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A Prospective Study of Demographics, Diet, and Prostate Cancer among Men of Japanese Ancestry in Hawaii[1]

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ABSTRACT

Prostate cancer incidence was prospectively studied among 7999 men of Japanese ancestry who were first examined between 1965 and 1968 and then followed through 1986. During this surveillance period, 174 incident cases of prostate cancer were recorded. Prostate cancer was not associated with any measure of socioeconomic status, including amount of education, type of occupation, and type of residence. There was also no relationship with the number of children, as a surrogate measure of sexual activity. Increased consumption of rice and tofu were both associated with a decreased risk of prostate cancer, while consumption of seaweeds was associated with an increased risk of prostate cancer. There was no relationship between prostate cancer and the intake of various nutrients, including total fat and total protein. Etiological implications of these associations are discussed, but more research is needed on these dietary factors and the subsequent development of prostate cancer before any firm conclusions can be drawn.

INTRODUCTION

Cancer of the prostate is one of the most common male cancers in the United States. Results from the National Cancer Institute's SEER Program show that the age-adjusted prostate cancer incidence rate for all races combined is exceeded only by the rate for lung and bronchus (1).

Data on migrating populations have shown that, as migrants move from areas of low incidence to areas of higher incidence, their incidence rate of prostate cancer increases. For example, Japan has one of the lowest incidence rates of prostate cancer in the world (2). Among Japanese in Hawaii, the age-adjusted incidence rate of prostate cancer is approximately 10 times that in Japan (1). However, it is about one-half the rate in Hawaii whites. This change in rates gives the impression that environmental factors play a major role in the etiology of prostate cancer.

There have been a number of studies which have attempted to identify factors related to this cancer. Their findings have been limited, however, and little is currently known regarding its etiology. SES[3] may be related to the development of prostate cancer. However, the evidence from previous studies concerning SES is equivocal with reports of a positive association (3), negative association (4), and no association (5). The relationship between prostate cancer and sexual activity is also uncertain. An increased number of sexual partners (6), greater use of prostitutes (7), past history of venereal disease (6–8), and increased number of children (9, 10) have all been associated with an increased risk of prostate cancer, but other studies have not supported these observations (11, 12).

Several potential dietary risk factors for prostate cancer have been tentatively identified, including meat (13–15) and fish (15), eggs (14), milk (14, 15), cheese (14), and other dairy products (13). Other studies have also found that nutrients such as fat (15–18) and protein (16) were positively associated with prostate cancer. Since few dietary studies have been done, however, the exact role of dietary factors in the etiology of prostate cancer remains unknown. Recent reviews have called for further studies (19, 20). This paper reports on the association between several of these environmental factors, including diet, and the development of prostate cancer in a cohort of 7999 Japanese males living in Hawaii.

MATERIALS AND METHODS

The subjects for this study were men of Japanese ancestry, born from 1900 to 1919 and residing on the Hawaiian island of Oahu. They were first identified by the HHP in 1965 with the use of the comprehensive 1942 Selective Service draft registration files (21). Of 11,136 identified men, 8,006 (71.9%) were interviewed and examined from 1965 to 1968, 180 (1.6%) died before they could be examined, and 2,950 (26.5%) did not participate in the program. During the interview session, information was recorded on the subject's marital history, smoking history, occupational history, type of residence, educational history, alcohol consumption, and medical history. Men born in Japan were designated as Issei (first generation), while men born outside of Japan (mostly in Hawaii) were designated as Nisei (second generation).

A physical activity index was calculated, following the method used in the Framingham Study (22). It was based on the weighted sum of the usual amount of time the subject spent per 24 h in the following types of activity: sleeping or lying down (weight of 1); sedentary as in sitting or standing (1.1); slight as in walking on a level surface (1.5); moderate as in gardening or carpentry (2.4); and heavy as in shoveling or digging (5).

Also at the time of interview, dietary practices were assessed by a food frequency questionnaire which was based on the subject's usual dietary practices at the time of the exam. Frequency of consumption was recorded for the following 20 food items: rice; bread; udon (any flour noodle except buckwheat noodles); fish; meat; eggs; miso soup (soup with soybean paste); fried vegetables; tofu; milk; pickles; tsukudani (mixed dish of fish, sugar, shoyu, and seaweed); seaweeds; green tea; black tea; coffee; pastry; fruit; ice cream; and salt/shoyu (soy sauce). These items were selected by the HHP as indicators of traditional Japanese or American food habits (23). Consumption of ham, bacon, or sausage was recorded as a single unit, as was consumption of butter, margarine, or cheese and candy, jelly, or soda pop. Consumption of these 23 foods was classified into five categories (for example, almost never, less than 2 times/wk, 2 to 4 times/wk, almost daily, and more than once/day), except for salt/shoyu, rice, and green tea.

In addition, a 24-h diet recall interview was administered to each study subject. Food models and serving utensils were used to illustrate portion size. Food composition values were compiled from the best available sources to calculate the individual intakes of calories and nutrients (23). The method has been used by other large-scale dietary studies, such as the Ten State Nutrition Survey of 1968 to 1970 (24) and the First Health and Nutrition Examination Survey, 1971–1972 (25).

Subsequent to the initial exam, the cohort has been followed for the development of incident cancers through continuous surveillance of Oahu hospitals and periodic linkage to the Hawaii Tumor Registry which is part of the National Cancer Institute's SEER Program (26). Only those cases that were newly diagnosed with histologically confirmed malignant prostate cancer were included in this analysis. The cases were divided into two clinical groups: (a) those with clinically overt prostate cancer; and (b) those with less apparent or latent, prostate

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[3] The abbreviations used are: SES, socioeconomic status; HHP, Honolulu Heart Program.

cancer diagnosed in tissue fragments obtained at transurethral resection for benign prostatic hyperplasia. It has been estimated that only 1.8% of the cohort has moved from Oahu (27), so the cohort is geographically stable, and case identification has been virtually complete.

A total of seven men with prostate cancer at the time of exam (prevalent cases) was eliminated from the analysis. The time at risk for each subject was computed as the time from examination to histologically confirmed diagnosis with prostate cancer, death, or September 30, 1986, whichever occurred first. These calculations resulted in a total of 139,727 person-years at risk of prostate cancer. The relative risk of prostate cancer associated with various exposures was estimated from proportional hazards regression models (28), while adjusting for age at examination.

RESULTS

During the surveillance period, 174 incident cases of prostate cancer were recorded in the cohort. Table 1 shows the relative risk of prostate cancer associated with cigarette smoking, socioeconomic status, marital status, number of children, and several other personal characteristics in the cohort. None of these variables showed a significant association with prostate cancer.

Table 2 shows the relative risk of prostate cancer associated with usual frequency of intake of dietary items recorded at the time of examination. There was a significant negative trend ($P = 0.017$) in the intake of rice among study subjects. A progressive decrease in prostate cancer risk with increasing rice consumption was observed. Subjects who consumed more tofu, another prominently oriental food item, also had a decreased risk, but the trend was not statistically significant ($P = 0.054$). Those subjects who consumed two to four servings of bread per week had a significantly increased risk compared to those with less consumption. Those subjects who consumed two to four servings of egg per week also experienced a significantly increased risk compared to those consuming less. However, there was no dose response associated with either of these two exposures.

Increasing consumption of nori, kobu, or other seaweeds was associated with a significantly increasing prostate cancer risk (test for trend, $P = 0.017$). Subjects who consumed at least five servings per week had a significantly increased risk compared to those who consumed no more than one serving per week.

Based on the 24-h diet recall questionnaire, several summary dietary measures were calculated including the consumption of total calories, total protein, total fat, saturated fat, unsaturated fat, total carbohydrates, and alcohol. The results are presented in Table 3. None of these variables provided any evidence of a relationship with prostate cancer, except for total protein. Subjects in the middle consumption group had a significantly increased risk, but it was lower than that of the high protein consumption group.

All of the analyses were repeated, excluding the 43 latent cases of prostate cancer, as previously described. The results (not shown) were very similar to those with all 174 cases included.

Table 1 *Age-adjusted relative risks and 95% confidence intervals for prostate cancer according to selected characteristics among Japanese men in Hawaii, 1965–1986*

Variable	No.[a]		RR[b]	95% CI
	Cases	Noncases		
Smoking				
Never	63	2341	1.00	
Past	46	2048	0.89	0.61, 1.29
Current	65	3435	0.87	0.61, 1.23
Residence				
Own home	137	5889	1.00	
Other	37	1932	0.85	0.59, 1.22
Education (yr)				
0–9	98	3973	1.00	
10–12 or technical school	56	3038	0.99	0.71, 1.39
13+	20	811	1.16	0.72, 1.89
Usual occupation				
Unskilled manual	26	1294	1.00	
Skilled manual	80	3552	1.30	0.83, 2.03
Nonmanual (clerical, supervisory, managerial)	67	2975	1.24	0.78, 1.95
Marital status				
Married/widowed	168	7427	1.00	
Other	6	392	0.86	0.38, 1.95
Generation				
Issei	38	900	1.00	
Nisei	136	6925	1.13	0.75, 1.71
Physical activity index				
0–29.9	50	2406	1.00	
30–33.9	52	2596	0.87	0.59, 1.28
34.0+	71	2757	1.16	0.81, 1.67
No. of children				
0–1	29	1544	1.00	
2–3	78	3673	1.16	0.76, 1.78
4+	67	2608	1.07	0.69, 1.66

[a] The numbers for each category do not always total 7999 because of unknown responses.
[b] RR, relative risk; CI, confidence interval.

DISCUSSION

Despite the large international differences in prostate cancer incidence rates and the well-known associations with age and race, very little is presently known regarding the etiology of prostate cancer. For this reason, we conducted this prospective study among the Japanese men living in Hawaii. The advantages of a prospective study are well known and include the ability to directly compare incidence rates in the exposed and unexposed groups. It is less likely that exposure information is subject to misclassification in prospective studies compared with case-control studies, since exposure is determined before disease occurrence. Two potential difficulties associated with cohort studies—subjects lost to follow-up and incomplete ascertainment of disease—are minimized in this study due to the residential stability of the cohort and continuous surveillance of Oahu hospitals with periodic linkage to the Hawaii SEER Tumor Registry.

We found no evidence of a relationship between SES and the subsequent development of prostate cancer. Our measures of SES included home ownership, usual occupation, and educational level. When our results are evaluated in light of the equivocal findings from previous studies (3–5), it seems unlikely that SES plays a major role in the etiology of prostate cancer.

Past studies have associated number of children with prostate cancer risk (9, 10), but our investigation did not find a similar relationship. Although this lack of an association is consistent with several other studies (12, 29), the number of children a man has is not necessarily an accurate reflection of sexual activity. Direct information on sexual activity was not obtained at the time of examination of the study subjects.

Although there has been some investigation of dietary items in the epidemiology of prostate cancer (13–15), little is currently known regarding their specific role. In general, dietary items suggested from previous case-control studies to have a potential role in prostate cancer had little effect in our data. We found no association between the consumption of meat, fish, or milk and prostate cancer. There was some evidence that men who consumed larger amounts of butter, margarine, cheese, and

Table 2 *Age-adjusted relative risks and 95% confidence intervals for prostate cancer according to usual dietary habits at first exam among Japanese men in Hawaii, 1965–1986*

Variable	No.[a] Cases	Noncases	RR[b]	95% CI
Salt/shoyu				
Never-seldom	44	2130	1.00	
After testing	89	3822	1.14	0.79, 1.63
Always	33	1734	0.93	0.59, 1.46
Rice				
≤1/day	100	3901	1.00	
2/day	70	3537	0.77	0.57, 1.04
≥3/day	4	387	0.38	0.14, 1.04
Bread				
≤1/wk	5	561	1.00	
2–4/wk	12	489	3.33	1.17, 9.47
≥5/wk	157	6774	2.31	0.95, 5.63
Udon				
≤1/wk	151	6860	1.00	
≥2/wk	23	964	1.09	0.70, 1.69
Fish				
≤1/wk	93	4290	1.00	
2–4/wk	62	2967	0.84	0.61, 1.17
≥5/wk	19	567	1.22	0.74, 2.01
Ham, bacon, sausage				
≤1/wk	86	3649	1.00	
2–4/wk	53	2541	1.04	0.74, 1.47
≥5/wk	35	1634	1.11	0.75, 1.65
Meat				
≤1/wk	35	1419	1.00	
2–4/wk	95	3997	1.02	0.69, 1.50
≥5/wk	44	2406	0.95	0.61, 1.49
Butter, margarine, cheese				
≤1/wk	26	1643	1.00	
2–4/wk	22	891	1.72	0.98, 3.04
≥5/wk	126	5289	1.47	0.97, 2.25
Egg				
≤1/wk	20	1412	1.00	
2–4/wk	61	2401	1.85	1.12, 3.06
≥5/wk	93	4011	1.57	0.97, 2.54
Miso soup				
≤1/wk	138	6568	1.00	
2–4/wk	31	1089	1.19	0.80, 1.76
≥5/wk	5	166	1.24	0.51, 3.04
Fried vegetables				
≤1/wk	158	7221	1.00	
≥2/wk	16	602	1.40	0.84, 2.34
Tofu				
≤1/wk	140	6162	1.00	
2–4/wk	32	1497	0.78	0.53, 1.14
≥5/wk	2	165	0.35	0.08, 1.43
Milk				
≤1/wk	98	4604	1.00	
2–4/wk	17	659	1.29	0.77, 2.16
≥5/wk	59	2560	1.00	0.73, 1.38
Nori, kobu, other seaweeds				
≤1/wk	126	6265	1.00	
2–4/wk	31	1116	1.32	0.89, 1.96
≥5/wk	17	440	1.74	1.05, 2.90
Pickles				
≤1/wk	64	3035	1.00	
2–4/wk	43	1913	1.12	0.76, 1.65
≥5/wk	67	2875	1.01	0.72, 1.43
Tsukudani				
≤1/wk	159	7222	1.00	
2–4/wk	10	385	1.07	0.56, 2.03
≥5/wk	5	217	1.03	0.42, 2.52
Coffee				
≤1/wk	22	926	1.00	
2–4/wk	6	252	0.96	0.39, 2.37
≥5/wk	146	6646	0.92	0.59, 1.44
Pastry				
≤1/wk	112	4953	1.00	
2–4/wk	42	1947	1.00	0.70, 1.42
≥5/wk	20	924	0.99	0.62, 1.60
Fruit				
≤1/wk	17	1226	1.00	
2–4/wk	16	1439	0.74	0.37, 1.46
≥5/wk	141	5158	1.57	0.95, 2.61
Ice cream				
≤1/wk	112	5211	1.00	
2–4/wk	38	1867	0.94	0.65, 1.36
≥5/wk	24	743	1.31	0.84, 2.03
Candy, jelly, or soda				
≤1/wk	41	2073	1.00	
2–4/wk	35	1608	1.12	0.72, 1.76
≥5/wk	98	4143	1.25	0.87, 1.80
Green tea				
Never	29	1850	1.00	
Ever	145	5971	1.47	0.99, 2.19
Black tea				
Never	105	4016	1.00	
Ever	69	3807	0.83	0.61, 1.13

[a] The numbers for each food category do not always total 7999 because of unknown responses.
[b] RR, relative risk; CI, confidence interval.

Table 3 *Age-adjusted relative risks and 95% confidence intervals for prostate cancer according to summary dietary exposures calculated from 24-h recall questionnaire among Japanese men in Hawaii, 1965–1986*

Variable	No. Cases	Noncases	RR[a]	95% CI
Total calories				
0–1999	75	2941	1.00	
2000–2499	41	2182	0.87	0.59, 1.27
2500+	58	2702	1.22	0.85, 1.74
Total protein				
0–74.9 g	51	2455	1.00	
75.0–99.9 g	69	2397	1.54	1.07, 2.22
100.0+ g	54	2973	1.13	0.76, 1.67
Total fat				
0–64.9 g	69	2534	1.00	
65.0–99.9 g	66	2911	0.99	0.70, 1.39
100.0+ g	39	2380	0.87	0.58, 1.31
Saturated fat				
0–39.9 g	65	2306	1.00	
40.0–69.9 g	59	3003	0.81	0.57, 1.15
70.0+ g	50	2516	1.00	0.68, 1.46
Unsaturated fat				
0–14.9 g	57	2687	1.00	
15.0–29.9 g	65	2500	1.29	0.90, 1.84
30.0+ g	52	2638	1.09	0.75, 1.60
Total carbohydrates				
0–199.9 g	53	2207	1.00	
200.0–299.9 g	61	3250	0.82	0.57, 1.19
300.0+ g	60	2368	1.26	0.87, 1.83
Alcohol				
None	126	5572	1.00	
0.1–29.9 g	23	1108	0.85	0.54, 1.33
30.0+ g	25	1145	1.09	0.71, 1.68

[a] RR, relative risk; CI, confidence interval.

eggs were at increased risk, but there was no evidence of a dose response.

There was a small negative relationship between consumption of rice and prostate cancer. Based on food frequency data, those who usually consumed rice often had a significantly decreased risk compared to those who usually consumed less than two servings per day. In addition to rice, the consumption of tofu

was inversely related to prostate cancer (trend $P = 0.054$). However, as can be observed in Table 2, the results for tofu are based on fairly small numbers.

The negative association of rice and tofu with prostate cancer may be due to chance. However, if the lifestyle in Japan is protective against this cancer, then it would be worthwhile to investigate further these associations. Tofu consists of water and soybean curd precipitated by calcium chloride and/or calcium sulfate. To our knowledge, none of these products has been directly associated with inhibition of carcinogenesis. It is also possible that increased consumption of rice and tofu may be associated with a reduced consumption of other foods, which could be positively associated with prostate cancer risk.

One might conclude from this that some traditional Japanese food items are negatively associated with prostate cancer, while some "western" dietary items are weakly positively associated with prostate cancer risk. In contradiction to this picture, however, our results suggest that increased consumption of seaweeds (such as nori and kobu) is associated with an increased risk of prostate cancer. Those who consumed at least five servings/week had a significantly increased risk compared to those who consumed at most one serving/week (relative risk = 1.74, 95% confidence interval = 1.05, 2.90), and there was a significant ($P = 0.017$) increasing trend.

In light of the international differences in both prostate cancer rates and consumption of seaweed between Japan and the United States, one would expect that seaweed consumption might reduce prostate cancer risk. Instead, we find the opposite. Several species of seaweeds (such as *Asparagopsis taxiformis*—commonly called ogo by Hawaii Japanese) have been shown to contain mutagenic compounds (30). In addition, several compounds isolated from seaweeds have been shown to be carcinogenic in laboratory animals (31–33). However, these results are preliminary, and this could be a chance finding, especially since seaweed is not consumed in large amounts in the usual western diet.

Besides food items, we also studied the effects of nutrient intake. There was no indication that the heavy consumers of fat, protein, or calories had an increased risk for prostate cancer. These data suggest that there is no relationship between nutrient intake and prostate cancer or that an association was not detected due to the limitations of a 24-h diet recall questionnaire even in a prospective study of almost 8000 subjects. A case-control study in Japan also did not find any association (34). However, four other studies in the United States observed that prostate cancer cases consumed more fat than controls (15–18). Protein intake was also greater in cases in one study (16), but not in another (17). Clearly, more work is needed with detailed, comprehensive diet histories to clarify the association between dietary factors and prostate cancer.

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