prostate cancer in California revealed very different patterns for incidence and mortality. Overall risk of being diagnosed with prostate cancer was not related to risk of dying from this cancer. This disparity suggests that the geographic variation in incidence of prostate cancer probably can be attributed in large part to variation in screening practices rather that variation in prevalence of risk factors for prostate cancer. Counties with low incidence and high mortality may need to consider public health interventions to increase screening and early diagnosis. It is likely that the variation in county and race-specific incidence rates observed in this report will diminish as statewide rates stabilize.

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Prostate Cancer Survival in the San Francisco Bay Area (Region 8), 1973-1994.

Angela W. Prehn, Ph.D.,[1] Scarlett Lin, M.P.H.[1] and Jeanne Darbinian, M.P.H.[1]

[1] Northern California Cancer Center, Union City, CA

CHAPTER V

INTRODUCTION

The prognosis for patients with prostate cancer is one of the most favorable of all cancers. In the United States, only slightly more than one-third of men diagnosed with prostate cancer will die of their disease.[1] Unfortunately, not all men have an equal chance of surviving prostate cancer. Age at diagnosis, race/ethnicity, stage of disease, tumor grade, and year of diagnosis are all factors that affect survival from prostate cancer. Given the magnitude of prostate cancer occurrence, it is important to understand the role that these factors may play in determining who survives this disease.

This chapter presents relative survival for men diagnosed with invasive, microscopically confirmed prostate cancer in the San Francisco Bay Area (SF Bay Area) by year of diagnosis, age at diagnosis, race/ethnicity, stage of disease, and tumor grade. In addition, it explores racial/ethnic differences in long-term survival by stage of disease and includes a discussion of the effects of prostate cancer screening (e.g., testing for prostate-specific antigen (PSA)) on survival. Data are presented for the major racial/ethnic groups in the SF Bay Area: non-Hispanic whites (whites), white Hispanics (Hispanics), non-Hispanic blacks (blacks), and Chinese, Japanese, and Filipinos (Asians) combined. The racial/ethnic diversity and size of the SF Bay Area's population (over 3.5 million residents, 500,000 of them Asians/Pacific Islanders[2]) also lend themselves to a more detailed examination of prostate cancer relative survival in specific Asian/Pacific Islander subgroups. Therefore, where possible, data are presented separately for Chinese, Japanese, and Filipinos.

METHODS AND DEFINITIONS

Data Source

Cancer data have been collected in Alameda, Contra Costa, Marin, San Francisco, and San Mateo Counties since 1973 as part of the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) program. These counties, referred to here as the SF Bay Area, also form one part (Region 8) of the California Cancer Registry. As the SF Bay Area registry is the only regional registry within the state system to have collected follow-up information on cases since 1973, only SF Bay Area data are included in this chapter. Relative survival rates in this chapter were calculated using the National Cancer Institute SEER*Stat software, version 1.0.[3] This version of the software contains cancer registry data on cases diagnosed and follow-up performed from January 1, 1973 through December 31, 1994.

Relative Survival

Relative survival estimates the probability that an individual diagnosed with a particular disease will not die from that disease; the calculation statistically removes competing causes of mortality. These survival estimates are based on the expected survival rate for persons in the total population (i.e., life tables), taking into consideration age, sex, race, and calendar year of observation. Values in the expected life tables are available for U.S. non-Hispanic whites, blacks, Chinese, Japanese, and Filipinos. Life tables for white Hispanics are also available, although based on data collected only in New Mexico. Thus, although data are presented in the other chapters of this publication for all Asian/others as a group, this chapter presents data for an Asian group that includes only Chinese, Japanese, and Filipino men, as life tables are only available for these three Asian populations. However, these three groups together represent 98 percent of all Asian/others diagnosed with prostate cancer in the SF Bay Area.

Relative survival for men with prostate cancer may be expressed as a percentage ranging from 0 percent (all died of prostate cancer) to 100 percent (none died of prostate cancer). Five-year relative survival for prostate cancer is the probability that an individual diagnosed with this disease will not die from it for at least five years after diagnosis. In this chapter, five-year relative survival percentages are presented for prostate cases

METHODS AND DEFINITIONS

Table V-1: Prostate Cancer Five-Year Relative Survival Rates by Year of Diagnosis, San Francisco Bay Area, 1973-1990

Year of Diagnosis	Count[1]	Rate	95% CI
1973	738	71.2	66.1-76.2
1974	785	70.8	65.8-75.7
1975	845	68.4	63.7-73.1
1976	838	72.5	67.7-77.2
1977	887	73.6	69.1-78.2
1978	851	75.9	71.3-80.5
1979	916	70.9	66.3-75.4
1980	918	77	72.5-81.5
1981	953	72.9	68.5-77.4
1982	941	73.4	68.9-77.9
1983	1,023	73.6	69.3-77.8
1984	1,107	75.3	71.2-79.4
1985	1,176	73.9	69.9-77.9
1986	1,123	75.4	71.3-79.4
1987	1,353	80	76.4-83.7
1988	1,437	85.3	81.8-88.8
1989	1,505	86.4	83.0-89.8
1990	1,662	93.5	90.1-96.9

[1] Count refers to the number of cases alive at the start of the observation period.

Prepared by California Department of Health Services, Cancer Surveillance Section.

diagnosed in two time intervals: 1) 1973-1990, for examining changes over time, and 2) 1986-1990, for examining recent variations in prostate cancer survival by race/ethnicity. In addition, up to 21-year relative survival is presented by race/ethnicity and tumor stage at diagnosis for men diagnosed between 1973 and 1994 in order to examine differences in long-term survival from prostate cancer.

Tests for statistical significance of differences between relative survival rates of two groups are performed using the Z-test function within the SEER*Stat program. This procedure compares the survival curves of the two groups up to a selected survival duration point (Z-test interval), in this case, five years.[4] In this chapter, because multiple comparisons are being made, statistical significance is determined using alpha=.01. In the tables presented at the end of the chapter, 95 percent confidence intervals are displayed to provide an indication of the stability of the survival estimate. In addition, data for all race/ethnic groups combined are included in the tables along with the race/ethnicity-specific data.

Stage of Disease

In this chapter, tumor stage at diagnosis is classified as follows: localized tumors are those confined within the prostate capsule; regional tumors have penetrated throughout the capsule or involve regional lymph nodes; and distant tumors are those that have metastasized (spread) to distant organs, bones, or lymph nodes.

METHODS AND DEFINITIONS

Tumor Grade

Tumor grade describes how much a tumor still looks like normal tissue of its same type. In general, tumor grade is related to its rate of growth, with more poorly differentiated tumors growing more aggressively. Prostate tumors are often graded using the Gleason score, with a higher score indicating a more poorly differentiated tumor. In this chapter, tumors are classified into the following grades: well differentiated (look like normal cells, Gleason score 2-4); moderately differentiated (Gleason score 5-7); and poorly differentiated/undifferentiated (abnormal-looking and disorganized cells, Gleason score 8-10).

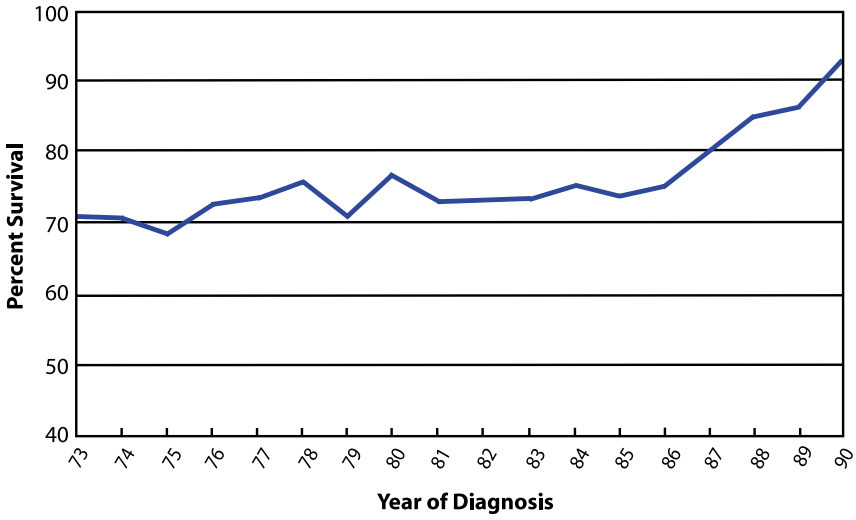
CHANGES OVER TIME IN FIVE-YEAR RELATIVE SURVIVAL, 1973-1990

Table V-1 and Figure V-1 presents patterns in five-year relative survival for men diagnosed with invasive prostate cancer while residing in the SF Bay Area between 1973 and 1990. These data show a gradual improvement in five-year survival over time, with the largest gains for men diagnosed in the late 1980s. Indeed, five-year survival from prostate cancer remained between 70 percent-75 percent until 1986, then rose to 94 percent by 1990, a statistically significant 24 percent increase in just four years. This improvement in survival was probably due to a combination of the increased use of screening measures and improved treatment techniques[5].

Age at Diagnosis

Prior studies have demonstrated that survival from prostate cancer may vary with age at diagnosis (6). Changes over time in five-year relative survival by age are shown in Figure V-2 and Table V-2. For most of the 18-year period from 1973 through 1990, men 75 years of age and older had poorer five-year survival from their prostate cancer than younger men. In addition, before 1986, relative survival remained fairly constant for all

Figure V–1: Five-Year Relative Survival Rates by Year of Diagnosis, San Francisco Bay Area, 1973-1990



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CHANGES OVER TIME IN FIVE-YEAR RELATIVE SURVIVAL, 1973-1990

Figure V–2: Five-Year Relative Survival Rates by Age and Year of Diagnosis, San Francisco Bay Area, 1973-1990

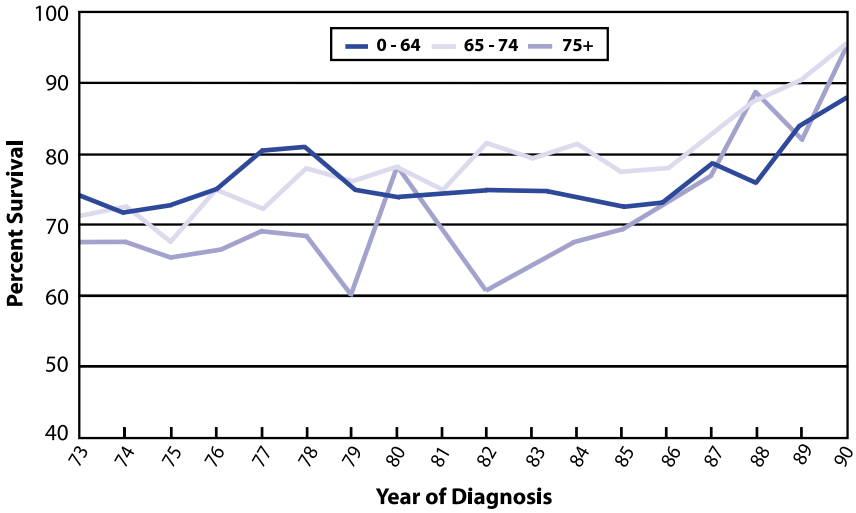

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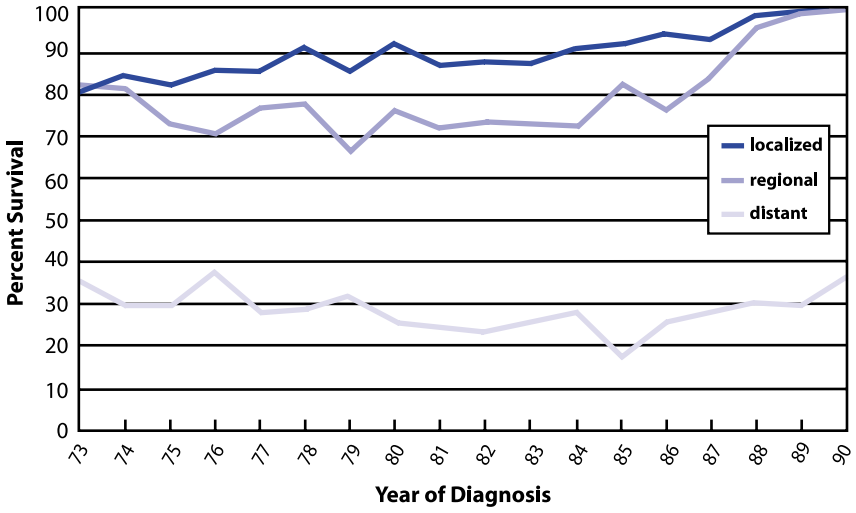
Figure V–3: Five-Year Relative Survival Rates by Stage and Year of Diagnosis, San Francisco Bay Area, 1973-1990



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CHANGES OVER TIME IN FIVE-YEAR RELATIVE SURVIVAL, 1973-1990

Table V-2: Prostate Cancer Five-Year Relative Survival Rates by Age and Year of Diagnosis, San Francisco Bay Area, 1973-1990.

Year of Diagnosis	0-64			65-74			75+		
	Count[1]	Rate	95% CI	Count[1]	Rate	95% CI	Count[1]	Rate	95% CI
1973	196	73.9	66.3-81.4	267	71.5	63.7-79.2	275	67.5	56.8-78.1
1974	192	71.6	64.0-79.3	290	72.5	65.1-79.9	303	67.5	57.3-77.6
1975	197	72.8	65.3-80.3	335	67.5	60.6-74.4	313	65.3	55.6-74.9
1976	196	75.0	67.6-82.4	318	75.0	68.1-81.9	324	66.2	56.5-75.9
1977	197	80.2	73.1-87.2	369	72.4	65.9-78.9	321	68.9	59.3-78.6
1978	197	80.8	73.8-87.8	329	78.1	71.3-84.9	325	68.3	59.0-77.6
1979	210	75.0	67.9-82.1	338	76.2	69.5-83.0	368	59.9	51.1-68.7
1980	199	74.0	66.6-81.4	368	78.1	71.6-84.5	351	78.3	68.9-87.8
1981	195	74.4	66.9-81.9	370	75.1	68.7-81.6	388	68.8	60.1-77.5
1982	190	75.1	67.6-82.7	378	81.5	75.2-87.7	373	60.7	52.1-69.3
1983	221	74.7	67.7-81.7	418	79.3	73.3-85.3	384	63.7	55.0-72.3
1984	223	73.8	66.7-80.8	452	81.4	75.7-87.1	432	67.6	59.4-75.8
1985	224	72.6	65.5-79.7	494	77.6	72.1-83.1	458	69.3	61.3-77.4
1986	186	73.5	65.7-81.2	488	77.8	72.3-83.3	449	73.2	65.2-81.1
1987	262	78.5	72.2-84.7	566	83.1	78.0-88.1	525	76.8	69.3-84.3
1988	275	75.9	69.7-82.2	626	87.7	83.0-92.4	536	89.1	81.5-96.7
1989	270	84.0	78.1-89.8	666	90.4	86.0-94.8	569	81.8	74.6-89.1
1990	319	87.6	81.9-93.2	756	95.4	90.9-99.8	587	95.2	87.5-100.0

[1] Count refers to the number of cases alive at the start of the observation period.

Prepared by California Department of Health Services, Cancer Surveillance Section.

Table V-3: Prostate Cancer Five-Year Relative Survival Rates by Stage and Year of Diagnosis, San Francisco Bay Area, 1973-1990.

Year of Diagnosis	Localized			Regional			Distant		
	Count[1]	Rate	95% CI	Count[1]	Rate	95% CI	Count[1]	Rate	95% CI
1973	260	80.9	72.3-89.6	85	82.2	68.2-96.1	88	34.8	22.1-47.4
1974	445	84.3	77.8-90.8	112	81.3	69.2-93.4	161	29.7	20.8-38.6
1975	426	82.1	75.5-88.7	193	73.1	63.8-82.5	167	29.7	21.1-38.3
1976	440	85.2	78.8-91.7	182	70.4	60.5-80.3	158	37.3	27.6-46.9
1977	543	85.5	79.8-91.3	170	76.2	65.9-86.5	143	27.6	18.8-36.4
1978	485	91.4	85.5-97.2	174	77.6	67.7-87.5	169	28.5	20.1-36.9
1979	528	85.2	79.4-91.0	197	66.2	56.4-76.0	179	31.8	23.1-40.4
1980	579	92.3	86.9-97.7	130	75.9	64.0-87.8	187	25.7	17.9-33.6
1981	623	86.7	81.3-92.1	127	71.9	59.8-84.0	175	24.3	16.4-32.2
1982	593	87.5	82.0-92.9	139	73.3	61.9-84.6	179	23.1	15.4-30.8
1983	623	87.1	81.8-92.4	126	72.4	60.5-84.2	198	24.9	17.4-32.4
1984	672	90.7	85.7-95.8	140	72.2	61.4-83.0	204	27.9	20.2-35.7
1985	646	91.9	86.7-97.1	185	82.4	73.3-91.6	240	16.8	11.0-22.6
1986	613	94.3	89.1-99.5	182	75.8	66.1-85.4	210	25.7	18.4-33.0
1987	734	92.9	88.1-97.7	226	83.8	75.6-92.0	221	27.7	20.3-35.1
1988	822	98.7	94.3-100.0	250	95.7	88.4-100.0	236	30.3	22.9-37.6
1989	790	100.0	98.5-100.0	284	98.8	92.3-100.0	265	29.8	22.8-36.8
1990	919	100.0	100.0-100.0	364	100.0	93.6-100.0	224	36.1	27.1-45.1

[1] Count refers to the number of cases alive at the start of the observation period.

Prepared by California Department of Health Services, Cancer Surveillance Section.

men, regardless of age at diagnosis. Between 1986 and 1990, however, five-year relative survival increased 19 percent for men under 65 years, 23 percent for men aged 65-74, and 37 percent for men aged 75 years and older at the time of their diagnosis, all statistically significant improvements. These data show that improvements in prostate cancer survival in recent years in the SF Bay Area occurred in men of all ages, although five-year survival for men 75 years and older remained poorer than for younger men.

Stage of Disease

As shown in Figure V-3 and Table V-3, five-year relative survival has differed considerably by tumor stage of disease at diagnosis. Most striking is the significantly poorer survival for men diagnosed with distant stage prostate cancer than for men diagnosed with local or regional disease. However, there have been some improvements over time in relative survival within stage. For men diagnosed with local stage prostate cancer, five-year relative survival improved gradually throughout the entire period, with 81 percent of men diagnosed in 1973 surviving their disease for at least five years and 100 percent

CHANGES OVER TIME IN FIVE-YEAR RELATIVE SURVIVAL, 1973-1990

Figure V–4: Five-Year Relative Survival Rates by Grade and Year of Diagnosis, San Francisco Bay Area, 1973-1990

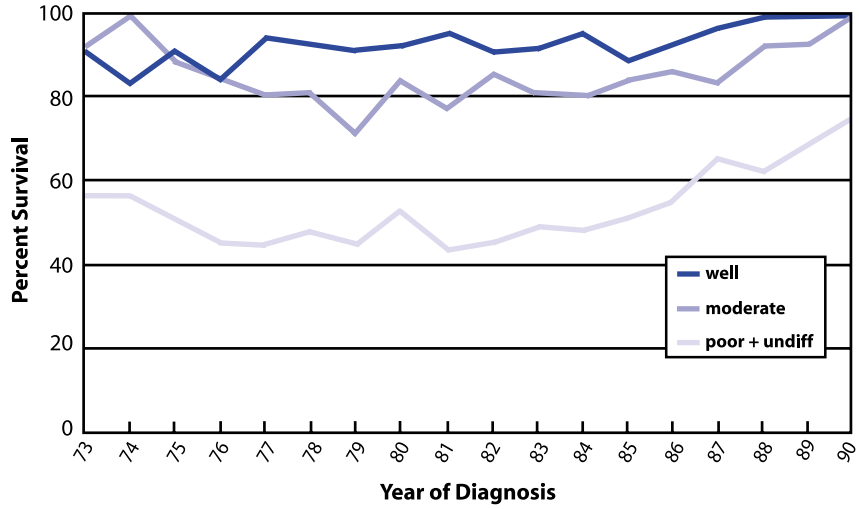

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Table V-4: Prostate Cancer Five-Year Relative Survival Rates by Grade and Year of Diagnosis, San Francisco Bay Area, 1973-1990.

	Well Differentiated			Moderately Differentiated			Poorly + Undifferentiated		
Year of Diagnosis	Count[1]	Rate	95% CI	Count[1]	Rate	95% CI	Count[1]	Rate	95% CI
1973	161	91.2	81.2-100.0	37	92.2	71.6-100.0	174	56.7	46.2-67.2
1974	169	83.8	73.6-93.9	38	100.0	84.0-100.0	252	56.2	47.4-65.1
1975	218	91.1	83.0-99.2	43	88.7	69.3-100.0	301	50.8	43.2-58.5
1976	249	84.4	76.2-92.5	177	84.6	74.3-94.9	167	45.3	35.2-55.3
1977	252	94.5	87.0-100.0	211	80.7	71.7-89.6	172	44.8	34.9-54.7
1978	272	93.2	85.5-100.0	244	81.1	72.8-89.5	165	47.9	37.6-57.9
1979	308	91.6	84.3-98.9	240	71.4	62.5-80.4	203	44.6	35.6-53.6
1980	273	92.2	84.7-99.7	251	84.2	75.6-92.8	226	52.7	43.6-61.7
1981	247	95.6	87.5-100.0	267	77.4	69.0-85.8	214	43.3	34.4-52.1
1982	243	91.1	82.7-99.5	261	85.5	77.5-93.5	214	45.4	36.5-54.3
1983	235	92.1	83.9-100.0	285	81.2	73.4-89.1	237	48.6	40.1-57.2
1984	282	95.6	88.1-100.0	348	80.0	72.7-87.2	272	48.3	40.3-56.2
1985	288	89.2	81.5-96.9	417	83.6	77.1-90.2	333	51.2	43.9-58.6
1986	256	92.6	84.4-100.0	426	86.6	80.4-92.9	349	55.1	47.9-62.3
1987	336	97.0	90.3-100.0	519	83.6	77.8-89.4	385	65.4	58.5-72.4
1988	339	99.7	93.0-100.0	625	92.5	87.4-97.6	360	62.3	55.1-69.5
1989	301	100.0	98.9-100.0	702	92.8	88.1-97.6	391	68.1	61.1-75.0
1990	351	100.0	100.0-100.0	857	99.0	94.5-100.0	391	74.7	67.2-82.1

[1]Count refers to the number of cases alive at the start of the observation period.

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of men diagnosed in 1990 surviving at least that long. For men diagnosed with regional prostate cancer, changes in survival did not occur until the mid-1980s but increased rapidly after that point, such that 100 percent of men diagnosed in 1990 with regional disease had survived their prostate cancer for at least five years. Unfortunately, for men

CHANGES OVER TIME IN FIVE-YEAR RELATIVE SURVIVAL, 1973-1990

Table V-5: Prostate Cancer Five-Year Relative Survival Rates by Race/Ethnicity and Year of Diagnosis, San Francisco Bay Area[1] 1973-1990.

Year of Diagnosis	Count[2]	Rate	95% CI
All races combined			
1973-1978	4,944	72.1	70.2-74.1
1979-1984	5,858	73.9	72.1-75.7
1985-1990	8,256	83.1	81.6-84.6
White (non-Hispanic)			
1973-1978	3,872	72.2	69.9-74.4
1979-1984	4,494	75.5	73.4-77.5
1985-1990	6,156	85.2	83.4-86.9
Black (non-Hispanic)			
1973-1978	694	71.2	66.1-76.2
1979-1984	845	69.5	64.8-74.1
1985-1990	1,157	75.0	70.9-79.0
Hispanic			
1973-1978	218	74.5	66.0-83.0
1979-1984	268	65.5	57.5-73.6
1985-1990	477	81.0	75.1-86.9
Asian[3]			
1973-1978	158	72.3	62.5-82.1
1979-1984	246	71.5	63.6-79.4
1985-1990	455	79.4	73.5-85.2
Chinese			
1973-1978	76	65.1	49.3-80.9
1979-1984	99	64.0	50.5-77.4
1985-1990	186	86.3	76.7-95.9
Japanese			
1973-1978	-	-	-
1979-1984	22	100.0	79.2-100.0
1985-1990	53	84.3	68.5-100.0
Filipino			
1973-1978	73	76.0	63.4-88.6
1979-1984	125	71.8	61.5-82.1
1985-1990	216	72.7	64.5-81.0

[1] Data are not shown for rates based on fewer than 10 cases at the start of the fifth year of observation.
[2] Count refers to the number of cases alive at the start of the observation period.
[3] Asian refers to Chinese, Japanese, and Filipino combined.

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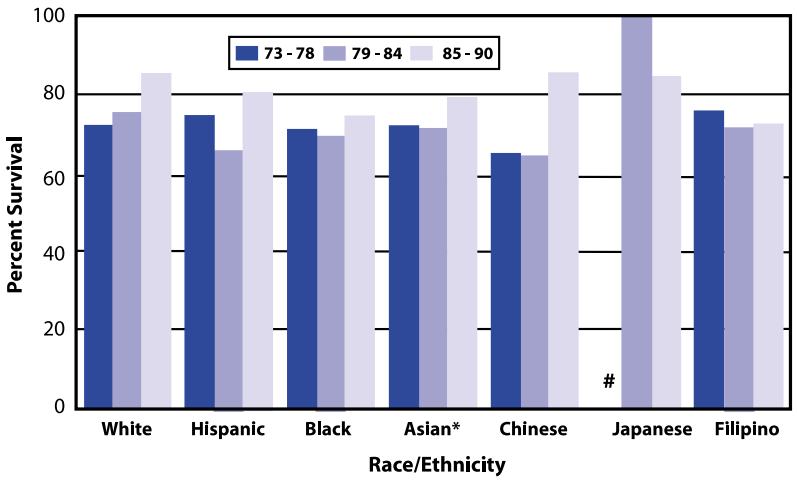
diagnosed with distant-stage disease, five-year relative survival has remained at approximately 30 percent. These statistics suggest that survival from regional stage prostate cancer was strongly affected by the introduction of screening measures and/or improved treatments in the 1980s, while the impact on survival from local and distant stage cancer was less pronounced.

CHANGES OVER TIME IN FIVE-YEAR RELATIVE SURVIVAL, 1973-1990

Figure V–5: Five-Year Relative Survival Rates by Race/Ethnicity and Year of Diagnosis, San Francisco Bay Area, 1973-1990



Data are not shown for rates based on fewer than 10 cases.
* Asian refers to Chinese, Japanese, and Filipino combined.

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Tumor Grade

Until the mid-1980s, five-year relative survival remained at approximately 50 percent for men with poorly differentiated or undifferentiated prostate cancer, compared to approximately 80 percent and 90 percent for moderately and well differentiated tumors, respectively, a statistically significant difference (Figure V-4 and Table V-4). However, survival from prostate tumors of all grades increased significantly during the mid- to late-1980s. For men diagnosed in 1990, five-year relative survival was 100 percent and 99 percent for well and moderately differentiated prostate cancer, respectively. In addition, survival from poorly differentiated and undifferentiated tumors improved from 55 percent in 1986 to 75 percent in 1990, a statistically significant 36 percent increase.

Race/Ethnicity

Figure V-5 and Table V-5 shows patterns over time in prostate cancer survival by race/ethnicity. Due to small numbers in some groups, the 18-year period has been summarized in three, six-year time periods. Within most racial/ethnic groups, men experienced an increase in prostate cancer survival over the 18-year time period. The five-year relative survival rates for the period 1985-1990 were statistically significantly higher than those for the earlier time periods among white, Hispanic and Chinese men. However, black and Filipino men did not experience similar substantial increases in survival over time.

For the first two time periods, there were no statistically significant differences in survival by race/ethnicity, although Japanese men experienced somewhat higher relative survival than men in other racial/ethnic groups during the time period 1979-1984. During the period 1985-1990, however, black and Filipino men experienced significantly worse five-year relative survival than white men. Fewer than 75 percent of black and Filipino men survived their disease for five years or more, while survival for the other racial/ethnic groups was over 80 percent during this time period.

RACIAL/ETHNIC DIFFERENCES IN PROSTATE CANCER RELATIVE SURVIVAL, 1986-1990

Table V-6: Prostate Cancer Five-Year Relative Survival Rates by Race/Ethnicity and Age at Diagnosis, San Francisco Bay Area[1] 1986-1990.

Age at Diagnosis	Count[2]	Rate	95% CI
All races combined			
0-64	1,312	80.3	77.5-83.1
65-74	3,102	87.7	85.6-89.8
75+	2,666	83.3	79.9-86.7
White (non-Hispanic)			
0-64	939	83.3	80.1-86.5
65-74	2,312	89.7	87.2-92.1
75+	2,009	84.8	80.8-88.9
Black (non-Hispanic)			
0-64	243	68.0	60.6-75.4
65-74	456	80.0	73.9-86.1
75+	297	79.8	69.7-89.8
Hispanic			
0-64	88	79.2	68.7-89.7
65-74	168	87.3	78.8-95.9
75+	157	82.3	68.9-95.7
Asian[3]			
0-64	38	84.6	70.9-98.3
65-74	161	82.3	73.8-90.7
75+	202	76.4	66.2-86.5
Chinese			
0-64	-	-	-
65-74	77	85.7	73.1-98.3
75+	75	88.3	69.7-100.0
Japanese			
0-64	-	-	-
65-74	23	90.0	70.6-100.0
75+	20	90.0	57.8-100.0
Filipino			
0-64	20	87.8	70.5-100.0
65-74	61	75.1	61.4-88.9
75+	107	67.0	54.1-80.0

[1] Data are not shown for rates based on fewer than 10 cases at the start of the fifth year of observation.
[2] Count refers to the number of cases alive at the start of the observation period.
[3] Asian refers to Chinese, Japanese, and Filipino combined.

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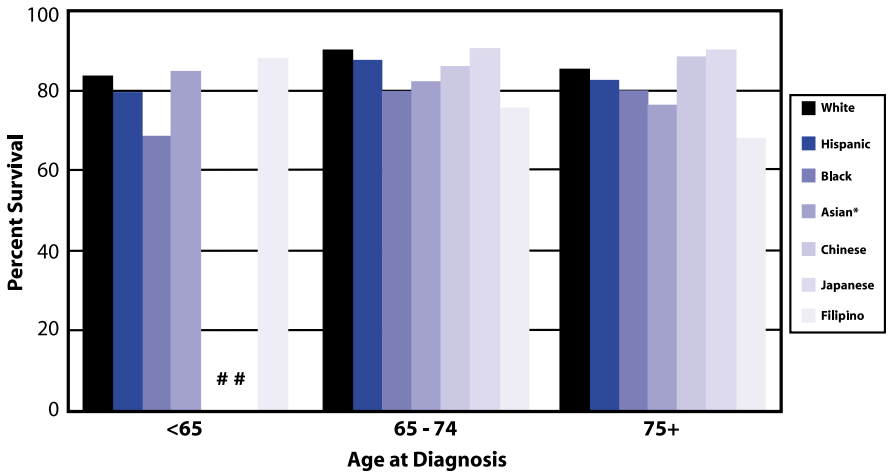
Age at Diagnosis

Figure V-6 and Table V-6 present five-year relative survival for cases diagnosed between 1986-1990 by age at diagnosis and race/ethnicity. For whites, Hispanics, and blacks, survival was highest in men aged 65-74 (90 percent, 87 percent and 80 percent, respectively) and lowest in men under the age of 65 (83 percent, 87 percent and 68 percent, respectively), although these age differences in survival were statistically significant only for white men. One possible explanation for these patterns is that men 65 years

RACIAL/ETHNIC DIFFERENCES IN PROSTATE CANCER RELATIVE SURVIVAL, 1986-1990

Figure V–6: Five-Year Relative Survival Rates by Age and Race/Ethnicity, San Francisco Bay Area, 1986-1990



Data are not shown for rates based on fewer than 10 cases.
* Asian refers to Chinese, Japanese, and Filipino combined.

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and older are more likely to be screened for prostate cancer, thus uncovering a greater proportion of either latent tumors or malignancies more responsive to therapy. Accordingly, younger men, for whom regular screening is not recommended, may be more likely to be diagnosed only after presenting with clinical symptoms consistent with more aggressive tumors that have a poorer prognosis. However, a different survival pattern was seen among Filipino men, for whom poorer survival was positively correlated with increasing age, though the differences in survival between each age group were not statistically significant.

Among men under age 65 at diagnosis, blacks experienced statistically significantly worse survival than whites; only 68 percent of young black men survived their disease for at least five years, compared to 83 percent of white men. Although Filipino men experienced the highest five-year relative survival for men under age 65 (88 percent), they had the worst survival among the racial/ethnic groups for men aged 65 and older. Notably, Filipino men aged 75 and older experienced significantly worse five-year survival than white men (67 percent vs. 85 percent, respectively).

Stage of Disease

Tumor stage at diagnosis is often used as a major prognostic factor for survival because men whose prostate cancer is diagnosed at a more distant stage are more likely to die of their disease within the first five years. It may also explain some of the observed racial/ethnic variations in survival since stage at diagnosis differs among racial/ethnic groups.

In general, prostate cancer survival in the SF Bay Area varied considerably by stage at diagnosis. Ninety-nine percent of men diagnosed with local disease and 93 percent with regional disease survived five years, compared with 30 percent of those with distant stage prostate cancer (Table V-7). However, there was some variation in survival

RACIAL/ETHNIC DIFFERENCES IN PROSTATE CANCER RELATIVE SURVIVAL, 1986-1990

Table V–7: Prostate Cancer Five-Year Relative Survival Rates by Race/Ethnicity and Stage at Diagnosis, San Francisco Bay Area,[1] 1986-1990

Stage at Diagnosis	Count[2]	Rate	95% CI
All races combined			
Localized	3,878	99.3	97.3-100.0
Regional	1,306	92.6	89.3-95.9
Distant	1,156	29.7	26.4-33.1
White (non-Hispanic)			
Localized	2,915	100.0	98.3-100.0
Regional	997	94.2	90.5-98.0
Distant	791	30.6	26.5-34.8
Black (non-Hispanic)			
Localized	518	97.5	91.9-100.0
Regional	165	79.4	69.0-89.8
Distant	211	24.2	17.0-31.5
Hispanic			
Localized	227	97.3	89.6-100.0
Regional	72	93.7	80.8-100.0
Distant	66	25.4	11.9-39.0
Asian[3]			
Localized	214	90.8	82.9-98.7
Regional	68	98.3	86.3-100.0
Distant	86	37.4	24.6-50.2
Chinese			
Localized	89	98.6	85.6-100.0
Regional	27	100.0	85.4-100.0
Distant	35	32.7	12.7-52.7
Japanese			
Localized	24	100.0	86.3-100.0
Regional	-	-	-
Distant	-	-	-
Filipino			
Localized	101	81.3	70.0-92.7
Regional	29	96.4	79.7-100.0
Distant	41	37.9	19.6-56.2

[1] Data are not shown for rates based on fewer than 10 cases at the start of the fifth year of observation.
[2] Count refers to the number of cases alive at the start of the observation period.
[3] Asian refers to Chinese, Japanese, and Filipino combined.

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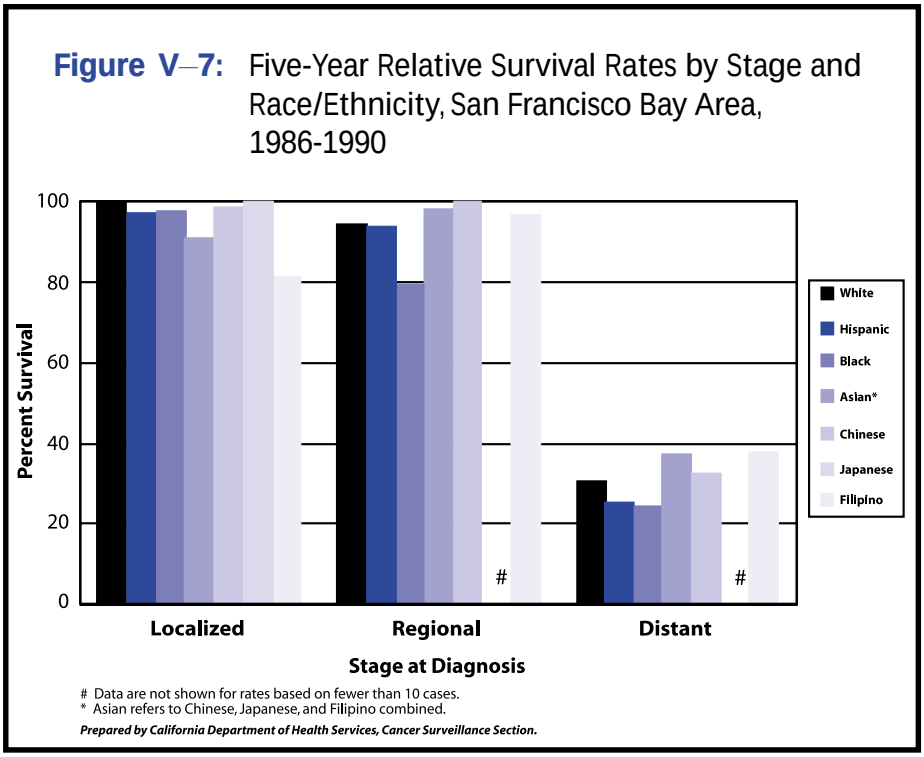
RACIAL/ETHNIC DIFFERENCES IN PROSTATE CANCER RELATIVE SURVIVAL, 1986-1990

Figure V–7: Five-Year Relative Survival Rates by Stage and Race/Ethnicity, San Francisco Bay Area, 1986-1990



Data are not shown for rates based on fewer than 10 cases.
* Asian refers to Chinese, Japanese, and Filipino combined.

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between racial/ethnic groups within stage (Figure V-7). Among those diagnosed with localized disease, Filipinos experienced considerably poorer five-year survival than the other racial/ethnic groups, with a relative survival statistically significantly lower than that for either whites or blacks (81 percent vs. 100 percent and 98 percent, respectively). Among men diagnosed with regional and distant stage disease, however, blacks experienced the poorest prognosis: only 79 percent with regional disease and 24 percent with distant disease survived their prostate cancer for at least five years, compared to 94 percent and 31 percent of whites, respectively.

Tumor Grade

Tumor grade is another prognostic factor that might influence racial/ethnic differences in prostate cancer survival. Figure V-8 and Table V-8 present racial/ethnic differences in prostate cancer relative survival within tumor grade. There were no significant differences in survival by race/ethnicity for men diagnosed with well differentiated prostate cancers, although black men had slightly worse survival than men of other racial/ethnic groups. However, for those with moderately differentiated tumors, black and Filipino men had statistically significantly worse five-year relative survival than white men (85 percent and 78 percent vs. 94 percent, respectively). While there were survival differences by race/ethnicity for men diagnosed with poorly differentiated or undifferentiated tumors, most notably that Japanese men had the best survival while Filipinos had the poorest, these differences were not statistically significant due to the small numbers of men in this grade category.

The data in Figures V-7 and V-8 illustrate that racial/ethnic differences in prostate cancer relative survival for SF Bay Area men exist even when stage at diagnosis and tumor grade are taken into account. Reasons for the remaining racial/ethnic disparities in

RACIAL/ETHNIC...

survival may include differences in concurrent diseases, dietary and other lifestyle factors, exposure to environmental risk factors, genetic predisposition, tumor stage misclassification, health care access and utilization, health behaviors, attitudes toward cancer prevention and prognosis, treatment, and other correlates of culture or socioeconomic status.[7]

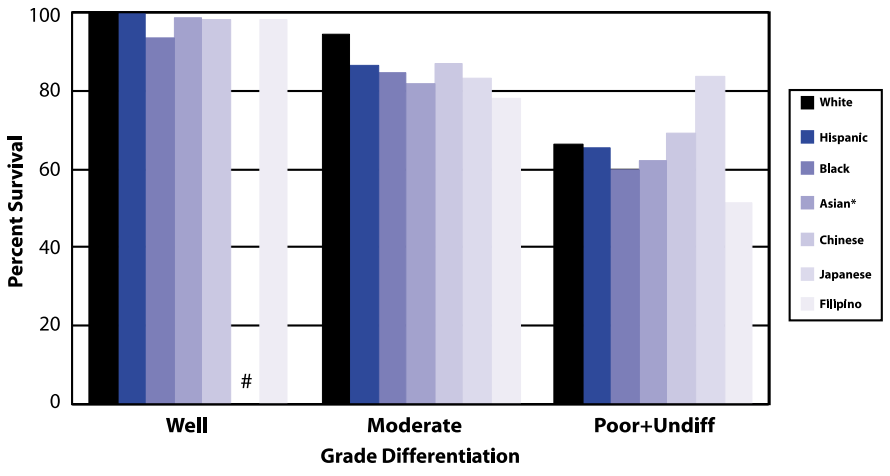
LONG-TERM TRENDS IN PROSTATE CANCER RELATIVE SURVIVAL BY RACE/ETHNICITY, 1973-1994

The risk of dying from cancer does not stop five years after diagnosis. Therefore, it is informative to examine relative survival over a longer period of time. Overall, for prostate cancer, there is an inverse relationship between long-term relative survival and extent of tumor beyond the prostate gland (stage of disease). For example, among men with localized prostate cancer, approximately 59 percent survive their disease for at least 21 years, compared to 38 percent for men with regional disease (data not shown).

Figures V-9 – V-11 and Tables V-9 – V-11 illustrate up to 21-year relative survival by stage at diagnosis for the four racial/ethnic groups: white, black, Hispanic, and Asian. Among patients diagnosed with localized disease (Figure V-9), long-term survival differed by race/ethnicity. While survival from prostate cancer was comparable for white, black and Hispanic men, Asian men had poorer survival. For example, ten-year relative survival was only 67 percent for Asian men compared to 80 percent for white men.

The most marked differences in survival between white and black men are seen for regional stage tumors (Figure V-10), for which whites consistently show a greater survival advantage, particularly 11 or more years following diagnosis. At 15 years after diagnosis, the relative survival for whites and blacks with regional stage tumors were 52 percent and 29 percent, respectively. This difference is of interest considering the

Figure V–8: Five-Year Relative Survival Rates by Grade and Race/Ethnicity, San Francisco Bay Area, 1986-1990



Data are not shown for rates based on fewer than 10 cases.
* Asian refers to Chinese, Japanese, and Filipino combined.
Prepared by California Department of Health Services, Cancer Surveillance Section.

Table V-8: Prostate Cancer Five-Year Relative Survival Rates by Race/Ethnicity and Grade at Diagnosis, San Francisco Bay Area[1] 1986-1990.

Stage at Diagnosis	Count[2]	Rate	95% CI
All races combined			
Well Differentiated	1,583	100.0	97.8-100.0
Moderately Differentiated	3,129	91.8	89.5-94.1
Poorly + Undifferentiated	1,876	65.0	61.7-68.2
White (non-Hispanic)			
Well Differentiated	1,156	100.0	98.3-100.0
Moderately Differentiated	2,386	94.2	91.6-96.9
Poorly + Undifferentiated	1,354	66.3	62.4-70.1
Black (non-Hispanic)			
Well Differentiated	238	93.6	85.3-100.0
Moderately Differentiated	401	84.5	77.8-91.2
Poorly + Undifferentiated	286	59.9	51.7-68.0
Hispanic			
Well Differentiated	93	100.0	98.1-100.0
Moderately Differentiated	170	86.4	76.9-95.9
Poorly + Undifferentiated	118	65.5	53.2-77.8
Asian[3]			
Well Differentiated	94	98.9	88.1-100.0
Moderately Differentiated	166	82.0	72.5-91.5
Poorly + Undifferentiated	116	62.2	50.2-74.3
Chinese			
Well Differentiated	49	98.0	82.0-100.0
Moderately Differentiated	58	87.1	69.5-100.0
Poorly + Undifferentiated	44	69.1	48.1-90.1
Japanese			
Well Differentiated	-	-	-
Moderately Differentiated	28	83.1	61.2-100.0
Poorly + Undifferentiated	15	83.7	53.8-100.0
Filipino			
Well Differentiated	39	98.4	83.2-100.0
Moderately Differentiated	80	78.2	65.1-91.2
Poorly + Undifferentiated	57	51.4	35.0-67.9

[1] Data are not shown for rates based on fewer than 10 cases at the start of the fifth year of observation.
[2] Count refers to the number of cases alive at the start of the observation period.
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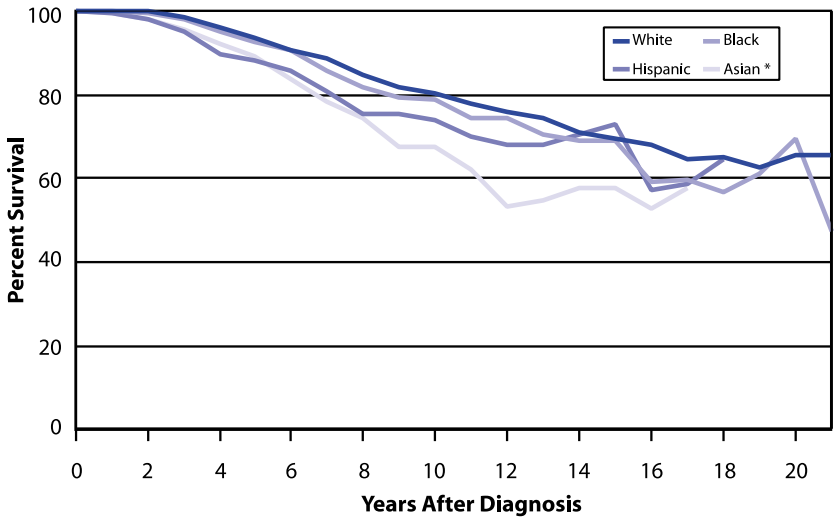
LONG-TERM TRENDS IN PROSTATE CANCER RELATIVE SURVIVAL BY RACE/ETHNICITY, 1973-1994

LONG-TERM TRENDS IN PROSTATE CANCER RELATIVE SURVIVAL BY RACE/ETHNICITY, 1973-1994

Figure V–9: Localized Prostate Cancer Long-Term Relative Survival, SFBA, 1973-1994



*Asian refers to Chinese, Japanese, and Filipino combined.

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Table V-9: Localized Prostate Cancer Relative Survival Rates by Race/Ethnicity, San Francisco Bay Area,[1] 1973-1994.

Years After Diagnosis	White (non-Hispanic)			Black (non-Hispanic)			Hispanic			Asian[3]		
	Count[2]	Rate	95% CI	Count[2]	Rate	95% CI	Count[2]	Rate	95% CI	Count[2]	Rate	95% CI
1	12,052	100.0	99.9-100.0	2,076	100.0	99.2-100.0	828	99.2	97.6-100.0	783	99.3	97.7–100.0
2	10,610	99.9	99.2-100.0	1,822	99.3	97.7-100.0	713	97.9	95.4-100.0	656	97.9	95.4-100.0
3	9,009	98.3	97.4-99.2	1,527	98.0	95.9-100.0	589	95.1	91.7-98.4	529	95.5	92.2-98.7
4	7,339	95.8	94.6-96.9	1,280	95.0	92.3-97.7	476	89.6	85.3-93.8	441	92.1	88.0-96.2
5	5,917	93.6	92.3-95.0	1,057	92.7	89.5-95.9	383	88.3	83.4-93.1	350	89.0	84.1-93.9
6	4,827	90.8	89.2-92.3	884	90.4	86.8-94.1	317	85.5	79.9-91.1	281	83.6	77.8-89.5
7	3,931	88.4	86.6-90.2	739	85.7	81.5-89.9	249	80.8	74.4-87.3	218	78.3	71.6-84.9
8	3,189	84.8	82.7-86.8	588	81.9	77.2-86.6	189	75.6	68.3-83.0	170	74.2	66.7-81.7
9	2,543	81.9	79.6-84.2	467	79.4	74.1-84.6	144	75.2	67.1-83.3	130	67.4	59.0-75.9
10	2,051	80.1	77.5-82.6	373	78.7	72.9-84.5	123	73.7	64.8-82.7	95	67.3	58.1-76.5
11	1,657	78.0	75.1-80.8	308	74.2	67.8-80.7	96	70.0	60.0-80.0	78	62.3	52.0-72.5
12	1,289	75.8	72.6-79.0	234	74.5	67.3-81.6	76	68.0	56.9-79.1	57	53.5	42.1-64.9
13	992	74.2	70.6-77.8	190	70.7	62.7-78.6	55	68.1	55.8-80.5	41	54.8	42.5-67.2
14	759	71.1	67.0-75.1	146	69.1	60.3-77.9	45	70.6	56.9-84.3	34	57.5	44.1-71.0
15	555	69.5	64.9-74.0	115	68.9	59.1-78.7	35	72.7	57.3-88.1	26	57.5	42.4-72.6
16	394	68.0	62.8-73.2	89	59.2	48.3-70.1	31	57.0	39.0-75.0	21	52.7	35.3-70.0
17	286	64.5	58.5-70.5	58	59.9	47.7-72.2	17	58.6	38.5-78.7	13	57.7	38.7-76.8
18	187	65.0	58.0-72.0	41	56.7	42.7-70.7	12	64.8	42.6-87.1	-	-	-
19	116	62.7	54.1-71.2	29	61.1	45.0-77.2	-	-	-	-	-	-
20	75	65.6	55.2-75.9	13	69.3	51.0-87.6	-	-	-	-	-	-
21	35	65.4	50.6-80.2	10	47.4	17.0-77.7	-	-	-	-	-	-

[1] Data are not shown for rates based on fewer than 10 cases at the start of the observation period.
[2] Count refers to the number of cases alive at the start of the observation period.
[3] Asian refers to Chinese, Japanese, and Filipino combined.

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LONG-TERM TRENDS IN PROSTATE CANCER RELATIVE SURVIVAL BY RACE/ETHNICITY, 1973-1994

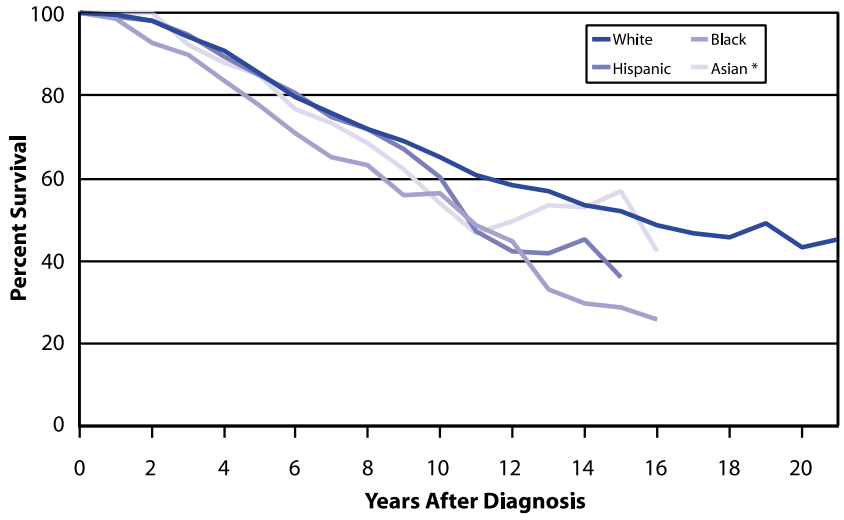
largest increase in prostate cancer incidence observed for black men diagnosed during the late 1980s and early 1990s appears to be attributable to an increase in regional stage disease.[8] These findings differ from those reported in other studies in which survival differences between white and black males with prostate cancer disappear after adjusting for stage at diagnosis.[9-10] However, a recent SF Bay Area study found that racial/ethnic differences in survival still remained even within an equal-access health care system.[11] Therefore, the survival disadvantage observed for SF Bay Area black men with regional stage prostate cancer compared to white men with similar stage of disease is likely associated with many factors. For example, there may be differences between these groups in age distribution, tumor biology, health care utilization, treatment options, and coexisting chronic diseases.

Long-term survival from distant-stage disease was generally very poor and did not differ markedly by race/ethnicity (Figure V-11). Approximately 15 percent of men survived their disease for ten years, and only 10 percent survived 18 years past their diagnosis.

EFFECTS OF SCREENING/EARLY DETECTION ON PROSTATE CANCER RELATIVE SURVIVAL

The observed trends in relative survival correspond to the increased screening and early detection activities occurring since testing for serum prostate specific antigen (PSA) became available in the late 1980s. Other procedures, particularly transurethral resection of the prostate (TURP), may also have impacted relative survival rates. It is important to consider the effects of screening when interpreting survival rates because changes in rates may be the result of artifactual increases in survival rather than actual improvements due to treatment or other factors.

Figure V–10: Regional Prostate Cancer Long-Term Relative Survival, SFBA, 1973-1994



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EFFECTS OF SCREENING/EARLY DETECTION ON PROSTATE CANCER RELATIVE SURVIVAL

Table V-10: Regional Prostate Cancer Relative Survival Rates by Race/Ethnicity, San Francisco Bay Area,[1] 1973-1994.

Years After Diagnosis	White (non-Hispanic)			Black (non-Hispanic)			Hispanic			Asian[3]		
	Count[2]	Rate	95% CI	Count[2]	Rate	95% CI	Count[2]	Rate	95% CI	Count[2]	Rate	95% CI
1	4,270	99.6	98.9-100.0	736	98.3	96.3-100.0	279	98.8	96.1-100.0	309	100.0	97.8-100.0
2	3,667	97.9	96.7-100.0	593	92.8	89.5-96.0	222	98.1	93.9-100.0	258	100.0	95.4-100.0
3	3,034	94.2	92.6-95.7	456	90.0	85.9-94.0	176	94.7	89.0-100.0	200	92.0	86.3-97.7
4	2,336	90.6	88.6-92.5	365	83.4	78.3-88.5	150	89.3	81.9-96.6	137	87.7	80.5-94.9
5	1,739	85.4	83.1-87.8	270	77.8	71.9-83.8	114	85.1	76.5-93.8	106	85.2	76.8-93.6
6	1,335	79.8	77.0-82.5	202	71.0	64.2-77.8	93	80.5	70.5-90.4	84	76.6	66.3-86.9
7	1,015	75.8	72.7-78.9	155	65.3	57.7-72.9	73	75.0	63.6-86.4	59	73.3	61.8-84.9
8	789	72.0	68.6-75.5	122	63.0	54.7-71.2	55	71.7	59.1-84.3	47	68.6	55.5-81.6
9	620	68.8	64.9-72.6	99	55.9	46.9-64.8	43	66.8	52.7-80.9	36	62.1	47.5-76.6
10	498	65.0	60.7-69.3	76	56.2	46.5-65.9	33	60.2	44.8-75.7	28	53.9	38.1-69.7
11	383	60.8	56.1-65.5	63	48.8	38.4-59.3	27	47.4	31.1-63.6	22	46.5	29.8-63.2
12	298	58.3	53.1-63.5	45	44.7	33.6-55.9	19	42.1	25.1-59.1	16	49.7	31.8-67.5
13	236	56.7	51.0-62.4	36	33.3	22.0-44.6	13	41.8	23.7-59.9	15	53.3	34.1-72.4
14	190	53.4	47.2-59.6	22	29.8	18.0-41.5	12	45.2	25.6-64.7	14	52.9	32.2-73.6
15	148	51.8	45.0-58.6	17	28.6	16.1-41.1	12	36.2	16.2-56.2	11	57.1	34.7-79.5
16	116	48.4	40.9-55.9	12	25.9	12.5-39.3	-	-	-	11	42.4	17.7-67.1
17	84	46.9	38.6-55.2	-	-	-	-	-	-	-	-	-
18	60	45.9	36.6-55.3	-	-	-	-	-	-	-	-	-
19	45	49.1	38.6-59.7	-	-	-	-	-	-	-	-	-
20	33	43.4	30.8-56.1	-	-	-	-	-	-	-	-	-
21	16	45.1	29.7-60.5	-	-	-	-	-	-	-	-	-

[1] Data are not shown for rates based on fewer than 10 cases at the start of the observation period.
[2] Count refers to the number of cases alive at the start of the observation period.
[3] Asian refers to Chinese, Japanese, and Filipino combined.

Prepared by California Department of Health Services, Cancer Surveillance Section.

Table V-11: Distant Prostate Cancer Survival Rates by Race/Ethnicity, San Francisco Bay Area,[1] 1973-1994.

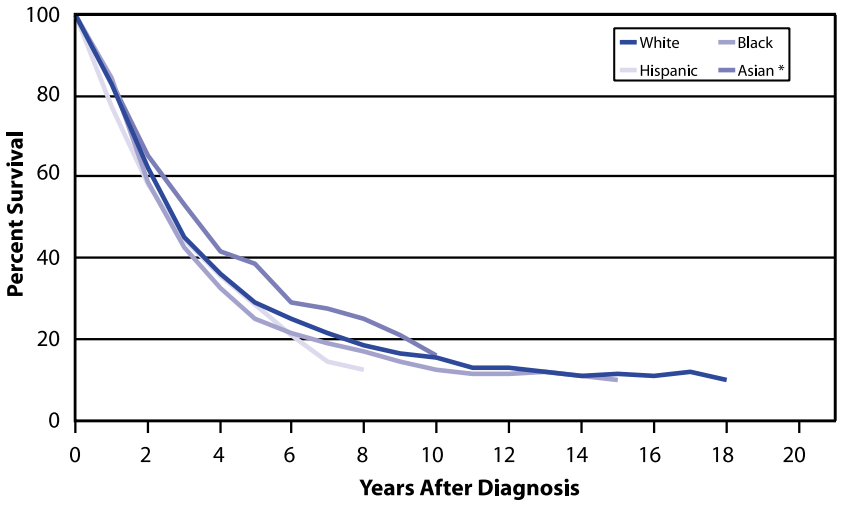
Years After Diagnosis	White (non-Hispanic)			Black (non-Hispanic)			Hispanic			Asian[3]		
	Count[2]	Rate	95% CI	Count[2]	Rate	95% CI	Count[2]	Rate	95% CI	Count[2]	Rate	95% CI
1	2,950	83.0	81.3-84.6	780	84.3	81.3-87.3	225	77.3	71.2-83.4	238	83.2	77.7-88.7
2	2,225	62.0	59.9-64.1	603	58.6	54.6-62.6	160	58.2	50.8-65.7	177	65.4	58.2-72.5
3	1,485	45.3	43.0-47.5	374	42.5	38.3-46.7	104	44.9	37.0-52.7	125	53.2	45.4-60.9
4	955	36.2	34.0-38.5	229	32.6	28.4-36.8	68	35.7	27.7-43.7	91	41.7	33.7-49.7
5	645	29.2	26.9-31.4	152	24.9	20.8-29.0	44	28.6	20.4-36.8	57	38.5	30.1-46.9
6	451	25.2	22.9-27.5	98	21.6	17.5-25.8	27	21.1	13.0-29.2	43	29.1	20.6-37.7
7	329	21.8	19.4-24.1	71	19.0	14.7-23.2	17	14.6	7.0-22.2	28	27.5	18.6-36.5
8	237	18.7	16.3-21.0	55	17.1	12.7-21.4	11	12.7	5.1-20.3	21	25.0	15.6-34.3
9	173	16.6	14.2-19.1	43	14.6	10.3-18.9	-	-	-	15	21.1	11.3-30.9
10	130	15.4	12.8-17.9	33	12.4	8.1-16.8	-	-	-	10	15.9	6.1-25.7
11	102	13.3	10.7-15.9	24	11.8	7.3-16.4	-	-	-	-	-	-
12	72	13.0	10.2-15.7	20	11.6	6.8-16.4	-	-	-	-	-	-
13	58	12.1	9.2-15.0	15	11.9	6.7-17.1	-	-	-	-	-	-
14	45	11.2	8.2-14.3	14	11.2	5.7-16.6	-	-	-	-	-	-
15	32	11.4	8.0-14.8	11	9.9	4.2-15.6	-	-	-	-	-	-
16	28	11.2	7.5-14.9	-	-	-	-	-	-	-	-	-
17	19	11.9	7.7-16.1	-	-	-	-	-	-	-	-	-
18	14	10.1	5.2-14.9	-	-	-	-	-	-	-	-	-
19	-	-	-	-	-	-	-	-	-	-	-	-
20	-	-	-	-	-	-	-	-	-	-	-	-
21	-	-	-	-	-	-	-	-	-	-	-	-

[1] Data are not shown for rates based on fewer than 10 cases at the start of the fifth year of observation.
[2] Count refers to the number of cases alive at the start of the observation period.
[3] Asian refers to Chinese, Japanese, and Filipino combined.

Prepared by California Department of Health Services, Cancer Surveillance Section.

Early detection of tumors may impact survival in one of several ways. Screening may detect malignant tumors at early stages, which are more responsive to effective therapy, thereby leading to increased disease-free survival. Alternatively, screening may allow the malignant tumor to be detected at an earlier point in time compared to when the cancer would have become clinically detectable, thus moving the date of diagnosis to an earlier date. This may result in lead time bias, where the time period between diagnosis and death is longer for tumors detected by screening than for tumors identified by clinical examination. Screening may also be more likely to detect slowly growing,

Figure V–11: Distant Prostate Cancer Long-Term Relative Survival, SFBA, 1973-1994



*Asian refers to Chinese, Japanese, and Filipino combined.

Prepared by California Department of Health Services, Cancer Surveillance Section.

EFFECTS OF SCREENING/EARLY DETECTION ON PROSTATE CANCER RELATIVE SURVIVAL

otherwise asymptomatic tumors which may exist for a longer period of time prior to clinical symptoms compared to faster growing tumors. This effect, known as length time bias, can artificially increase survival rates by overrepresenting men who have slower growing tumors.[12-13] Finally, screening may also be associated with a special type of length time bias called over-diagnosis. In this situation, screening detects non-lethal tumors that are subsequently treated and "cured," even though the patient would not have died of prostate cancer. Relative survival rates are thus inflated, but no reduction in disease-specific mortality is achieved. Though the exact nature of screening effects on relative survival (i.e., real or artifactual) cannot be determined from these data, these issues must be considered when interpreting survival trends.

SUMMARY

In general, survival from prostate cancer has increased over time, with the most dramatic improvements in survival from regional stage disease; however, there have not been similar increases in survival for men diagnosed with distant stage disease. In addition, black and Filipino men are less likely to survive their prostate cancer for five years than men of other racial/ethnic groups, and these survival differences are not completely explained by differences in stage of disease at diagnosis or tumor grade. Long-term survival from prostate cancer differs by both stage of disease and race/ethnicity. Asians have poorer survival than other men with localized disease, while black men are less likely to survive regional disease. The advent of screening tests such as PSA has affected prostate cancer survival in unknown ways, and may be influencing some of the racial/ethnic variations presented here. These findings underscore the importance of studying potentially modifiable socioeconomic and cultural determinants impacting survival from this common cancer.

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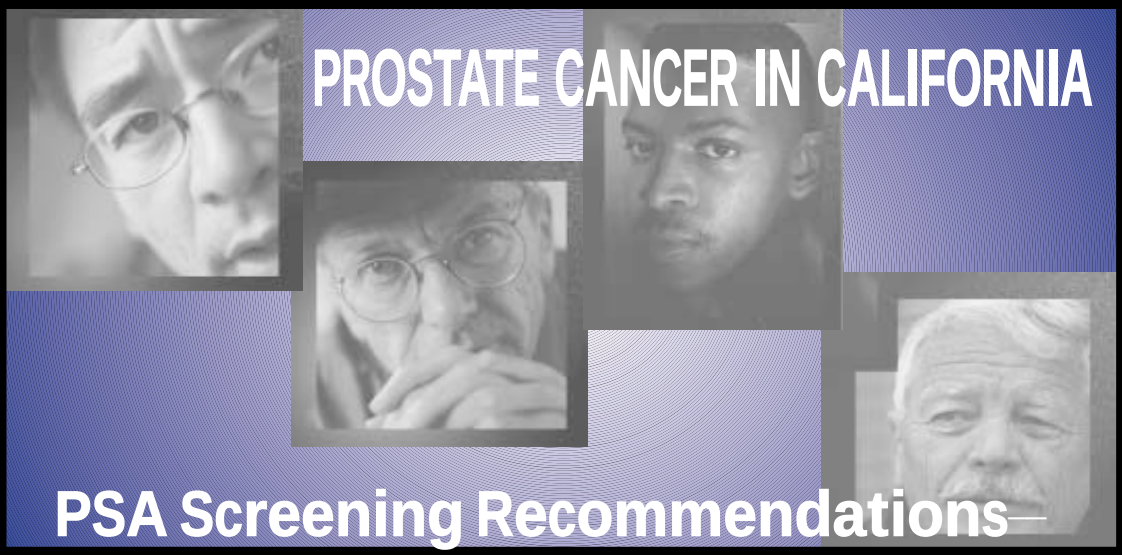

PSA Screening Recommendations—Why Experts Disagree

John Morgan, Dr.P.H.,[1-2] Arnold Vang, M.P.H.[2] and Wilan Wong, M.P.H.[2]

[1] Desert Sierra Cancer Surveillance Program, Loma Linda, CA
[2] Loma Linda University School of Public Health

CHAPTER VI

BACKGROUND

This chapter reviews the medical literature regarding prostate-specific antigen (PSA) testing and the impact of PSA testing on four prostate cancer outcomes: incidence, stage at diagnosis, relative survival, and mortality. PSA testing recommendations for five organizations are provided and are related to the four outcomes listed, identify ing reasons for disagreement. Additionally, this chapter reports estimates of PSA testing penetration in California during 1993 and 1995 and relates these findings to trends in prostate cancer outcomes.

THE PROSTATE-SPECIFIC ANTIGEN (PSA) TEST

Since its introduction in 1986, the prostate-specific antigen (PSA) test has become the most frequently used blood test for cancer detection.[1] The PSA test measures blood levels of a serine protease antigen that is specific for prostate epithelial tissue.[2] Because PSA is specific to prostate epithelia, circulating levels form an index of prostate glandular cell activity. PSA levels can be elevated among men with benign prostatic hypertrophy/hyperplasia (BPH),[2] trauma to the prostate,[3-4] prostatitis,[2,5] and prostate malignancies. Other factors that may influence circulating PSA levels include digital rectal exam,[6] recent ejaculation,[7] and palpation of the prostate,[8] although there remains some uncertainty regarding whether any of these characteristics significantly alter blood PSA levels, particularly among older men.[9] In spite of the lack of specificity of the PSA test for prostate cancer, a total PSA level of greater than 4 ng/mL has gained acceptance as sufficient to raise suspicion about the existence of a prostate malignancy[2] and frequently serves as a stimulus for follow-up testing.

Of the four categories of PSA (free, bound, complexed, and total), only total[10] and free[11] PSA measurements are currently used in clinical settings. The distinction between PSA subtypes holds some promise for separating malignancies from other conditions that elevate PSA and possibly for assessment of disease progression, although the precise role of PSA subtyping in relation to prostate cancer detection is currently under investigation.[12]

Use of the ratio of free-PSA to total-PSA may hold promise for distinguishing between prostate malignancies and other conditions that elevate total PSA level.[13] A number of investigations have demonstrated improvement in specificity of prostate cancer detection when the ratio of free to total PSA is used in place of total PSA.[13-17] In spite of optimism regarding the benefits that might be realized by adopting the free-PSA test, the added cost and complexity involved in measuring free and total PSA challenges their combined use in screening for latent disease. Other assessments that may hold some promise for distinguishing between the reasons for elevated PSA levels or determination of disease severity include: PSA level;[5] trend in PSA levels measured in serial testing;[5, 18] PSA level divided by prostate volume determined using transrectal ultrasound (PSAd);[2, 4] PSA level combined with findings from digital rectal exam (DRE),[19] and assessment of PSA level in conjunction with other clinical or diagnostic findings.

Prostate cancer is defined as a malignant neoplasm arising in the prostate gland and is classified using the second edition of the International Classification of Diseases for Oncology (ICD-O-2) coding manual using the anatomic site designation of C61.9.[20] Although an array of morphologic subtypes of prostate cancer exist, the overwhelming majority of cases are adenocarcinomas arising in the androgen sensitive, glandular acinar cells.[21] Prostate cancer is characterized by multifocal lesions. Nearly two-thirds of prostate malignancies are clinically localized and are confined to the prostate at the time of diagnosis.[22] Most prostate cancer cases exhibit either symptoms of lower urinary obstruction or no symptoms at the time of diagnosis.[23]

PSA TESTING RECOMMENDATIONS

The spectrum of prostate cancer cases ranges from quiescent, slow growing tumors that can remain confined to the prostate gland for decades, to rapidly growing malignancies that have the propensity for invasion of tissue adjacent to the prostate and metastasis. Metastatic prostate cancer has a predilection for bone, although metastases can be disseminated to any body organ.

Currently, there are conflicting recommendations regarding PSA testing among asymptomatic men. Among these, recommendations formed by the American Urological Association, Inc. (AUA)[27] and the American Cancer Society (ACS)[28] propose annual PSA testing among asymptomatic men beginning at age 50 or younger. The American College of Radiology (ACR) has adopted the ACS recommendations, while the United States Preventive Services Task Force (USPS-TF)[29] and the National Cancer Institute (NCI)[30] recommend against PSA testing among asymptomatic men. Specific recommendations from the AUA, ACS, ACR, USPS-TF, and NCI follow.

Recommendation of the American Urological Association, Inc. - Revised: January 1995[27]

"Annual digital rectal examination (DRE) and serum prostatic specific antigen (PSA) measurement substantially increase the early detection of prostate cancer. These tests are most appropriate for male patients 50 years of age or older and for those 40 or older who are at high risk, including those of African American descent and those with a family history of prostate cancer. Patients in these age/risk groups should be given information about these tests and should be given the option to participate in screening or early detection programs. PSA testing should continue in a healthy male who has a life expectancy of 10 years or more."

Recommendation of the American Cancer Society (ACS) - Revised: June 10, 1997[28]

The ACS recommendation has also been adopted by the American College of Radiology

"The ACS recommends that both the PSA test and the digital rectal examination be offered annually, beginning at age 50, to men who have a life-expectancy of at least 10 years and to younger men who are at high risk... ." "Men who choose to undergo screening should begin at age 50 years. However, men in high risk groups, such as those with a strong familial predisposition (e.g., two or more affected first-degree relatives), or African Americans may begin at a younger age (e.g., 45 years). More data on the precise age to start prostate cancer screening are needed for men at high risk... ." "No direct evidence exists to date to show that PSA screening decreases prostate cancer mortality rates. Indirect evidence, however, suggests that prostate cancer screening has resulted in the diagnosis of early-stage disease in more younger men, which could influence mortality."

Recommendation from the Report of the U.S. Preventive Services Task Force (USPS-TF)

Guide to Clinical Preventive Services, 2nd Edition - 1996 [29]

"Routine screening for prostate cancer with digital rectal examinations, serum tumor markers (e.g., prostate-specific antigen), or transrectal ultrasound is not recommended. ...There is fair evidence to support the recommendation that the condition (PSA-screening) be excluded from consideration in a periodic health examination. Support for these recommendations is based on "Evidence obtained from at least one properly randomized controlled trial...Evidence obtained from well-designed cohort or case-control analytic studies, preferably from more than one center or research group...Opinions of respected authorities, based on clinical experience; descriptive studies and case reports; or reports of expert committees."

PSA TESTING RECOMMENDATIONS

Summary Statement of National Cancer Institute (NCI) regarding PSA testing - January 1998[30]

"There is insufficient evidence to establish whether a decrease in mortality from prostate cancer occurs with screening by digital rectal examination, transrectal ultrasound, or serum markers including prostate-specific antigen."

Each of the organizations making recommendations about screening for prostate cancer using PSA testing base their positions on scientific evidence and the opinions of individual experts and expert panels, while nearly opposite positions are evident.

WHY EXPERTS DISAGREE ON PSA TESTING

There are at least three related, yet distinctly different rationales for PSA testing. One involves use of PSA testing among men displaying palpable prostate nodules, BPH, prostatitis, difficulty urinating, or other conditions that may relate to prostate health. Here the PSA test is used in a group of symptomatic men to facilitate identification of their specific disease condition. Additionally, serial PSA testing can be used in an attempt to monitor disease progression among men displaying localized prostate cancer.[31]

Screening, on the other hand, is defined as "application of tests or procedures to a group of asymptomatic people in order to identify those who likely have the disease that is the object of the screening."[32] Some of the controversy regarding use of the PSA test has resulted from a failure to distinguish between use for differential diagnosis, follow-up of prostate cancer cases, or for screening. Additionally, differences in the outcome used to evaluate PSA testing have widened the controversy regarding successful use of the test. In this chapter, we consider four measures that can be used to assess the benefits of PSA testing. These include changes in prostate cancer incidence, prostate cancer stage at diagnosis or extent of disease at diagnosis, relative survival among prostate cancer cases, and prostate cancer mortality.

Prostate Cancer Incidence: Among the prostate cancer outcomes that can be measured, there is general agreement that the increased use of the PSA test that began in the early 1990s resulted in substantial increases in detection of prostate cancer,[22,33-37] producing a sharp increase in reported incidence. In an assessment of the age-adjusted prostate cancer incidence in California, Mills and Cohen identified a 70 percent increase in incidence between 1988 and 1992, followed by a somewhat less precipitous decline that approached the 1988 risk by 1995.[38]

The moderate rise in prostate cancer incidence observed during the late 1980s is attributed to availability of pathologic specimens associated with widened use of transurethral resection of the prostate (TURP) and other procedures used to treat or diagnose BPH.[39-41] The sharp rise in prostate cancer risk from 1990-1992 is the result of expanded PSA testing that detects early-stage cases that were previously missed.[1,34,42-43]

Various theories have been proposed in an attempt to explain the decline in prostate cancer incidence in the United States and in California after 1992. Among these, Merrill, *et al* speculated that the decline may have resulted from a fall in popularity of PSA testing after 1992,[33] while Gann contemplated that the incidence drop may have been produced by a depletion of undetected prevalent cases found using PSA screening.[1]

We assessed data from the Behavioral Risk Factor Survey (BRFS)[44-45] conducted in California during 1993 and repeated in 1995. The survey methods used in the BRFS ensure equal numbers of participants in various demographic categories, including age, sex,

WHY EXPERTS DISAGREE ON PSA TESTING

and race/ethnicity during the 1993 and 1995 assessments. The proportion of men who reported ever having had a PSA test was significantly higher in 1995 than in 1993. The proportion of men who reported having had a recent PSA test was slightly higher in 1995 than in 1993, but the increase was not statistically significant. Collectively, these findings indicate that there was not a decline in the popularity of PSA testing in California between 1993 and 1995.

The fall in prostate cancer incidence among California men after 1992[38] is most likely the result of PSA testing. This conclusion is supported by the data's showing a continuation of high PSA testing penetration among California men from 1993 to 1995. Taken together, the incidence decline in California after 1992 and maintenance of high PSA testing penetration between 1993 and 1995 provide convincing evidence that regular PSA screening[1,44,46] during the early 1990s, depleted the number of latent prostate malignancies among California men during subsequent years. This depletion of cases results from detection that is earlier in the natural history of disease progression (lead-time). The lead-time resulting from earlier detection of many prostate cancer cases acted to initially increase incidence during the early 1990s, as a result of improved case finding, with a subsequent decline in detection of new cases observed as the pool of prevalent cases was depleted (after 1992) among men receiving regular testing.[47]

Prostate Cancer Stage at Diagnosis: Recent studies assessing trends in the proportion of prostate cancer cases diagnosed at various stages in the progression of prostate cancer have confirmed the belief that growth in popularity of PSA testing has been accompanied by detection of more earlier-stage cases.[28,46] In 1986, approximately 53 percent of prostate cancer cases were diagnosed with localized disease. By 1996, localized disease cases represented 74 percent of the newly diagnosed cancer cases.[48] Recommendations made by the ACS, ACR, and AUA rely substantially on demonstration of a larger fraction of cases diagnosed with localized disease when evaluating the success of PSA screening for prostate cancer.

Use of the growing number of early-stage prostate cancer cases as an indicator of the success of PSA testing has a significant limitation. An undeterminable number of early-stage prostate cancer cases are indolent; many would never become clinically important if not diagnosed.[49-50] As a result of symptoms and spread, aggressive cases were diagnosed in the past,[1,51] even without PSA testing. Use of PSA test findings to distinguish between cases that will eventually be aggressive and those that will remain indolent may not be possible or practical in clinical practice.[50] Consequently, PSA testing among asymptomatic men ensures inclusion of indolent prostate cancer cases, many which would not have been detected without PSA testing.[49] Although the PSA test is likely responsible for the growing number of early-stage prostate cancer cases, diagnosis and treatment of cases that would never become virulent will have no impact on prostate cancer mortality and will increase the morbidity consequences of treatment.

The need to distinguish between early-stage cases that will eventually be virulent and those that will remain indolent holds even greater importance for young men diagnosed with low-grade prostate cancer. Young men diagnosed with prostate cancer will, on average, experience a longer period of time during which the tumor may spread, when compared to older men diagnosed with similar stage disease. The assertion that the PSA test has led to, "...diagnosis of early-stage disease in more younger men..."[28] only represents success if early diagnosis eventually yields a reduction in mortality or morbidity. The dilemma surrounding use of an increasing fraction of early-stage cases as an indicator of successful PSA testing is underscored by the certainty that some low-grade malignancies that would remain indolent if not detected will receive radical treatment that degrades the quality of life.

WHY EXPERTS DISAGREE ON PSA TESTING

Relative Survival Among Prostate Cancer Cases: The five-year relative survival rates (ratios) for prostate cancer among Americans increased from 68 percent in 1974-76 to 90 percent in 1986-93 for whites and from 58 percent in 1974-76 to 75 percent in 1986-93 among African Americans.[24] The capacity of PSA testing to identify low virulence cases has created a dilemma when using relative survival as a measure of the success of PSA screening. Inclusion of an increasing number of indolent cases with those that are aggressive, ensures a case mix that exhibits longer relative survival, even if there is no improvement in survival for virulent cases.[47] Additionally, improved detection of low-grade cases that were previously missed, creates lead-time bias. Collectively, the change in case mix to include a larger fraction of low-grade cases and lead-time bias have produced significant increases in relative survival for prostate cancer during the late 1980s and 1990s. Although a substantial increase in five-year relative survival for prostate cancer has been observed, this increase may not indicate any actual improvement in survival among men having aggressive disease.

Prostate Cancer Mortality: Assessment of the trend in prostate cancer mortality will provide the final measure of the success of widespread PSA testing, regardless of changes in incidence, stage at diagnosis, or relative survival. Age-adjusted risk of death from prostate cancer has been less capricious than incidence, as PSA testing has grown and sustained popularity.[52] Evaluation of age-adjusted prostate cancer mortality rates for the United States between 1973 and 1990 discloses a gradual, statistically significant rise when data for all race/ethnic groups are combined, with the suggestion of a slight decline between 1991 and 1995 that is not statistically significant.[53] In the chapter on prostate cancer incidence in California in this volume, Nasseri describes a similar upward trend in the age-adjusted risk of death for prostate cancer between 1970 and 1991 for all race/ethnic groups combined.[52] The California data further disclose a statistically significant, albeit slight, decline in the age-adjusted risk of death from prostate cancer between 1991 and 1996[52] that is concomitant to the high penetration of PSA testing.

CONCLUSION/ DISCUSSION

The cost of PSA screening and treatment of prostate cancer in the U.S. has been estimated. This estimate assumed acceptance of the ACS recommendation of annual testing of all United States men between 50 and 75 years of age (more than 21 million men in 1992). The conclusion was that adherence to this practice would cost an estimated $12.7 billion each year and would have profound morbidity consequences with uncertain benefits regarding mortality.[54] The authors of the assessment suggest caution regarding use of PSA testing among asymptomatic men 50-75 years of age in the absence of evidence showing a reduction in risk of death. A contrasting assessment presented by McLaren argues that watchful waiting too often leads to disease progression.[31]

When attempting to predict future health needs for London in the early 1800s, William Farr noted that, "*the death rate is a fact; anything beyond this is an inference*." Gann noted that increases in incidence of early-stage prostate cancer that accompanied use of procedures that detected more early-stage cases during the late 1980s had no detectable impact on risk of death from prostate cancer in the early 1990s.[1] He further predicted that it may take a decade before data are available that will allow complete evaluation of whether the increased use of PSA testing will result in a change in prostate cancer mortality that is sufficient to justify the monetary and morbidity costs associated with testing. The most recently-reported mortality data from the SEER program (1973-1995) [53] suggest the presence of a slight downturn in prostate cancer mortality concomitant with the rise and subsequent fall in prostate cancer incidence. The California data [25,52] include one additional year and provide more convincing evidence of a declining trend in prostate cancer mortality.

CONCLUSION/ DISCUSSION

During the next decade, nearly 400,000 United States men will succumb to prostate cancer. It is certain that failure to aggressively treat some prostate cancer cases will result in premature deaths. Alternatively, failure to treat indolent cases that are never detected will reduce the frequency of iatrogenic morbidity outcomes with no impact on mortality. The dilemma surrounding assessment of the costs versus benefits of widespread PSA testing results from the equivocal meaning of trends in prostate cancer incidence, stage at diagnosis, relative survival, and the inevitable lag-time inherent to measurement of mortality changes. The age-adjusted prostate cancer mortality rates calculated in population-based cancer registries will ultimately measure the success of PSA testing, as we strive to follow Hippocrates advice to *primum non nocere,* or first do no harm.

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Risk of Developing Prostate Cancer by Race/Ethnicity, 1991-1995

Cyllene R. Morris, D.V.M., Ph.D.[1]

[1] Cancer Surveillance Section, California Department of Health Services, Sacramento, CA

CHAPTER VII

INTRODUCTION

Prostate cancer is the most commonly diagnosed cancer among California men, accounting for 27.4 percent of all cancers diagnosed and 12.1 percent of all cancer-related deaths statewide in 1995.[1] Data from the California Cancer Registry (CCR) show that prostate cancer incidence and mortality rates vary markedly by race/ethnicity. Accordingly, it is important that men of different race/ethnic groups be informed about their projected risks of being diagnosed with this life-threatening malignancy. In this report, current estimates of the risk of being diagnosed with prostate cancer in California are presented for men of four different race/ethnicities: Asian/Pacific Islander(PI) (non-Hispanic), black (non-Hispanic), Hispanic, and white (non-Hispanic). The unique diversity of California's population allows for stable estimates of risk for these race-ethnic groups. Risk estimates for white and black men reported by the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) Program[2] are also presented to facilitate comparisons. The SEER Program covers approximately ten percent of the US population and has reported cancer statistics for these two race/ethnic groups only.

METHODS

In this report, the terms "risk of developing cancer" and the "risk of being diagnosed with cancer" are used interchangeably. Two types of risk estimates are presented: interval and cumulative (including lifetime) risk. Interval risk is defined as the probability that a cancer-free man of a certain age will develop prostate cancer within a specified number of years. Cumulative risk is defined as the probability that a newborn male will be diagnosed with prostate cancer by a certain age, while lifetime risk is the probability that this newborn male will eventually be diagnosed with prostate cancer.

The risk of developing cancer was calculated as a function of statewide prostate cancer incidence rates and non-cancer-related mortality rates from 1991 through 1995. Age-specific incidence rates (five-year intervals) and non-cancer related mortality rates were calculated for each race/ethnicity. Incidence rates were based on the number of invasive primary prostate cancers diagnosed in California from 1991 to 1995 and reported to the CCR as of January 1998. Mortality data were obtained from the Center for Health Statistics, California Department of Health Services. Age- and race-specific population estimates were obtained from the Demographic Research Unit, California Department of Finance. Risk estimates were based on the life table methodology proposed by Feuer et al.[3, 4] During each five-year age interval, men in a hypothetical cohort were considered to be at risk for two mutually exclusive events: developing prostate cancer or dying of other causes, prostate cancer-free. Incidence and mortality rates were used to derive probabilities of developing prostate cancer or dying of other causes during each interval. These probabilities were then applied to the hypothetical cohort to obtain the expected number of men developing prostate cancer or dying of other causes during each age-interval. All men surviving the interval cancer-free became the population at risk at the beginning of the next age-interval.

The risk of developing prostate cancer within a particular age-interval was estimated as the number of expected new cancers developing during that interval divided by the population at risk (cancer-free) at the beginning of that period. The cumulative risk was estimated as the sum of all cancers up to a specified age divided by the initial population, and the lifetime risk was the sum of all cancers in the life table divided by the birth cohort.

RESULTS

Cumulative Risk

Table VII-1 and Figure VII-1 show the estimated risk of developing prostate cancer from birth to a specified age by race/ethnicity. Before age 40, the accumulated risk was still close to zero, but by age 70, 1 in every 16 men is expected to have been diagnosed with prostate cancer. Variations in cumulative risk by race/ethnicity were evident in all age groups (Figure VII-1). The estimated risk for black men up to age 75 was higher than the risk for men in any other race/ethnic groups. For example, by age 65, 1 in every 20 black males is expected to have been diagnosed with prostate cancer. In contrast, the projected risks accumulated by 65 years of age for men of other race/ethnic groups were 1 in 30 for white, 1 in 50 for Hispanic, and 1 in 91 for Asian/PI men. The risk accumulated by age 80 was similar for white and black males (one in six). However, regardless of age, Asian/PI men had the lowest cumulative risk of developing prostate cancer.

Lifetime Risk

Based on current data, a newborn male in California will have a 16.6 percent (one in six) probability of being diagnosed with prostate cancer during his lifetime (Table VII-1 and Figure VII-2). The estimated lifetime risk was highest for black males (18.1 percent) followed by risks for white (17.2 percent), Hispanic (15.2 percent), and Asian/PI males (12.3 percent). These percentages correspond to one in 5.5, one in 5.8, one in 6.6, and one in 8.1 men (black, white, Hispanic and Asian/PI, respectively), developing prostate cancer over a lifetime.

Compared to California, lifetime risk estimates from the SEER Program were slightly lower for white men (16.6 percent versus 17.2 percent), but were identical for black men (18.1 percent). Some of the difference may be attributed to the SEER Program's policy of including persons of Hispanic ethnicity within the white race category,[2] versus CCR's mutually exclusive Hispanic and non-Hispanic white categories. Because the incidence of prostate cancer is lower among Hispanic men, risk estimates for white plus Hispanic men are likely to be lower than estimates for white non-Hispanic men.

Table VII-1: Cumulative Risk From Birth of Being Diagnosed with Prostate Cancer, by Age and Race/Ethnicity[1], California, 1991-1995

Birth to Age	All Races	Asian/PI	Black	Hispanic	White
	one in:	one in:	one in:	one in:	one in:
40	52,632	71,429	34,483	76,923	43,478
45	7,634	33,333	4,065	11,765	6,494
50	1,258	4,310	673	2,364	1,072
55	265	833	143	471	232
60	80	249	45	135	72
65	33	91	20	50	30
70	16	35	12	22	15
75	10	19	8	13	10
80	8	13	7	10	7
85	7	10	6	8	6
Lifetime	6	8	6	7	6

[1] Race/ethnicity categories are mutually exclusive. Persons of Hispanic ethnicity are identified by medical records and/or surname, and may be of any race.

Prepared by California Department of Health Services, Cancer Surveillance Section.

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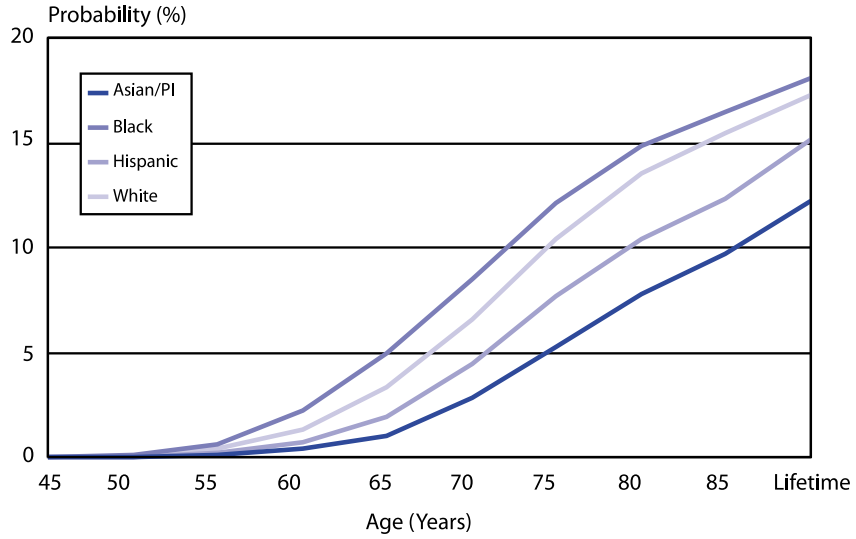
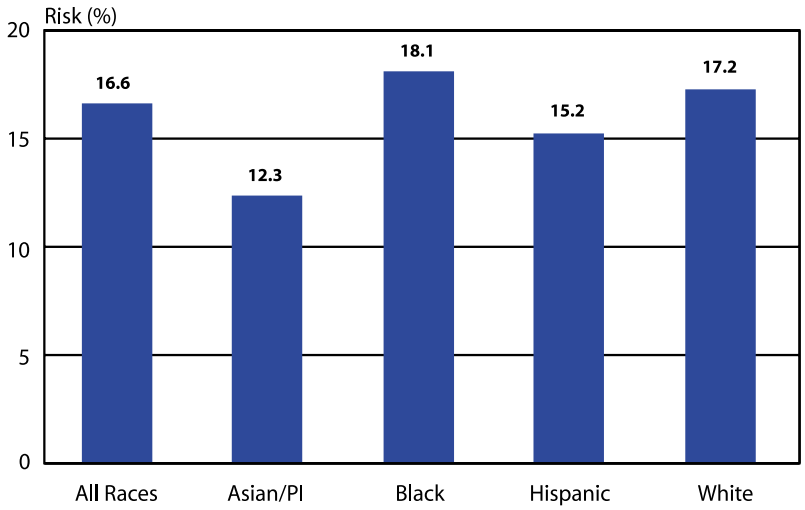
Figure VII–1: Cumulative Probability, From Birth, of Being Diagnosed With Prostate Cancer by Race/Ethnicity, California, 1991-1995



Prepared by California Department of Health Services, Cancer Surveillance Section.

Figure VII–2: Lifetime Risk of Being Diagnosed With Prostate Cancer by Race/Ethnicity, California, 1991-1995



Prepared by California Department of Health Services, Cancer Surveillance Section.

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Table VII-2 Risk (percent) of Being Diagnosed with Prostate Cancer Within the Next 10, 20 Years or in Remaining Lifetime[1], by Race/Ethnicity[2], California and SEER[3] Estimates, 1991-1995

Current Age	+10 years		+20 years		Eventually	
(cancer free)	CA	SEER	CA	SEER	CA	SEER
All Races						
0	< 0.1	< 0.1	< 0.1	< 0.1	16.6	17
40	0.1	0.1	1.3	1.8	17.6	18.1
50	1.3	1.8	6.7	7.9	18.3	18.8
60	6	6.8	14	14.8	18.7	18.8
70	10.5	-	-	-	16.5	-
Asian/Pacific Islander						
0	< 0.1	-	< 0.1	-	12.3	-
40	< 0.1	-	0.4	-	12.7	-
50	0.4	-	3	-	12.9	-
60	2.8	-	8.2	-	13.3	-
70	6.4	-	-	-	12.3	-
Black						
0	< 0.1	< 0.1	< 0.1	< 0.1	18.1	18.1
40	0.2	0.3	2.5	3.2	20.5	20.8
50	2.6	3.3	10.4	11.7	22.4	22.8
60	9.4	10.5	19	20.4	23.9	24.4
70	14.8	-	-	-	22.2	-
Hispanic						
0	< 0.1	-	< 0.1	-	15.2	-
40	< 0.1	-	0.8	-	16.2	-
50	0.8	-	5	-	16.8	-
60	4.5	-	11.6	-	17.3	-
70	8.8	-	-	-	15.8	-
White						
0	<0.1	< 0.1	< 0.1	< 0.1	17.2	16.6
40	0.1	0.1	1.5	1.7	18.2	17.5
50	1.4	1.7	7.2	7.6	18.9	18
60	6.4	6.5	14.7	14.2	19.2	17.9
70	10.9	-	-	-	16.8	-

[1] Lifetime risk is the probability that a newborn (current age = 0) will eventually be diagnosed with prostate cancer.

[2] Race/ethnicity categories are mutually exclusive. Persons of Hispanic ethnicity are identified by medical records and/or surname, and may be of any race.

[3] SEER: National Cancer Institute's Surveillance, Epidemiology and End Results Program, which covers approximately 10% of the US population. SEER risk estimates are available for white or black race/ethnic groups only.

Source: California Cancer Registry (01/98) and SEER Cancer Statistics Review, 1973-1995 (2).

Prepared by California Department of Health Services, Cancer Surveillance Section.

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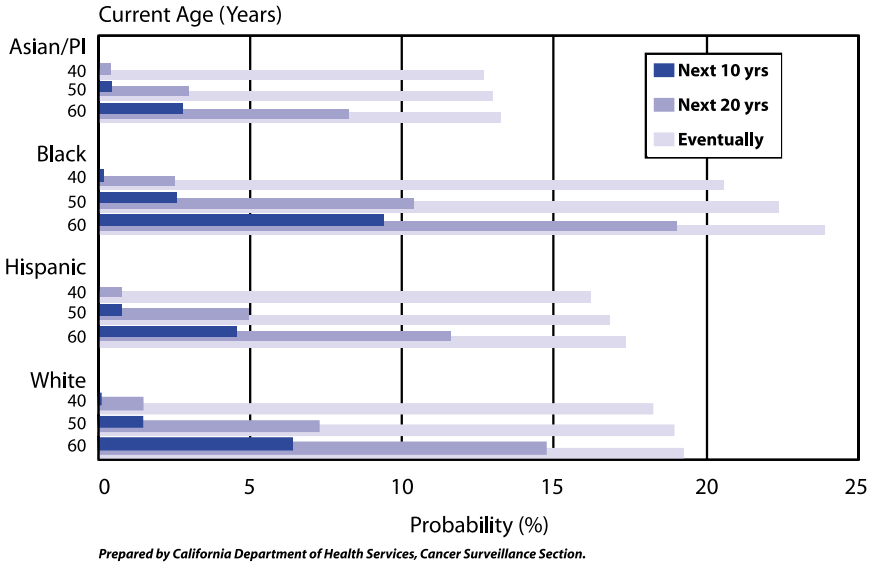
Figure VII–3: Probability of Being Diagnosed With Prostate Cancer in the Next 10 Years, 20 Years, or in Remaining Lifetime by Race/Ethnicity and Current Age, California, 1991-1995



Prepared by California Department of Health Services, Cancer Surveillance Section.

Interval Risk

Estimates of the risk of developing prostate cancer within the next 10 years, 20 years, or during the remaining lifetime, by current age, are displayed in Table VII-2 and Figure VII-3. The older the man, the higher the probability of being diagnosed in the next 10 to 20 years, reflecting the dramatic increase in prostate cancer incidence with age. Variations in risk by race/ethnicity were similar to what was described above for cumulative/lifetime risk. Regardless of their current age, black males were at highest risk of being diagnosed with prostate cancer. For example, the probabilities for a black man currently 50 years old and cancer-free of developing prostate cancer within the next 10 or 20 years were 2.6 percent and 10.4 percent, respectively. The probabilities for an Asian/PI man of the same age would be substantially lower, specifically 0.4 percent within the next ten years and 3.0 percent within the next 20 years. The risk during the remaining lifetime for men of all race/ethnicities was highest at age 60.

COMMENTS AND CAUTIONS ON INTERPRETATION

Risk estimates presented in this report are based on population data and do not take into account individual risk factors other than age and race/ethnicity. Clearly, these two factors alone are not sufficient to predict the risk of developing cancer for any individual man. In addition, because risk estimates are future projections based on current

COMMENTS AND CAUTIONS ON INTERPRETATION

rates, the underlying, and questionable, assumption is that these rates will remain constant. In reality, incidence and mortality rates are likely to change over time. Improvements in diagnostic methods and changes in population level risk factors both contribute to increased or decreased cancer incidence rates. Mortality rates may also be expected to change over time, which again may substantially impact risk estimates. Because most cancers are age-dependent, a longer life span increases the likelihood of being diagnosed with prostate cancer. The lifetime risk of prostate cancer is, therefore, particularly influenced by improved mortality rates or increased detection of asymptomatic cancers.[5] Another potential limitation of the use of lifetime risk for communicating risk of developing cancer is that, albeit powerful and appealing, such a statistic has been frequently misinterpreted. For example, one common misconception is that risk estimates assume that a person will live to a specific age, when in fact the calculations take into account the likelihood of dying of other causes at any age.[3] It should also be noted that lifetime risk is applicable only to newborns, and not to persons of all ages. For all these reasons, interval risk estimates, based on current age, may be less prone to misconceptions, more accurate and possibly more relevant to the general public.

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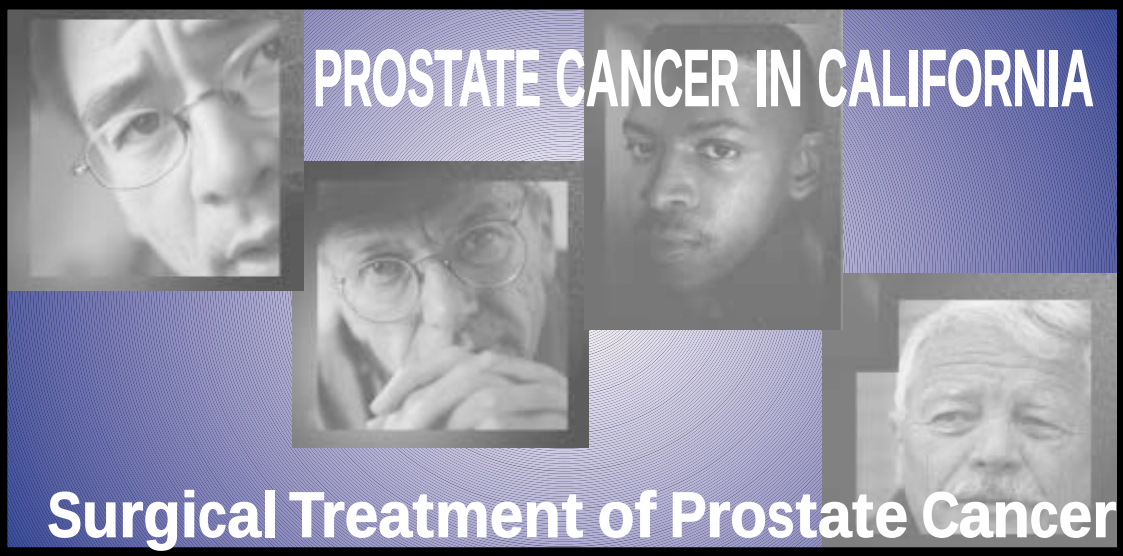

Surgical Treatment of Prostate Cancer in California, 1994-1995

Sandy L. Kwong, M.P.H.[1]

[1] Cancer Surveillance Section, California Department of Health Services, Sacramento, CA

CHAPTER VIII

INTRODUCTION

The optimal treatment of prostate cancer has been a source of controversy and intense debate. The difficulty in resolving this debate has been due in part to the nature of prostate cancer. If diagnosed early, one treatment option for localized prostate cancer is observation ("watchful waiting").[1] Relatively few untreated men with localized prostate cancer die from the disease within five years of diagnosis, and only a minority die within ten years.[2] Because prostate cancer tends to occur in elderly men, competing comorbidities have significant impact on treatment plans. Treatment of cancer is dictated by the stage of the disease, patient's age, and overall health. Surgery is reserved for men in good health who are under the age of 70. Asymptomatic patients of either advanced age and/or with other comorbidities may be advised to delay treatment and consider observation only, especially patients with early stage tumor.[1] Radiation therapy is also a common treatment for prostate cancer. However, data for radiation is not analyzed in this chapter because data regarding radiation are not complete at the California Cancer Registry (CCR).

A study performed by the National Cancer Database has found a trend toward treatment with curative intent rather than management of the disease.[3] Similar conclusions of more aggressive treatment also have been found with National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) Program data.[4] In California, the introduction of prostate-specific antigen (PSA) screening test in the late 1980s led to an increase in prostate cancer incidence, which peaked in 1992. The influence of PSA testing has not only affected incidence of prostate cancer but also the patterns of clinical presentation and treatment of this disease. This chapter will examine the most recent data for surgical treatment for prostate cancer in California men. Surgery is a common treatment of cancer of the prostate with the aim to remove the cancer.

Radical prostatectomy consists of the removal of the prostate and some of the surrounding tissue. Prostatectomies are only performed if the cancer has not yet spread outside of the prostate. Transurethral resection of the prostate (TURP) is the removal of the cancer with a small wire loop inserted through the urethra. TURP is performed to relieve symptoms caused by the tumor in men who cannot have radical prostatectomy due to advanced age or illness.[1]

METHODS

For this analysis, prostate cancer-directed surgery was categorized as prostatectomy (simple and radical), TURP, other surgery (includes cystoprostatectomy, radical cystectomy, pelvic exenteration, and surgery of regional and/or distant sites/lymph nodes), and no surgery. Data for cancer surgery codes has been classified utilizing the surgery codes used by the American College of Surgeons.[5] Data for summary stage were converted from Extent of Disease (EOD) codes to summary stage. Race/ethnicity for cases was grouped into four mutually exclusive categories of non-Hispanic white, non-Hispanic black, Hispanic, and non-Hispanic Asian/Pacific Islander. Hispanic ethnicity was based on information on surname in the medical record. Persons with race coded as white, black, or unknown, but with a last name on the 1980 U.S. census list of 12,497 Hispanic surnames were categorized as Hispanic for these analyses.[6]

Prostate cancer cases presented in this chapter were diagnosed in California men from 1994-1995 and reported to the California Cancer Registry as of January 1998. The CCR is considered to have complete statewide coverage, and details of its operation have been published elsewhere.[6,7] Since 1988, the CCR has collected statewide information on cancer-directed surgical procedures performed as part of the first course of treatment for men with prostate cancer; however, this chapter focuses on patterns of surgical treatment in California from 1994-1995. Data on surgery have been limited to the two most

METHODS

recent years because of changes in staging methodology for cancers. Beginning with cancers diagnosed in 1994, the CCR began staging per the SEER Extent of Disease codes instead of the SEER summary stage used from 1988-1993. Throughout this chapter, information is presented by race/ethnicity, age at diagnosis (0-59, 60-69, 70-79, and 80+), and summary stage at diagnosis.

RESULTS

In 1994 and 1995 combined, 35,581 cases of invasive prostate cancers were diagnosed among California men. Of these cases, 55 (0.2 percent) were excluded from analysis because of unknown age. Additionally, another 382 (1.1 percent) cases were reported to CCR by autopsy or death certificate only and therefore were also excluded. A total of 34,756 cases with known age and valid stage information were analyzed for this chapter.

Overall, prostatectomy was the most common surgical procedure for men with prostate cancer diagnosed in 1994 and 1995. A total of 11,389 (32.7 percent) men received a prostatectomy as first course of treatment. TURP was performed in 4,135 (11.9 percent) men with prostate cancer. Only 1,060 (1.3 percent) men received alternative forms of surgery, other than prostatectomy or TURP, as their first course of treatment. Prostate cancer diagnosed at the regional stage had the highest percentage of surgery (71.7 percent). The proportion of cases that were treated with surgery was lower for localized, remote and unstaged prostate cancers at 52.4 percent, 18.0 percent and 15.6 percent, respectively. During this two-year period, more than half of all the men diagnosed with prostate cancer (n=18,172), did not receive surgery as their first course of treatment (Table VIII-1).

During this two-year period, there were racial/ethnic differences in utilization of surgery for treatment of prostate cancer (Table VIII-2). For all stages combined, the proportion of black men receiving surgery was lower than any other race/ethnic group (p-values < 0.001) when compared to either non-Hispanic whites, Hispanics, and Asian/Pacific Islanders. There were no other significant differences in surgical treatment among the three other race/ethnic groups. When examined by type of surgery, black and Asian/Pacific Islander men were less likely to receive a prostatectomy as first course of treatment than non-Hispanic white men (p-values < 0.001). No significant differences were observed in prostatectomy utilization between non-Hispanic white men and Hispanic men. Black men also were significantly less likely to receive TURP than white men (p-value = 0.006). However, Hispanic and Asian/Pacific Islander men were significantly more likely to receive TURP than white men (p-values < 0.001).

Table VIII–1: Surgical Procedures During the First Course of Treatment of Prostate Cancer, by Stage, California,1994-1995

	Prostatectomy		TURP[1]		Other Surgery		No Surgery/ Unknown		Total
Stage at Diagnosis	No.	%	No.	%	No.	%	No.	%	No.
Localized	9,751	37.3	3,314	12.7	653	2.5	12,447	47.0	26,165
Regional	1,354	51.6	256	9.7	272	10.4	744	28.3	2,626
Direct Extension	1,008	8.9	219	11.3	65	3.3	652	33.4	1,944
Lymph Nodes	223	2.0	23	4.6	189	38.0	63	12.6	498
Extension & Nodes	123	1.1	14	7.6	18	9.8	29	15.8	184
Remote	23	1.0	330	14.7	51	2.3	1,843	80.9	2,247
Unstaged	261	7.0	235	6.3	84	2.3	3,138	76.4	3,718
Total	11,389	32.7	4,135	11.9	1,060	1.3	18,172	52.3	34,756

[1] TURP: Transurethral resection of the prostate
Source: California Cancer Registry (01/98)

Prepared by California Department of Health Services, Cancer Surveillance Section.

RESULTS

Table VIII–2: Surgical Procedures During the First Course of Treatment of Prostate Cancer, by Race/Ethnicity[1] and Stage at Diagnosis, California, 1994-1995

	Prostatectomy		TURP[2]		Other Surgery		No Surgery/ Unknown		Total
Race/Ethnicity	No.	%	No.	%	No.	%	No.	%	No. (100%)
All Stages Combined									
All Races	11,389	32.8	4,135	11.9	1,060	3.0	18,172	52.3	34,756
Asian/Pacific Islander	516	30.8	285	17.0	54	3.2	818	48.9	1,673
Black	873	29.6	312	10.6	100	3.4	1,663	56.4	2,948
Hispanic	1,260	33.8	530	14.2	115	3.1	1,818	48.8	3,723
White	8,654	35.9	2,965	12.3	782	3.2	11,712	48.6	24,113
Localized									
All Races	9,751	37.3	3,314	12.7	653	2.5	12,447	47.6	26,165
Asian/Pacific Islander	438	35.8	228	18.6	35	2.9	524	42.7	1,225
Black	763	35.7	228	10.7	60	2.8	1,085	50.8	2,136
Hispanic	1,075	39.4	420	15.4	60	2.2	1,174	43	2,729
White	7,405	39.8	2,403	12.9	494	2.7	8,281	44.6	18,583
Regional									
All Races	1,354	51.6	256	9.7	272	10.4	744	28.3	2,626
Asian/Pacific Islander	54	0.4	16	12.8	11	8.8	44	35.2	125
Black	86	36.9	26	11.2	28	12	93	39.9	233
Hispanic	157	51.3	34	11.1	40	13.1	75	24.5	306
White	1,045	54.8	180	9.4	189	9.9	492	25.8	1,906
Remote									
All Races	23	1	330	14.7	51	2.3	1,843	81	2,247
Asian/Pacific Islander	2	1.3	28	18.8	4	2.7	116	77.3	150
Black	2	0.7	37	13	8	2.8	238	83.5	285
Hispanic	1	0.4	49	18.3	2	0.7	216	80.6	268
White	17	0.2	216	14.5	36	2.4	1,220	81.9	1,489
Unstaged									
All Races	261	7	235	6.3	84	2.3	3,138	84.4	3,718
Asian/Pacific Islander	22	12.7	13	7.5	4	2.3	134	77.5	173
Black	22	7.5	21	7.1	4	1.4	247	84	294
Hispanic	27	6.4	27	6.4	13	3.1	353	84	420
White	187	8.8	166	7.8	63	2.9	1,719	80.5	2,135

[1] Race/ethnicicty categories are mutually exclusive. Persons of Hispanic ethnicity may be of any race. Men of unknown race/ ethnicity are excluded from race-specific data.
[2] TURP: Transurethral resection of the prostate
Source: California Cancer Registry (01/98)

Prepared by California Department of Health Services, Cancer Surveillance Section.

Table VIII–3: Surgical Procedures During the First Course of Treatment of Prostate Cancer, by Age and Stage at Diagnosis, California, 1994-1995

	Prostatectomy		TURP[1]		Other Surgery		No Surgery/ Unknown		Total
Age (Years)	No.	%	No.	%	No.	%	No.	%	No. (100%)
All Stages Combined									
All Ages	11,389	32.8	4,135	11.9	1,060	3.0	18,172	52.3	34,756
< 60	2,750	65.1	161	3.8	141	3.3	1,172	27.7	4,224
60-69	6,004	53.6	880	7.9	437	3.9	3,877	34.6	11,198
70-70	2,576	18.8	1,916	13.9	425	3.1	8,821	64.2	13,738
≥ 80	59	1.3	1,178	25.6	57	1.2	3,302	71.8	4,596
Localized									
All Ages	9,751	37.3	3,314	12.7	653	2.5	12,447	47.6	26,165
< 60	2,385	70.4	122	3.6	73	2.1	808	23.8	3,388
60-69	5,137	52.8	716	7.4	271	2.8	2,610	37.1	9,734
70-79	2,181	21.1	1,545	14.9	277	2.7	6,333	61.3	10,336
≥ 80	48	1.7	931	34.4	32	1.2	1,696	62.6	2,707
Regional									
All Ages	1,354	51.6	256	9.7	272	10.4	744	28.3	2,626
< 60	309	76.5	12	3	48	11.9	35	8.7	404
60-69	723	64.4	60	5.3	126	11.2	213	19	1,122
70-79	317	35.3	116	12.9	90	10.0	376	41.8	899
≥ 80	5	2.5	68	33.8	8	4.0	120	59.7	201
Remote									
All Ages	23	1	330	14.7	51	2.3	1,843	81	2,247
< 60	3	1.4	18	8.7	8	3.8	179	86.1	208
60-69	7	1.2	73	12.9	14	2.5	471	83.4	565
70-79	11	1.2	150	16.7	24	2.7	710	79.3	895
≥ 80	2	0.3	89	15.4	5	0.9	483	83.4	579
Unstaged									
All Ages	261	7.0	235	6.3	84	2.3	3,138	84.4	3,718
< 60	53	23.7	9	4.0	12	5.3	150	67.0	224
60-69	137	17.6	31	4.0	26	3.3	583	75.0	777
70-79	67	4.2	105	6.5	34	2.1	1,402	87.2	1,608
≥ 80	4	0.4	90	8.1	12	1.1	1,003	8.3	1,109

[1] TURP: Transurethral resection of the prostate
Source: California Cancer Registry (01/98)

Prepared by California Department of Health Services, Cancer Surveillance Section.

RESULTS

Among men with localized tumors, blacks and Asian/Pacific Islanders were less likely to be treated with prostatectomy than non-Hispanic white men (p-values < 0.001). There was no significant difference in prostatectomy utilization between Hispanics and non-Hispanic whites. At the regional stage, blacks and Asian/Pacific Islanders also were less likely to be treated with prostatectomy than non-Hispanic whites. There was no statistical difference between Hispanics and non-Hispanic whites receiving prostatectomies. Most men diagnosed with prostate cancer at remote stage were not surgically treated (81.0 percent). Of the cases diagnosed at the remote stage and received surgery, 81.7 percent were treated with TURP. It should be noted that 73.1 percent (n=2,717) of all unstaged prostate cancer occur in men over the age of 70, and 88.5 percent of these men do not have surgery.

Surgical treatment for prostate cancer was correlated with the age of the patient (Table VIII-3). Among all stages combined, men less than 60 years of age had the highest proportion of surgery (72.0 percent), whereas men over the age of 80 were the least likely to receive surgical treatment (28.1 percent). This likely reflects the reality that for the older patient the risk of death from a competing cause may be greater than death from prostate cancer. For men aged less than 60 and aged 60-69 who had surgery, prostatectomy was performed most frequently at 89.9 percent and 81.8 percent, respectively. For men over the age of 70, TURP was performed more frequently. By the age of 80+, 91.1 percent of all surgical procedures performed were TURP.

The median age of men who chose surgical treatment as their first course of treatment was 67.0 years. The median age did vary slightly by race/ethnic groups. For non-Hispanic whites and Hispanics, the median age was 67.0. However for blacks, the median age was at 64.0 years and for Asian/ Pacific Islanders, the median age was 69.5 years of age (Table VIII-4).

SUMMARY

During 1994-1995, there were differences in the utilization of surgery among California men with prostate cancer by race/ethnicity and age. Overall, black males were the least likely to receive surgery of any type. Blacks and Asian/Pacific Islanders were less likely to be treated with a prostatectomy than non-Hispanic white men. For all stages combined, Hispanics and Asian/Pacific Islanders were more likely to be treated with TURP than non-Hispanic whites. The occurrence of less surgery and higher mortality among black men suggests that more emphasis needs to be placed on improving prostate cancer detection in this population.

Overall, surgical treatment was strongly influenced by age. Younger men received more aggressive surgical treatment, and prostatectomies declined with age. Among men who received surgery as the first course of treatment, black men had the youngest median age

Table VIII–4: Median Age of Men who had Surgical Procedures During the First Course of Treatment of Prostate Cancer by Race/Ethnicity[1], California, 1994-1995

Race/Ethnicity	Cases	Median Age
All Races	16,686	67.0
Asian/Pacific Islander	862	69.5
Black	1,295	64.0
Hispanic	1,915	67.0
White	12,476	67.0

[1] Race/ethnicicty categories are mutually exclusive. Persons of Hispanic ethnicity may be of any race. Men of unknown race/ethnicity are excluded from race-specific data.

Source: California Cancer Registry (01/98)

Prepared by California Department of Health Services, Cancer Surveillance Section.

SUMMARY

of 64.0 while Asian/Pacific Islander men had the highest median age at 69.5. When surgical treatment was examined by stage, black and Asian/Pacific Islanders had similar experiences while Hispanics and non-Hispanic whites were similar.

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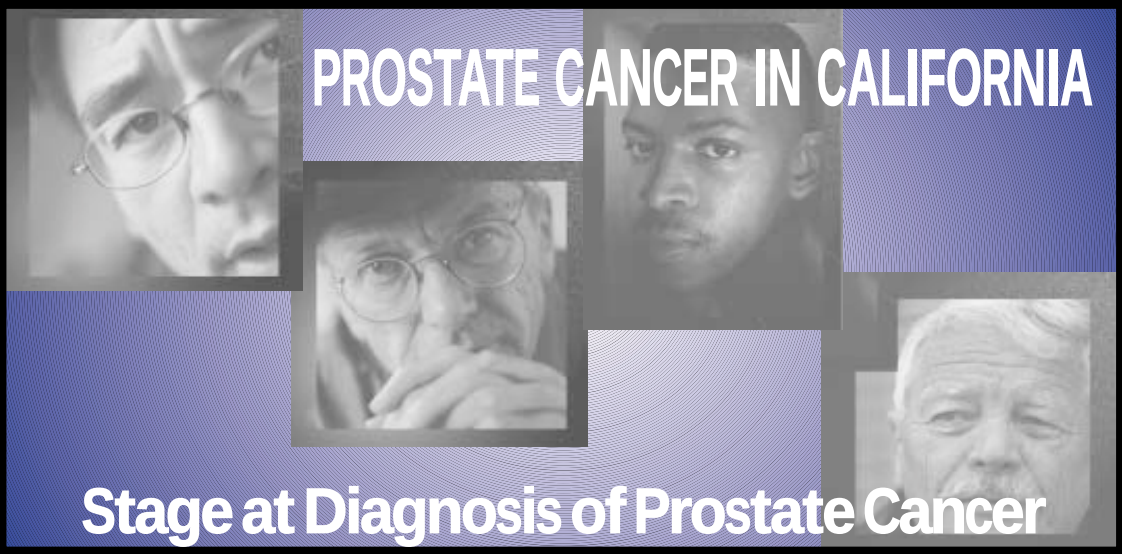

Stage at Diagnosis of Prostate Cancer in California, 1988 – 1995

Agnes Boyer-Chammard, M.D.,[1] Thomas H. Taylor, Ph.D.[1] and Hoda Anton-Culver, Ph.D.[1]

[1] Cancer Surveillance Program of Orange County, San Diego/Imperial Organization for Cancer Control, University of California Irvine

CHAPTER IX

INTRODUCTION

Prostate cancer is the most commonly diagnosed invasive cancer in California, accounting for 16,993 new diagnoses in 1995 or 27.5 percent of all cancer cases in men.[1] It is the second leading cause of cancer mortality: a total of 3,191 men died of prostate cancer in 1995 in California, accounting for 12.1 percent of cancer-related deaths and 2.5 percent of all deaths in US men. The prostate cancer mortality rate in California in 1995 was 22.1 deaths per 100,000 men.[1] Rules for staging prostate cancer (i.e., determining the extent or spread of this disease at diagnosis) have evolved over time and differ between various public health and medical organizations. There are different ways to evaluate the extension or spread of a tumor into surrounding tissues , and different coding systems have been devised for describing stage at diagnosis. Three of these coding systems are described in the next section.

The American Joint Committee on Cancer (AJCC) staging of cancer[2] is based on the Tumor Node Metastasis (TNM) classification. As the AJJC Manual for Staging of Cancer explains, the TNM system is based on the premise that cancers of similar histology (i.e., microscopic appearance) or site of origin share similar patterns of growth and extension. The size of the untreated tumor (T) increases progressively, and at some point in time regional lymph node involvement (N) occurs. Finally, distant metastases (M) occur. The AJCC manual also contains instructions for coding summaries of TNM staging: Stage 0 (*in situ*), I, II, III, IV.

The Surveillance, Epidemiology, and End Results Program of the National Cancer Institute (SEER) uses the "Extent of Disease" (EOD) code[3] which has five components: size of the tumor; extent to which the primary tumor has spread; lymph node involvement; number of nodes found positive in a pathological examination of regional lymph nodes; and number of regional nodes examined by the pathologist. In effect, the EOD is a coded descriptive summary of the tumor, including clinical as well as pathological findings and observations made during surgery. Coding must be supported by textual information entered under Diagnostic Procedures. It allows for collection of more detailed and specific information than the AJCC staging system and has been defined more consistently over time.[4] EOD was not required until 1994. Beginning with cases diagnosed January 1, 1994 Extent of Disease coding was required for all California reporting facilities.

The SEER Summary Stage System. [5] While extent of disease is a detailed description of the spread of the disease from the site of origin, stage is a grouping of cases into broad categories: in situ, localized, regional, and distant disease. The summary stage system is based upon tumor involvement indicated by all the evidence obtained from diagnostic and therapeutic procedures performed during the first course of treatment or within four months after the date of diagnosis, whichever is earlier. The time limitation ensures that the stage recorded is based on the same information that was used to plan patient treatment.

In this coding system, *in situ* describes tumors with characteristics of malignancy but which have not penetrated the basement membrane of the tissue nor extended beyond the epithelium. A localized tumor is defined as one which is malignant and invasive but confined entirely to the organ of origin. Regional neoplasms have extended beyond the organ of origin into surrounding tissues, involve regional lymph nodes, or both. Distant tumors have spread to remote parts of the body from primary site either by direct extension or by metastasis. For prostate cancer, because some patients may not undergo surgery, the diagnostic information may be limited to biopsy, radiographic, and clinical information. With surgery, more detailed histopathological information would be available. The SEER Summary Stage system was utilized by the California Cancer Registry (CCR) from 1988 to 1993. Stage at diagnosis was not required beginning with cases diagnosed January 1, 1994. Cases diagnosed prior to January 1, 1994 must continue to be staged using SEER Summary Staging.

INTRODUCTION

Table IX–1: Number of Cases and Percent Distribution of Male Prostate Cancer by Stage at Diagnosis, Race/Ethnic Group and Age at Diagnosis, California, 1988-1995

	Localized	Disease	Regional	Disease	Distant	Disease	Unknown	Disease
Age at Diagnosis	N	%	N	%	N	%	N	%
All races								
All ages	84,001	59.5	22,827	16.2	13,635	9.7	20,640	14.6
<=66	24,897	63.5	8428	21.5	3,134	8.0	2,722	7.0
67-71	20,571	63.4	6333	19.5	2,542	7.8	3,014	9.3
71-77	22,598	61.9	5368	14.7	3,325	9.1	5,221	14.3
>=78	15,935	48.4	2698	8.2	4,634	14.1	9,683	29.4
White								
All ages	65,982	61.2	17,874	16.6	9,774	9.1	14,119	13.1
<=66	18,907	65.3	6,415	22.2	2,055	7.1	1,580	5.5
67-71	16,278	65.3	5,035	20.2	1,761	7.1	1,867	7.5
71-77	18,192	63.6	4,348	15.2	2,475	8.7	3,586	12.5
>=78	12,605	49.9	2,076	8.2	3,483	13.8	7,086	28.1
Black								
All ages	5,784	54.9	1,742	16.5	1,603	15.2	1,409	13.4
<=66	2,418	59.1	847	20.7	516	12.6	313	7.7
67-71	1,319	56.6	406	17.4	346	14.8	260	11.2
71-77	1,235	54.7	319	14.1	362	16.1	340	15.1
>=78	812	43.7	170	9.2	379	20.4	496	26.7
Hispanic								
All ages	7,017	58.0	1,902	15.7	1,409	11.6	1,772	14.6
<=66	2,439	61.6	798	20.2	411	10.4	313	7.9
67-71	1,799	61.4	537	18.3	277	9.5	315	10.8
71-77	1,615	60.5	364	13.6	302	11.3	390	14.6
>=78	1,164	45.8	203	8.0	419	16.5	754	29.7
Asian								
All ages	2,964	56.7	860	16.5	677	13.0	724	13.9
<=66	661	60.9	245	22.6	110	10.1	70	6.5
67-71	721	59.9	247	20.5	130	10.8	105	8.7
71-77	851	60.3	226	16.0	150	10.6	184	13.0
>=78	731	47.9	142	9.3	287	18.8	365	23.9

Prepared by California Department of Health Services, Cancer Surveillance Section.

In this chapter, data are presented on stage at diagnosis for male prostate cancer cases diagnosed in California from 1988 to 1995. Stage at diagnosis categories used in the following analyses are based on the SEER summary stage, as described above, for the period 1988-1993. For 1994 and 1995, as the SEER summary stage was no longer required, we used the extent of disease (EOD) to evaluate and code the SEER summary stage, with the rules described below.

INTRODUCTION

Table IX–2: Number of Cases and Percent Distribution of Male Prostate Cancer by Stage at Diagnosis and by Region, California, 1988-1995

Region	Local Disease		Regional Disease		Distant Disease		Unknown Disease	
	N	%	N	%	N	%	N	%
1 Santa Clara	4,957	56.8	1,587	18.2	839	9.6	1,348	15.4
2 Central Valley	5,683	52.7	1,722	16.0	1,098	10.2	2,282	21.2
3 Sacramento	7,905	61.0	2,010	15.5	1,265	9.8	1,783	13.8
4 Tri-county	4,330	62.7	1,081	15.7	463	6.7	1,027	14.9
5 Inland Empire	8,236	62.9	1,981	15.1	1,259	9.6	1,623	12.4
6 North	4,045	52.8	1,281	16.7	742	9.7	1,592	20.8
7 San Diego	8,771	62.8	2,078	14.9	1,194	8.5	1,931	13.8
8 Bay Area	11,010	57.8	3,770	19.8	2,125	11.2	2,153	11.3
9 Los Angeles	22,836	60.4	5,637	14.9	3,739	9.9	5,583	14.8
10 Orange	6,228	61.4	1,680	16.6	911	9.0	1,318	13.0
All	84,001	59.5	22,827	16.2	13,635	9.7	20,640	14.6

Prepared by California Department of Health Services, Cancer Surveillance Section.

Beginning with cases diagnosed January 1, 1995 there were different rules for coding prostate cases. The two-month rule for assigning extent of disease codes has been changed to four months, and a new extension field has been added for coding cases which undergo prostatectomy. The codes of the EOD classification have also been modified between 1994 and 1995. In particular, cases with "invasion of prostatic capsule" (EOD=40 or 41 in 1994, EOD=32 in 1995) were coded as regional disease in 1994 and as localized disease in 1995. The same situation applied to cases with extension "into prostatic apex" (EOD=48 or 49 in 1994, EOD =31 in 1995) which were coded as regional disease in 1994 and as localized disease in 1995.

Cases with "further extension to bone, soft tissue, or other organs" (EOD=80 in 1994, EOD=70 in 1995) were coded as distant disease in 1994 but were coded in 1995 as regional disease. Cases with "extension or fixation to pelvic wall or pelvic bone" (EOD=70 in 1994, EOD=60 in 1995) are coded as regional disease in 1994 and 1995 but should be coded as distant disease. For reasons that are unclear, in 1995, all the cases with EOD 42 to 45 are coded as unstageable.

To be consistent over the period studied, we decided in this report to code invasion to prostatic capsule as localized disease (as it is done before 1994) and extension into prostatic apex as regional disease. Cases with "further extension to bone, soft tissue, or other organs" or cases with "extension or fixation to pelvic wall or pelvic bone" are coded as distant disease. Cases with EOD 42 to 45 in 1995 are recode to regional disease.

In this report, stage at diagnosis is examined by age group, by race/ethnicity, by California Cancer reporting Region (CCR), and by socioeconomic status. Trends in age adjusted incidence rates of stage at diagnosis from 1988 to 1995 are presented for all the cases, and by race/ethnicity.

Age at diagnosis was grouped into quartiles according to the distribution of the population: cases diagnosed at age less than 66, 67-71, 71-77 and 78 years and older. Cases with unknown age at diagnosis were excluded.

INTRODUCTION

Race/ethnicity includes five mutually exclusive groups: non-Hispanic white, black, Hispanic, Asian, and other or unknown race/ethnicity.

RESULTS

From 1988 to 1995, 141,347 cases of prostate cancer were diagnosed in California and reported to the CCR. There were 244 cases of *in situ* disease (0.2 percent) which were not further included in the analysis. Among the remaining 141,103 cases of invasive prostate cancer, 59.5 percent of the cases were diagnosed with a localized disease, 16.2 percent with regional disease, and 9.7 percent with distant disease. There were 20,640 cases (14.6 percent) of unknown stage at diagnosis, and 5,405 cases (3.8 percent) of other or unknown race/ethnicity. There were no differences in the proportion of unknown stage by race/ethnicity. The percentage of unknown stage increased with age: 7 percent of patients under age 66 were unstageable, 9.3 percent of patients aged 67-71, 14.3 percent of patients aged 72-77, and 29.4 percent of patients over 78 years. An explanation for this is that older patients were undergoing fewer definitive surgical procedures. This could be the result of physician recommendations for a watchful waiting approach as a reasonable option for men with shorter life expectancy. There were no important differences of unknown stage by reporting region, except region 2 (Central Valley) and region 6 (North) having higher percentage of unknowns (21 percent) but these regions also have more older cases.

BY AGE

There were differences in stage distribution by age group (Table IX-1). There was a decreasing rate of localized disease with increasing age. Cases under 71 years of age had the highest proportion of localized disease (63 percent), followed by cases aged 72-77 (61.8 percent). Cases older than 78 had the lowest percentage (48.4 percent). We found the same trend in the distribution of regional disease which represented 21.5 percent among cases under 66 years of age, 19.5 percent among cases aged 67-71, 14.7 percent among cases aged 72-77, and only 8.2 percent in cases older than 78 years of age. On the other hand, the percentage of distant disease increased with the age at diagnosis. Cases under 71 had the lowest percentage of distant disease (7.9 percent), followed by cases aged 72-77 (9.1 percent). Cases older than 78 years of age had the highest percentage of distant disease (14.1 percent).

The consistent trend towards later diagnosis as age increased (greater proportion of distant disease and decreasing proportion of localized disease) could be explained by the fact that the older cases were not detected earlier, because the screening tests (particularly PSA) were not available. For the same reason, younger cases are now detected at early stage, before clinical symptoms occur, due to the common use of screening tests.

BY RACE/ETHNICITY

There were differences in stage distribution by race/ethnicity (Table IX-1). Whites had the highest proportion of localized disease (61.2 percent), followed by Hispanics (58 percent), and Asians (56.7 percent), while blacks had the lowest percentage of local disease (54.9 percent). The distribution of regional disease was approximately 16 percent in each racial/ethnic group. There were wide differences in the distribution of distant disease among race/ethnic groups. Whites had the lowest percentage of distant disease (9.1 percent), followed by Hispanics (11.6 percent), and Asians (13.0 percent). Blacks had the highest percentage of distant disease (15.2 percent). These differences were observed in each age group and could not be explained by the differences in the age distribution between race/ethnic groups. Moreover, most of the blacks were diagnosed at a young age (38.9 percent were less than 66 year-old). The percent of distant disease among these youngest blacks cases (12.6 percent) was almost twice the percent of distant disease among the youngest whites (7.1 percent). This figure is approximately the same as the amount of distant disease among the oldest whites (13.8 percent).

BY CCR REPORTING REGION

Some variation between CCR regions was observed in stage at diagnosis for prostate cases diagnosed from 1988-1995 (Table IX-2). Region 2 (Central Valley) and 6 (North) had the lowest percentage of localized disease (52.7 percent and 52.8 percent respectively) but also the highest percentage of unknown stage (21.3 percent and 21.3 percent at diagnosis). This could be explained by the fact that in these regions, which had more older cases, detection was not followed by surgery but by a watchful waiting approach, leading to more unstageable cases. In the South, Region 5 (Inland), 7 (San Diego), 4 (Tri-county), and 10 (Orange) had the highest percentage of localized disease (62.9 percent, 62.8 percent, 62.7 percent, and 61.4 percent respectively).

For distant disease, region 2 (Central Valley) had the highest percentage (10.2 percent) and region 4 (Tri-county) the lowest (6.7 percent). This could be explained by the demographic characteristics of these areas, region 2 having more older cases and region 4 having fewer black and Asian cases than other regions.

BY SOCIOECONOMIC STATUS

The CCR data do not allow the evaluation of socioeconomic status on a state-wide basis. Instead, we report here the results of a recent work made by Delfino, et al (6) concerning socioeconomic status in Orange and San Diego Counties. One objective of the study was to determine whether there are differences in the incidence of prostate cancer between non-Hispanic white men living in census tracts of San Diego County with higher versus lower per capita incomes. The San Diego/Imperial Organization for Cancer Control database was used to access the patient's home residence in San Diego at the time of the diagnosis, which was linked to the average per capita income for the census tract of residence. Per capita income was ascertained through the patient's home address using 1990 U.S. Census tract data. White patients residing in census tracts below the 65th percentile distribution of per capita income for whites were classified as "lower income," whereas those residing in tracts at or above 65th percentile were classified as "higher income." Delfino, et al found that white men below the 65th percentile of per capita income have a significantly lower incidence of localized and regional-stage prostatic cancer and a significant higher incidence of distant-stage disease than did with higher per capita incomes. Consequently, the incidence rate ratio (defined as the ratio localized + regional/distant stage) was significantly smaller in the lower than in the higher income area (5.7 vs. 8.8, respectively).

BY YEAR OF DIAGNOSIS

The total number of prostate cancer cases increased from 12,123 in 1988 to 23,206 in 1992 and then decreased to 16,993 in 1995 (Table IX-3). As the population in California was increasing, we focused on the age adjusted incidence rate (AAIR), which also increased from 101.8 per 100,000 in 1988 to 172.5 per 100.000 in 1992 and then decreased to 121.6 per 100,000 in 1995. The percentage of localized disease increased from 55 percent in 1988 to 67 percent in 1995. The regional diseases represented 14.5 percent in 1988 and 16.2 percent in 1995. Distant disease decreased from 15.7 percent in 1988 to 6.5 percent in 1995. With the increasing use of screening tests, the initial increase in AAIR was followed by a decrease, and cases are detected over time with less advanced disease.

BY YEAR OF DIAGNOSIS

Table IX–3: Number of Cases and Percent Distribution of Male Prostate Cancer by Stage at Diagnosis, Year of Diagnosis, and by Race/Ethnicity, California 1988-1995

Year of Diagnosis	Local N	Disease %	Regional N	Disease %	Distant N	Disease %	Unknown N	Disease %
All races								
1988	6,685	55.1	1,758	14.5	1,901	15.7	1,779	14.7
1989	7,015	53.9	1,950	15.0	2,008	15.4	2,032	15.6
1990	8,528	55.0	2,477	16.0	2,025	13.1	2,469	15.9
1991	11,632	58.3	3,355	16.8	2,032	10.2	2,950	14.8
1992	14,167	61.1	3,835	16.5	1,813	7.8	3,391	14.6
1993	13,044	59.9	3,243	14.9	1,577	7.2	3,908	18.0
1994	11,430	61.7	3,456	18.7	1,174	8.3	2,476	13.4
1995	11,500	67.7	2,753	16.2	1,105	6.5	1,635	9.6
Whites								
1988	5,571	56.5	1,449	14.7	1439	14.6	1,397	14.2
1989	5,798	55.2	1,628	15.5	1502	14.3	1,571	15.0
1990	7,085	56.7	2,074	16.6	1472	11.8	1,855	14.9
1991	9,689	60.0	2,794	17.3	1526	9.4	2,154	13.3
1992	11,492	63.2	3,101	17.1	1283	7.1	2,309	12.7
1993	9,991	63.0	2,450	15.4	1045	6.6	2,378	15.0
1994	8,176	63.0	2,510	19.3	772	6.0	1,526	11.8
1995	8,180	69.8	1,868	16.0	735	6.3	929	7.9
Blacks								
1988	430	49.1	127	14.5	201	23.0	118	13.5
1989	426	46.5	115	12.5	218	23.8	158	17.2
1990	517	47.6	185	17.0	238	21.9	147	13.5
1991	667	50.3	219	16.5	212	16.0	229	17.3
1992	901	56.2	276	17.2	234	14.6	193	12.0
1993	1,008	58.8	259	15.1	211	12.3	236	13.8
1994	906	57.8	308	19.7	154	9.8	199	12.7
1995	929	64.3	253	17.5	135	9.3	129	8.9
Hispanics								
1988	442	51.3	117	13.6	178	20.7	124	14.4
1989	517	51.5	144	14.3	190	18.9	153	15.2
1990	615	53.0	141	12.1	216	18.6	189	16.3
1991	799	55.6	229	15.9	190	13.2	219	15.2
1992	1,073	58.7	296	16.2	175	9.6	284	15.5
1993	1,153	57.1	325	16.1	186	9.2	354	17.5
1994	1,193	61.9	360	18.7	134	7.0	241	12.5
1995	1,225	65.8	290	15.6	140	7.5	208	11.2
Asians								
1988	174	48.8	54	15.2	72	20.2	56	15.7
1989	213	53.5	52	13.1	85	21.4	48	12.1
1990	225	48.4	63	13.6	87	18.7	90	19.4
1991	330	56.4	85	14.5	84	14.4	86	14.7
1992	447	55.9	132	16.5	97	12.1	124	15.5
1993	524	57.2	153	16.7	98	10.7	141	15.4
1994	525	59.7	156	17.7	79	9.0	120	13.6
1995	526	63.8	165	20.0	75	9.1	59	7.2

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TRENDS IN STAGE AT DIAGNOSIS BY RACE/ETHNICITY

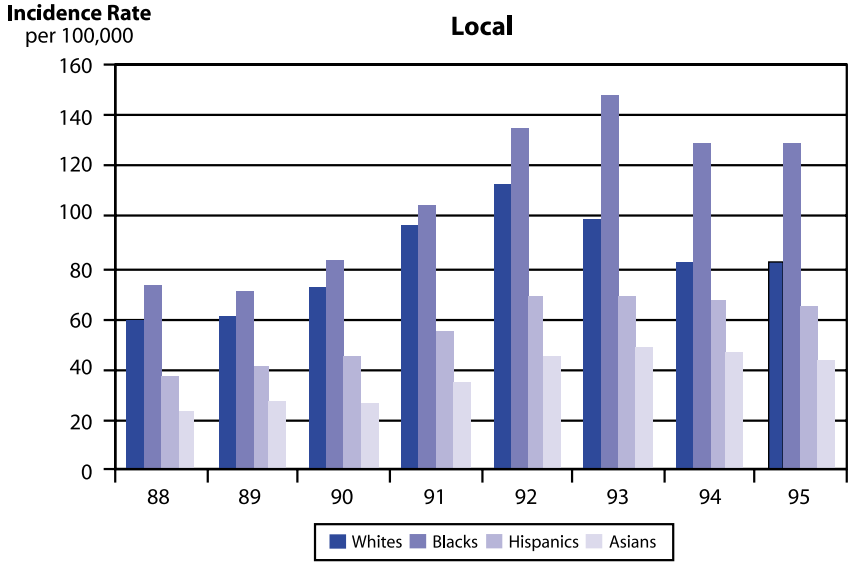
When stratified by race/ethnicity blacks always had the highest AAIR (203.8 per100,000 in 1995), followed by whites (116.8 per 100,000 in 1995), Hispanics (99 per 100,000 in 1995), and Asians (68.9 per 100,000 in 1995). Among whites, the AAIR increased from 1988 to 1992 and then decreased through 1995; among other ethnic groups, the AAIR peaked one year later, in 1993.

Local disease. Among whites and Hispanics, the AAIR increased from 1988 to 1992 and then decreased; among blacks and Asians, the AAIR peaked in 1993 (Figure IX-1). Among all race/ethnicity subgroups, the AAIR in 1995 were similar to those in 1994. The differences in AAIR between blacks and whites were larger in 1995 than in 1988.

Regional disease. The AAIR increased from 1988 to 1992 for whites and then decreased (Figure IX-2). For blacks, the AAIR increased until 1994. For Hispanics and Asians, the increase started later, in 1991. The differences in AAIR between blacks and whites are larger in 1995 than in 1988.

Distant disease. For whites, Hispanics and Asians, the AAIR decreased from 1990 to 1994 (Figure IX-3). In 1995, the rates were similar to those in 1994 and were the same for whites, Hispanics and Asians. For blacks, there was an initial increase until 1990 followed by a steady decrease until 1995. The differences in AAIR between blacks and whites are smaller in 1995 than in 1988.

Figure IX–1: Trends in Age-Adjusted Prostate Cancer Incidence Rate Per 100,000 Population by Race/Ethnic Group: Localized Disease



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TRENDS IN STAGE AT DIAGNOSIS BY RACE/ETHNICITY

Figure IX–2: Trends in Age-Adjusted Prostate Cancer Incidence Rate Per 100,000 Population by Race/Ethnic Group: Regional Disease.

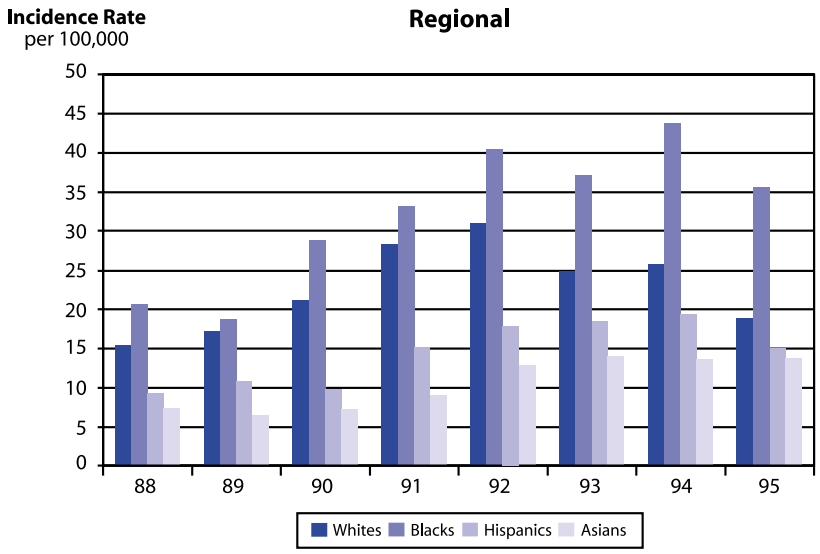

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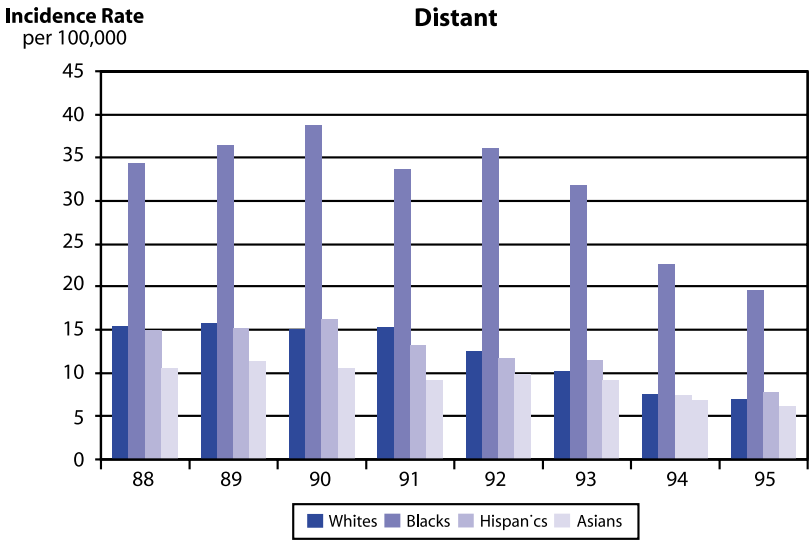
Figure IX–3: Trends in Age-Adjusted Prostate Cancer Incidence Rate Per 100,000 Population by Race/Ethnic Group: Distant Disease.



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SUMMARY

Between 1988 and 1995, 141,103 cases of invasive prostate cancer were reported to the CCR. Of these, 59.5 percent were diagnosed at a localized stage of the disease. When analyzed by race/ethnicity, 61.2 percent of non-Hispanic white men, 58 percent of Hispanic men, 56.7 percent of Asian, and 54.9 percent of black men were diagnosed at a localized stage. We found a later diagnosis as age increased (increasing proportion of distant disease and decreasing proportion of localized disease).

The AAIR was highest for blacks, followed by whites, Hispanics, and Asians. After an increase until 1992 for whites and 1993 for other ethnic groups, the rates decreased. Regardless of age, blacks, Hispanics and Asians were more likely than non-Hispanic whites to be diagnosed at a distant stage. However, the AAIR of distant disease has decreased over the eight year period, and the differences between the ethnic group has diminished.

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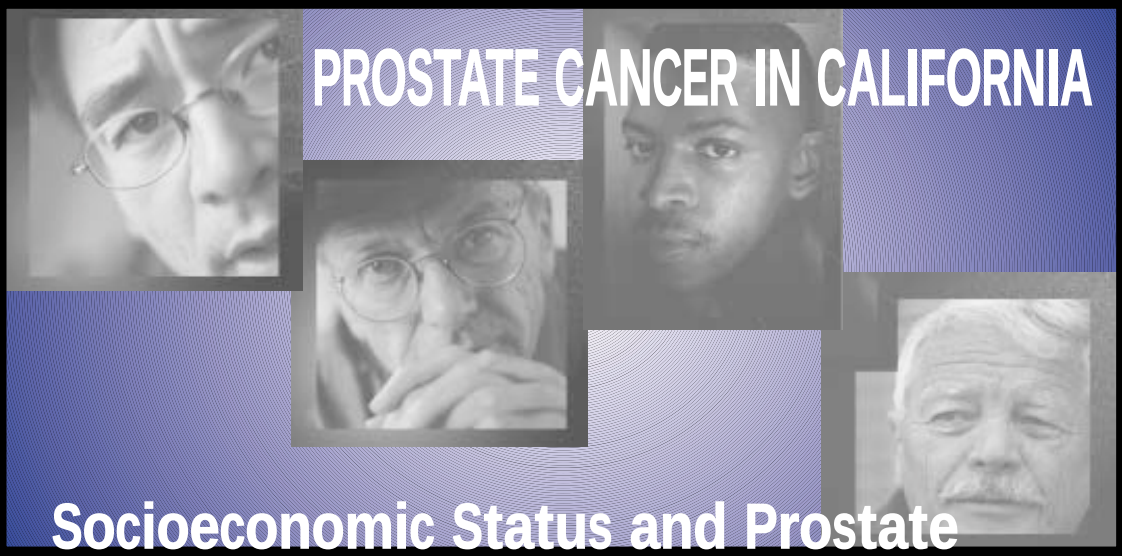


Socioeconomic Status and Prostate Cancer in Los Angeles County, 1972–1995

Lihua Liu, Ph.D.[1] and Wendy Cozen, D.O.[1]

[1] Cancer Surveillance Program, University of Southern California, Los Angeles CA

INTRODUCTION

Socioeconomic status (SES) represents distinctions between lifestyles and resources among population subgroups. It is associated with variations in customs, disparities in wealth and education, and differences in the conditions of daily life. The socioeconomic environment within which people develop adaptive life patterns can influence their health in many ways. Knowledge of the relationship between SES and disease facilitates hypothesis generation about etiology and risk factors of the disease and may also facilitate the targeting of subpopulations who would benefit most from screening, intervention, and prevention programs.

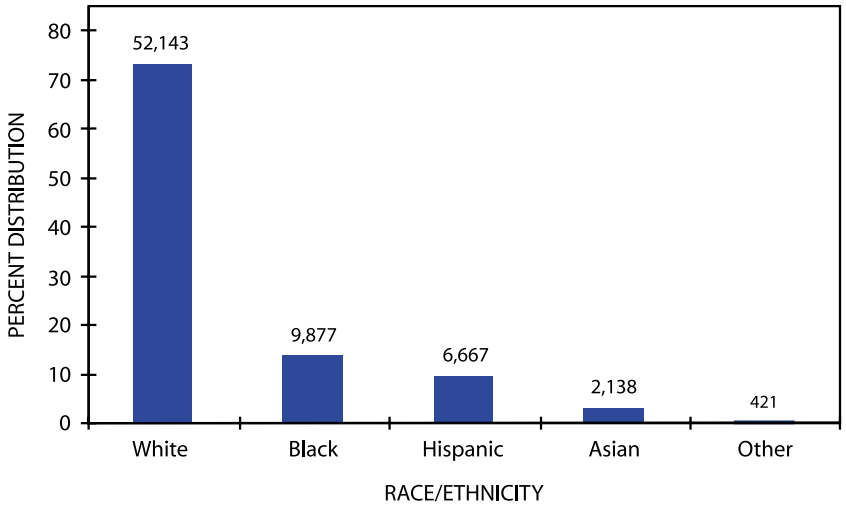
Various studies have examined SES patterns among prostate cancer patients using different methodologies. The results reveal little consensus on this subject with a wide range of findings: from positive associations[1-6] to negative associations,[7] as well as no association.[8-14]

Using data from the University of Southern California Cancer Surveillance Program (USC-CSP), the population-based cancer registry for Los Angeles County, we evaluated the relationship between SES and prostate cancer incidence among patients diagnosed in Los Angeles County during the period of 1972-1995.

MATERIAL AND METHODS

The USC-CSP has collected information on all incident cancer cases diagnosed among Los Angeles County residents since 1972. A high priority is always placed on monitoring demographic patterns and collecting demographic data of cancer patients. Recently we developed a methodology to estimate the SES of cancer patients with reliable accuracy utilizing population census data. A patient's SES was characterized by the average income and educational levels of his/her immediate neighborhood, defined by census tract of residence at diagnosis of cancer. Based on the past three decennial census results, five SES groups were identified for each census year, according to the quintile of the census tracts

Figure X–1: Racial/Ethnic Distribution of Prostate Cancer Patients, Los Angeles County, 1972-1995



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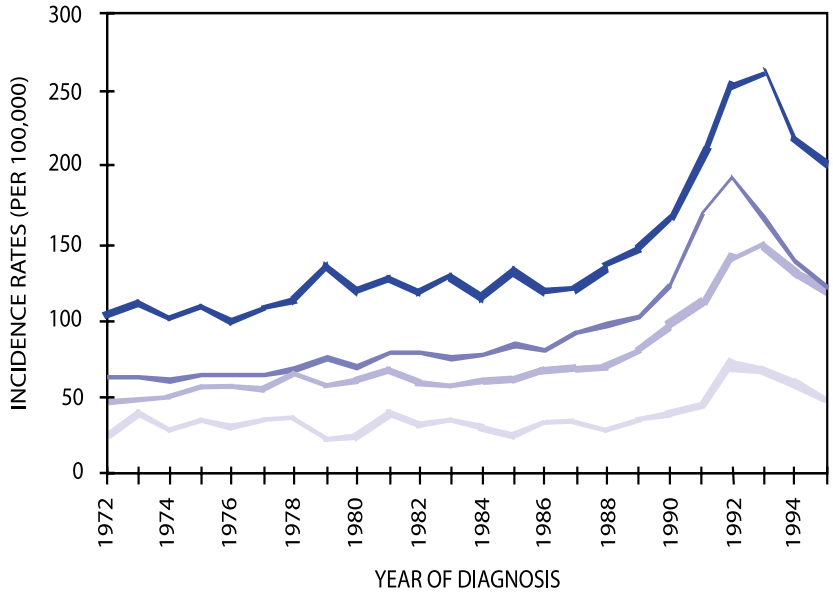
MATERIAL AND METHODS

Figure X–2: Age-adjusted (1970 US) Incidence Rate of Prostate Cancer by Year of Diagnosis and Race/Ethnicity, Los Angeles County, California, 1972-1995.



ranked by income and education. The SES of each census tract in the intercensal years was estimated.[15] Every cancer patient was assigned a SES score according to the residential address given at the time of the prostate cancer diagnosis.

Subjects of our analysis consisted of men diagnosed with invasive primary prostate cancer during the period of 1972-1995 while residing in Los Angeles County (n=71,246). Variables examined included year of diagnosis, race/ethnicity, SES, and stage of disease at diagnosis. Subjects were classified into five mutually exclusive racial/ethnic groups (whites, blacks, Hispanics, Asians and others) based on information from medical records and Spanish surname lists used by the Census Bureau. The definition of race/ethnicity follows the USC-CSP's convention: whites are non-Spanish-surnamed white; Hispanics are Spanish-surnamed white; blacks and Asians are not further distinguished by Hispanic origin or Spanish surnames. Major Asian ethnic groups (including Chinese, Japanese, Korean, and Filipino) are categorized as Asians. The rest of the county's population, including American Indians, Pacific Islanders, other Southeast Asians, and nonspecified populations are classified as "other" race/ethnicity. The racial/ethnic composition of prostate patient cases diagnosed in Los Angeles County for 1972-1995 is shown in Figure X-1. Since the "other" race/ethnicity is a highly-mixed group and contributes less than one percent of the prostate cancer cases in Los Angeles County, we will not present data for this group in the following analysis.

Annual population estimates for the intercensal years of Los Angeles County by sex, age, race/ethnicity, and SES were obtained by interpolating between the census years with the adjusted racial/ethnic distribution and SES classification of census results. For 1991-1995, we utilized the age-sex-race-specific annual population growth rates estimated by the California Department of Finance.[16] Age-adjusted incidence rates (AAIR) standardized to the 1970 US population by race/ethnicity and SES were calculated for analysis.

RESULTS

The incidence rate of prostate cancer in Los Angeles County was relatively stable during the 1970s and early 1980s (Figure X-2). After the Federal Food and Drug Administration (FDA) approved the prostate-specific antigen (PSA) test in 1987, prostate cancer incidence increased dramatically nationwide. In Los Angeles County, the increase of prostate cancer diagnoses after the introduction of PSA occurred in every racial/ethnic population. However, the increase in prostate cancer diagnosis among white men started earlier and peaked earlier than in any other racial /ethnic group (Figure X-2).

The SES association with prostate cancer incidence varied dramatically in the pre-PSA and PSA period. No apparent SES trends were found in any population during the 1972-1987 (pre-PSA) period (Figures X-3). However, a strongly positive and statistically significant SES association was found in all non-Asian populations during the 1988-1995 (PSA) period (Figure X-4).

Examination of AAIR by stage of disease at diagnosis during 1987-1995 showed that SES is positively associated with local and regional stage and inversely correlated with distant stage, suggesting that early diagnosis is occurring more frequently among men of high SES while men of low SES are likely to have delayed diagnoses (Figure X-5).

DISCUSSION

We found no relationship between SES and prostate cancer before 1988, but a strongly positive SES association with prostate cancer after the introduction of PSA into clinical practice, and this positive association was not mediated by age or race/ethnicity. The findings suggest that SES probably is not etiologically related to prostate cancer risk. Instead, the appearance of an SES trend after PSA became available indicates differences in access to prostate care among SES groups. Higher prostate cancer incidence among men of high

Figure X–3: Annual Age-Adjusted (1970 US) Incidence Rates by SES and Race/Ethnicity, Prostate Cancer, Los Angeles County, California, 1972-1987.

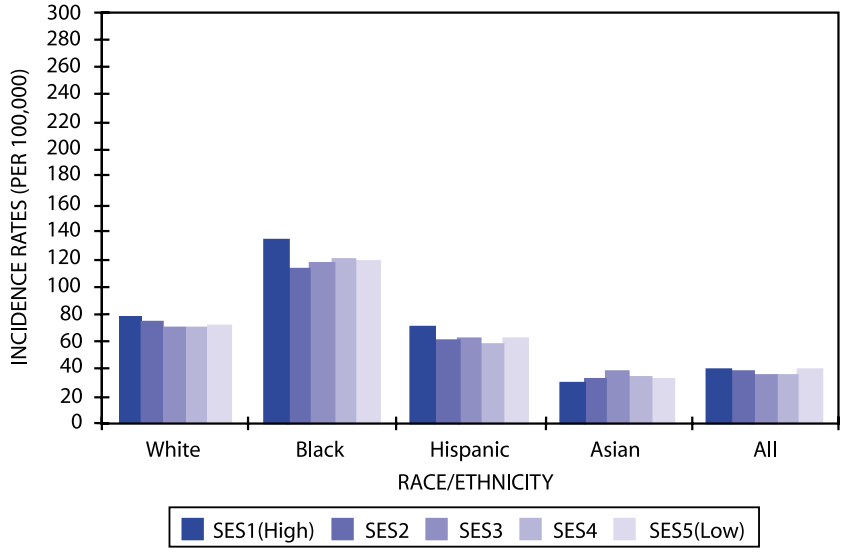


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Figure X–4: Annual Age-Adjusted (1970 US) Incidence Rates by SES and Race/Ethnicity, Prostate Cancer, Los Angeles County, California, 1988-1995.

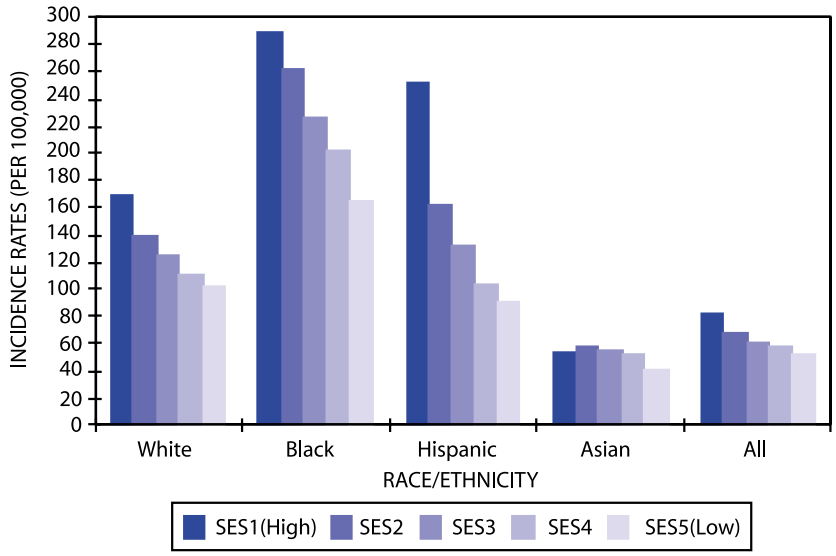

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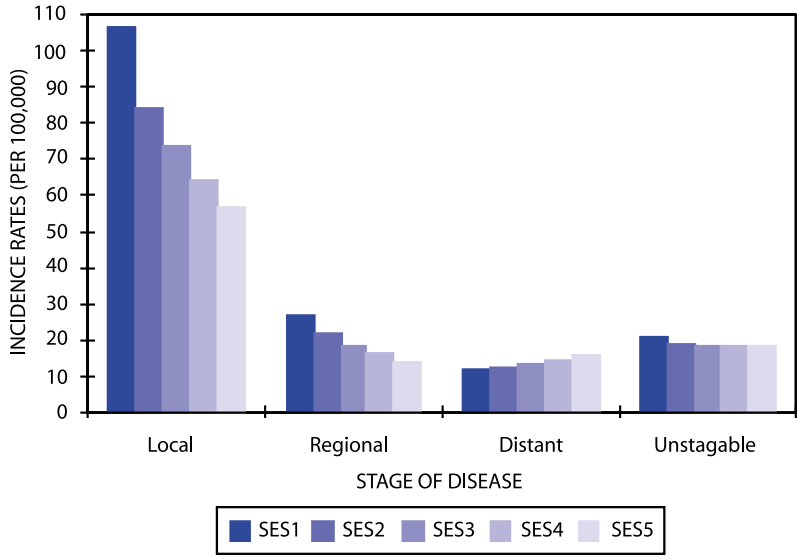
Figure X–5: Annual Age-Adjusted (1970 US) Incidence Rates by SES and Stage of Disease, Prostate Cancer, All Males, Los Angeles County, California, 1988-1995.



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DISCUSSION

SES is contrasted with lack of prostate cancer screening among low SES men. The correlation between high SES and early detection of prostate cancer further underlines the differences in health behaviors among different SES populations.

The lack of an SES association in prostate cancer risk among Asian men may be a result of one or more factors, including 1) the validity of using area-based SES measurement among Asians, 2) a baseline prostate cancer risk that is so low as to be unaffected by increased screening, or 3) less variation in medical utilization among Asian SES groups.

Our data support a familiar social phenomenon. Specifically, when changes occur in diseases, treatments, risks, and/or knowledge about risks, those with financial resources always have the advantage, while people with no or limited resources are disadvantaged with respect to access, quality, and utilization of medical services.[17, 18] Despite the higher incidence rates, high SES groups are found to have better survival from prostate cancer compared to the low SES groups.[19, 6] When there is equal access, SES does not correlate with stage of disease at diagnosis or survival with prostate cancer.[20]

The widespread use of PSA screening and early detection programs are thought to explain most of the changing patterns in prostate cancer incidence.[21, 22] Despite the recent increase in incidence, the mortality rates of prostate cancer in the U.S. remained relatively stable over the last decades, indicating that PSA screening and early detection uncovered a high proportion of latent and non-life threatening tumors.

Screening has been an important means of cancer control for many types of cancer, however the benefit of screening for prostate cancer is uncertain.[23, 24] There is a great need for long-term evaluation of the benefit and harm related to PSA screening before screening is promoted as a strategy to control prostate cancer mortality.

In conclusion, our data suggest that SES does not play an etiological role in prostate cancer pathogenesis. Nevertheless, SES should be considered as an important factor that affects diagnosis and survival of prostate cancer.

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Prostate Cancer in California: *A Special Report*.
Published June 2000.