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Legume and isoflavone intake and prostate cancer risk: The Multiethnic Cohort Study

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Abstract

Findings from studies of legume, soy and isoflavone intake and prostate cancer risk are as yet inconclusive, although soy has received considerable attention due to its high phytoestrogen content. Therefore, the present study investigated the relationship of these dietary exposures to prostate cancer risk in the Multiethnic Cohort Study in Hawaii and Los Angeles. The analyses included 82,483 men who completed a detailed quantitative food frequency questionnaire in 1993–1996. A total of 4,404 prostate cancer cases including 1,278 nonlocalized or high-grade cases were recorded during the average follow-up period of 8 years. Multivariate relative risks (RR) and 95% of confidence intervals (CI) were estimated using Cox proportional hazards models with age as the time metric. Among men with the highest intake of legumes, the risk reduction was 11% for total prostate cancer (RR = 0.89, 95% CI = 0.80–0.99, *p* for trend = 0.007) and 26% for nonlocalized or high-grade cancer (RR = 0.74, 95% CI = 0.61–0.90, *p* for trend = 0.007) compared to men with the lowest intake. Similar risk reductions were observed for soy products and for legumes excluding soy products in separate analyses. We found no significant risk reduction associated with intake of total or specific isoflavones for either total prostate cancer or for nonlocalized or high-grade cancer. The findings of our study suggest that legume intake is associated with a moderate reduction in prostate cancer risk and that the isoflavones in soy products are probably not responsible for this effect.

Keywords

legumes; soy; isoflavones; prostate cancer; cohort study; multiethnic population

Legumes have long been recognized for being high in protein and lately have been noted as an excellent source of dietary fiber. However, there has been relatively little research about nutritional features of legumes in relation to chronic diseases.[1-4] Recently, soy has received considerable attention because high intake of soy has been proposed to contribute to a lower risk of certain cancers in Asians.[5-8] Soybeans and soy-based foods have high content of phytoestrogens, most notably isoflavones, and because of their estrogenic and antiestrogenic effects, researchers have focused on the possible protective effects of phytoestrogens against hormonerelated cancers, including breast and prostate cancer.[9,10]

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A meta-analysis of 2 cohort and 6 case-control studies produced an estimate of a 30% reduction in prostate cancer risk with soy consumption.[11] However, prospective epidemiologic studies investigating soy and prostate cancer are still limited and inconsistent.[5] To date, only a few prospective cohort studies conducted among populations with relatively high intake of soy[12-15] have reported on this association. The Multiethnic Cohort Study includes one ethnic group with very high legume intake (Latinos) and another with very high intake of soy products (Japanese Americans). Thus, the present study was undertaken to further explore the relationship of these dietary exposures to prostate cancer risk.

Material and methods

Study cohort

The design and implementation of the Multiethnic Cohort Study (MEC) has been described in detail previously.[16] In brief, the cohort consists of more than 215,000 adults aged 45–75 years who completed a mailed questionnaire in 1993–1996. Participants are mainly from 5 racial/ethnic populations in Hawaii and Los Angeles, California: African Americans, Native Hawaiians, Japanese Americans, Latinos and Whites. The study was approved by the review boards of the University of Hawaii and the University of Southern California. Among 96,958 men in the cohort, we excluded from the current study men who did not self-identify as 1 of the 5 racial/ethnic groups (n = 5,944), men with a history of prostate cancer at baseline (n = 2,890), men with implausible diet based on total energy intake or its components (n = 3,653),[17] and men with incomplete answers to height, weight, education, or smoking (n = 1,988), leaving a total of 82,483 men for the analyses.

Dietary assessment

Dietary intake at baseline was assessed by a self-administered quantitative food frequency questionnaire (QFFQ). The over-180 item QFFQ was developed from three-day measured food records[16] and was validated in a calibration study.[18] Legume consumption was computed as the sum of legumes from single food items as well as from mixed dishes. Soy product intake was estimated from tofu, miso and vegetarian meat. We also calculated intakes of legumes excluding soy products. Isoflavone intake from the QFFQ was calculated using the food composition table that has been developed and maintained by the Cancer Research Center of Hawaii for the Multiethnic Cohort Study. In this analysis, total isoflavone intake was defined as the sum of the 3 individual isoflavones, genistein, daidzein and glycitein.

Case ascertainment

Prostate cancer cases were ascertained by linking the cohort to the Surveillance, Epidemiology and End Results (SEER) cancer registries covering Hawaii and California through December 31, 2002. Deaths were identified by linkage to death-certificate files in Hawaii and California and the National Death Index through December 31, 2002. We considered all prostate cancers as events except *in situ* (high-grade prostatic intraepithelial neoplasia) cases. Nonlocalized prostate cancer was defined as cancer that was regional or distant, while high-grade prostate cancer was defined as Gleason score ≥7 (poorly differentiated).[19] A total of 4,404 prostate cancer cases were recorded during the average follow-up period of 8 years.

Statistical analysis

Dietary intake was expressed as densities (intake per 1,000 kcal) in the analysis, since we found in a calibration study that energy-adjusted nutrients were better correlated with the

reference instrument than were absolute intakes.[18] Legume and isoflavone intakes were categorized into quintiles based on intakes of all men in the cohort. For soy intake, tertiles were used because over 40% of the men in the cohort did not consume soy.

Averages of selected variables at baseline were adjusted for ethnicity and age group by poststratification.[20] Relative risks (RR) of prostate cancer and 95% of confidence intervals (CI) were estimated using Cox proportional hazards models with age as the time metric. The proportionality assumption was tested by Schoenfeld residuals and found to be met. All Cox models were adjusted for time since cohort entry (≤2, 2–5 and >5 years), ethnicity, body mass index (BMI; <22.46, 22.46–24.99, 25.00–29.99 and ≥30.00), educational level (≤8th grade, 9th–12th grade, some college or vocational school and graduated college or higher), family history of prostate cancer (yes and no/do not know) and smoking status (never smoker, former smoker, current smoker <10 cigarettes/day, current smoker 10–19 cigarettes/day, current smoker ≥20 cigarettes/day) as strata variables, and were also adjusted for energy intake (log transformed kcal/day) as a covariate. The adjustment variables were either associated with prostate cancer risk, or likelihood of PSA screening.[19] Other potential confounders, such as intake of fruit, vegetables, fat, meat, dairy products, calcium and vitamin D as well as physical activity were considered. However, they were not included in the models because these factors were not related to prostate cancer risk in our cohort[19,21,22] and did not significantly alter RRs in the current analyses. Linear trends were tested by entering a variable assigned the racial/ethnic-specific median values within the appropriate overall quantiles into the models as continuous variables. Analyses were performed considering all incident prostate tumors, and then limited to nonlocalized or high-grade prostate tumors to investigate whether legume intake has different effects by tumor stage. Since the range of intake varied substantially across the racial/ethnic groups, we also performed separate analyses by racial/ethnic group. In the ethnic-specific analyses, we assigned ethnic-specific cutpoints for legume and isoflavone intakes. In further analyses, we also used identical cutpoints across ethnic groups. In these ethnic-specific analyses, native Hawaiians were excluded, because the numbers of prostate cancer cases were too few for stable estimates. All analyses were performed using SAS statistical software (version 9.1, SAS Institute, Cary, NC) and all *p*-values are two-sided.

Results

Mean daily intakes (±SE) of men in the cohort with adjustment for age and ethnicity by poststratification were 52.0 ± 0.23 g (20.4 ± 0.07 g/1,000 kcal) for all legumes, 10.5 ± 0.07 g (4.6 ± 0.03 g/1,000 kcal) for soy products and 11.8 ± 0.04 mg (4.9 ± 0.02 mg/1,000 kcal) for total isoflavones. Intake of all legumes was highest in Latinos (36.0 ± 0.23 g/1,000 kcal) and lowest in Native Hawaiians (12.5 ± 0.15 g/1,000 kcal); soy product intake was highest in Japanese Americans (11.2 ± 0.09 g/1,000 kcal) and lowest in African Americans (0.5 ± 0.03 g/1,000 kcal) and Latinos (0.5 ± 0.02 g/1,000 kcal). Men with higher intake of all legumes were likely to be older, have less education, to not currently smoke, and to have high energy intake, compared to men with lower intake (Table I).

The multivariate RRs of prostate cancer according to intake of legumes are presented in Table II. The highest quintile of intake of all legumes was associated with a 11% decrease in risk of prostate cancer compared to the lowest quintile. For nonlocalized or high-grade prostate cancer, the risk reduction in the highest quintile of total legume intake was 26%. Legumes contain various potential cancer-protective components including phytoestrogens. Since isoflavone phytoestrogens are present in large amounts in soy, we examined soy products and other legumes (legumes excluding soy products) separately. The highest tertile of soy product intake showed a 10% nonsignificant decrease in total prostate cancer risk and a 22% decrease in nonlocalized or high-grade prostate cancer risk. Intake of legumes

excluding soy products was also associated with risk reduction in the highest quintile, which was similar to that observed for all legumes. Overall, the table shows similar risk reduction (point estimates) for soy and nonsoy legumes, and this is much stronger for nonlocalized or high-grade disease.

Table III shows the associations between specific isoflavone intakes and prostate cancer risk. Although the associations were in the same direction as legume intake, neither the RRs for the individual isoflavones nor for total intake reached statistical significance. Notably, unlike the results in Table II, the findings for isoflavone intake were not significant for nonlocalized or high-grade prostate cancer risk.

In ethnic-specific analyses, Latino men, who had the highest intake of nonsoy legumes and the lowest soy intake among the 4 ethnic groups included, showed an inverse association for the intake of both nonsoy legumes and soy products, as well as for all legumes (Table IV). Notably, Japanese American men, with the highest soy consumption, showed no association of the intakes of all legumes or soy with prostate cancer risk. None of the ethnic groups showed significant inverse associations with specific or total insoflavones (data not shown). When overall cutpoints were used for the 4 ethnic groups, the findings were similar; also a test of heterogeneity was not significant ($p = 0.45$), indicating that the inverse association for legumes was not limited to Latinos (data not shown).

Discussion

This study examined legume and isoflavone intake in relation to prostate cancer risk in a large, ethnically diverse cohort. Among men with the highest intake of legumes, the risk reduction was 11% for total prostate cancer and 26% for nonlocalized or high-grade prostate cancer compared to men with the lowest intake. In separate analyses for soy products and for legumes excluding soy products, similar risk reductions were also observed for the men with highest intake in each subgroup. For isoflavone intake, however, we did not find any significant inverse associations for either total or nonlocalized/high grade prostate cancer, and this was true for the Japanese American subgroup with the highest soy product consumption.

Findings from studies of legume, soy and isoflavone intake and prostate cancer risk are as yet inconclusive. Few epidemiologic studies of prostate cancer have reported on legumes specifically. In a Netherlands cohort, legumes were inversely associated with prostate cancer risk.[23] Two case–control studies reported inverse associations for beans,[24-26] whereas 2 other case–control studies found no association for legumes.[27,28]

Recently, researchers have been focusing on soy consumption rather than intake of all legumes in relation to prostate cancer risk, because of an interest in the high phytoestrogen content of soy. A meta-analysis of soy intake and prostate cancer risk from 2 cohort and 6 case–control studies yielded an overall risk estimate of 0.70 (95% CI = 0.59–0.83).[11]

Because Asians, especially Japanese and Chinese populations, generally consume large amounts of soy products, studies in these groups are particularly relevant. In cohort studies, soy product consumption (tofu or miso soup) was not significantly associated with prostate cancer risk among Japanese men living either in Hawaii[14] or in Japan.[12] An ecologic study in Japan also did not observe any association between soy intake and prostate cancer mortality.[29] In case–control studies, soy product intake showed an inverse association with prostate cancer risk in studies conducted in China[30] and Japan[31] but not in Taiwan.[32] Among 3 studies conducted in North America with a majority of Caucasian participants, a prospective study of Adventist men in California reported that high soy milk consumption was associated with reduced risk of prostate cancer.[13] One case–control study reported a

suggestive inverse association for isoflavones,[33] while another study found no association for tofu or soybean intake.[28] Possible reasons for the inconsistent findings have been suggested,[5,15] including misclassification of soy intake due to measurement errors, lack of adequate data on food composition, and levels of soy consumption in Western populations that are too low to detect a protective effect against prostate cancer.

Earlier, we conducted a multiethnic case–control study in Hawaii, California and Canada from which we reported that legume and soyfood consumption was inversely associated with prostate cancer risk and that the findings were generally consistent across ethnic groups (African Americans, Whites, Japanese and Chinese).[26] In the racial/ethnic specific analysis of the present cohort study, an inverse association between legume consumption and prostate cancer risk was seen mainly in the Latino group, which had by far the highest consumption of legumes, only a modest proportion of which was comprised of soy products. However, there was no statistical evidence of a difference across ethnic groups.

Clinical studies of soy administration are inconclusive partly because they did not investigate prostate cancer as the outcome but rather focused on markers of the disease such as PSA or reproductive hormone levels.[10,34,35] A clinical study of prostate cancer patients taking 160 mg of daily isoflavone supplement for 7–54 days suggested that dietary isoflavones may halt the progression of prostate cancer by inducing apoptosis in low to moderate-grade tumors.[36] In our study, we observed more risk reduction in nonlocalized or high-grade prostate cancer than in total prostate cancer among the men with high soy intake but not among those with high isoflavone intake. The range of isoflavone intake in our study was wide (0–220 mg/day) and the mean daily intake was 11.8 mg, which is much higher than the 1.2 mg reported for Caucasian men[33] or the 3.2–3.3 mg (phytoestrogen) for non-Asian women in the United States[37] but much lower than the 76 mg (genistein and daidzein) for men in China[30] or 25–50 mg for older adults in Japan.[38]

Although intake of genistein and daidzein was highly correlated with soy intake (correlation coefficients = 0.72 for both isoflavones), we did not observe any significant reduction in prostate cancer risk associated with intake of these isoflavones, especially for nonlocalized or high-grade prostate cancer. While it is possible that our food composition table does not correctly quantify isoflavone intakes, we believe this is unlikely because isoflavone intakes are based on comprehensive analyses of a wide range of foods, including those with soy additives commonly consumed in Hawaii and California.[39-42] However, there is great variability in isoflavone content between sources and even between brands/batches of the same soy foods.[43] To minimize this variability, our food composition table presents the mean values across multiple brands and multiple samples of the same brand.[42] We also found a protective effect of legumes whether or not we limited the analysis to soy, which is consistent with the previous report from our multiethnic case–control study.[26] Furthermore, a protective effect of legumes was strong among Latinos who consumed the highest intake of legumes that were mostly non-soy legumes, while among Japanese who had the highest intake of soy and thus isoflavones, no effect was seen. This observation suggests that the finding likely is due to other, more widely distributed components of legumes. In addition to isoflavones, legumes contain a variety of constituents such as dietary fiber, omega-3 fatty acids, protease inhibitors and saponins, which have been studied as anticarcinogenic agents.[2] It is also possible that the combination of several different components in legumes has an additive effect on reducing prostate cancer risk.[26]

There are some limitations to this study. We only considered tofu, miso and vegetarian meat as sources of soy. The consumption of other soy foods, such as soybean milk, cooked soybeans and fried soybean curd were not captured by the QFFQ. Thus, this might lead to misclassification of participants according to soy consumption in our study, especially for

Japanese Americans who consume higher amounts of soy foods compared to other ethnic groups. However, tofu appears to be the major source of soy in Japanese Americans, contributing >80% of total intake.[8] Furthermore, based on multiple 24-hr recalls on more than 3,200 Multiethnic Cohort participants in a calibration study, we found that soy milk was consumed by 0.7% of the Japanese American participants, whereas tofu was reported by 26%. In contrast to these limitations, our prospective cohort study had several strengths: avoidance of recall bias, detailed information that permitted us to control for several potential confounding factors for prostate cancer, and a substantial number of cases of total prostate cancer, as well as of nonlocalized or high-grade prostate cancer.

In conclusion, the findings of our study suggest that legume intake is associated with a moderate reduction in prostate cancer risk, and that the isoflavones in soy products are probably not significant contributors to this effect.

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TABLE I

BASELINE CHARACTERISTICS OF MEN ACCORDING TO INTAKE OF LEGUMES IN THE MULTIETHNIC COHORT STUDY, 1993—2002

Characteristics	Intake of all legumes (g/1,000 kcal)				
	<6.4 (*n* = 15,730)	6.4–<10.8 (*n* = 16,422)	10.8–<16.5 (*n* = 16,678)	16.5–<28.2 (*n* = 16,800)	≥28.2 (*n* = 16,853)
Age at cohort entry (years)	59.3 (0.07)[1]	59.4 (0.07)	59.7 (0.07)	60.4 (0.07)	60.7 (0.06)
Ethnicity (%)					
African Americans	24.3[2]	22.2	21.9	21.0	10.7
Native Hawaiians	27.5	27.9	23.1	15.3	6.2
Japanese Americans	12.9	22.6	25.1	23.5	15.9
Latinos	8.9	9.7	12.6	21.6	47.2
Whites	31.0	22.9	19.9	16.6	9.6
Family history of prostate cancer (%)[3]	6.5	7.2	7.2	6.7	6.8
Current smoking (%)[3]	19.0	17.9	17.8	17.0	16.0
Education (years)[3]	13.6 (0.02)	13.6 (0.02)	13.6 (0.02)	13.4 (0.02)	13.0 (0.02)
BMI (kg/m^2)[3]	25.9 (0.03)	26.1 (0.03)	26.2 (0.03)	26.1 (0.03)	25.8 (0.03)
Physical activity (METS of activity/d)[3]	1.7 (0.003)	1.7 (0.003)	1.7 (0.003)	1.7 (0.003)	1.7 (0.003)
Daily dietary intake[3]					
Total energy (kcal)	2,262 (8.3)	2,345 (8.0)	2,341 (7.8)	2,344 (8.2)	2,426 (8.6)
All fruits and juices (g/1,000 kcal)	151 (1.0)	146 (0.9)	142 (0.8)	144 (0.8)	153 (0.8)
Vegetables without legumes (g/1,000 kcal)	101 (0.5)	115 (0.5)	125 (0.4)	139 (0.5)	161 (0.6)
Total fat (% energy)	28.7 (0.06)	30.1 (0.05)	30.8 (0.05)	30.8 (0.05)	29.3 (0.05)
Saturated fat (% energy)	8.6 (0.02)	8.9 (0.02)	9.1 (0.02)	9.1 (0.02)	8.4 (0.02)
Total meat (g/1,000 kcal)	46.2 (0.21)	49.8 (0.18)	52.9 (0.17)	54.0 (0.18)	49.8 (0.19)
Red meat (g/1,000 kcal)	16.5 (0.10)	19.4 (0.09)	21.3 (0.09)	22.0 (0.10)	19.9 (0.11)
Total calcium (mg/1,000 kcal)	400 (1.7)	402 (1.6)	409 (1.5)	418 (1.4)	423 (1.6)
Total vitamin D (IU/1,000 kcal)	162 (1.6)	158 (1.5)	156 (1.4)	159 (1.4)	164 (1.5)
Genistein (mg/1,000 kcal)	0.9 (0.006)	1.4 (0.007)	1.8 (0.008)	2.4 (0.009)	4.4 (0.022)
Daidzein (mg/1,000 kcal)	0.9 (0.006)	1.4 (0.007)	1.8 (0.008)	2.4 (0.010)	4.6 (0.023)
Glycitein (mg/1,000 kcal)	0.19 (0.001)	0.32 (0.001)	0.43 (0.002)	0.60 (0.002)	1.25 (0.007)
Total isoflavones (mg/1,000 kcal)	1.9 (0.012)	3.1 (0.015)	4.0 (0.018)	5.4 (0.021)	10.3 (0.051)

[1] Mean (SE).

[2] Row percentage.

[3] Adjusted for ethnicity and age group by poststratification.

TABLE II

RELATIVE RISKS (RR) AND 95% CONFIDENCE INTERVALS (CI) OF PROSTATE CANCER ACCORDING TO INTAKE OF LEGUMES IN THE MULTIETHNIC COHORT STUDY, 1993—2002

	Total prostate cancer		Nonlocalized or high-grade prostate cancer	
	Cases (*n* = 4,404)	RR (95% CI)[1]	Cases (*n* = 1,278)	RR (95% CI)[1]
All legumes (g/1,000 kcal)				
<6.4	854	1.00	268	1.00
6.4–<10.8	864	0.97 (0.88–1.07)	253	0.89 (0.74–1.06)
10.8–<16.5	932	1.02 (0.92–1.12)	250	0.86 (0.71–1.03)
16.5–<28.2	905	0.94 (0.85–1.04)	267	0.84 (0.70–1.01)
≥28.2	849	0.89 (0.80–0.99)	240	0.74 (0.61–0.90)
p for trend[2]		0.007		0.007
Soy products (g/1,000 kcal)[3]				
0	2282	1.00	696	1.00
0.1–<2.8	896	0.92 (0.85–1.01)	248	0.82 (0.70–0.96)
≥2.8	1226	0.90 (0.80–1.01)	334	0.78 (0.62–0.98)
p for trend		0.20		0.05
Legumes excluding soy products (g/1,000 kcal)				
<3.6	804	1.00	253	1.00
3.6–<6.8	815	1.01 (0.91–1.12)	231	0.87 (0.72–1.05)
6.8–<11.2	889	1.02 (0.92–1.12)	250	0.86 (0.72–1.04)
11.2–<21.3	997	1.01 (0.91–1.11)	282	0.83 (0.69–1.00)
≥21.3	899	0.90 (0.81–1.01)	262	0.72 (0.59–0.89)
p for trend		0.01		0.01

[1] Adjusted for time since cohort entry, ethnicity, family history of prostate cancer, education, BMI (body mass index), smoking status and energy intake.

[2] *p*-value is based on the Wald test of a trend variable assigned the racial/ethnic-specific median values within the appropriate quantiles.

[3] Soy product intake was calculated from tofu, miso and vegetarian meat.

TABLE III

RELATIVE RISKS (RR) AND 95% CONFIDENCE INTERVALS (CI) OF PROSTATE CANCER ACCORDING TO INTAKE OF ISOFLAVONES IN THE MULTIETHNIC COHORT STUDY, 1993–2002

	Total prostate cancer		Nonlocalized or high-grade prostate cancer	
	Cases (*n* = 4,404)	RR (95% CI)[1]	Cases (*n* = 1,278)	RR (95% CI)[1]
Genistein (mg/1,000 kcal)				
<0.7	1011	1.00	301	1.00
0.7–<1.2	951	1.02 (0.93–1.12)	284	1.02 (0.86–1.22)
1.2–<1.9	854	0.97 (0.88–1.07)	250	0.95 (0.80–1.14)
1.9–<3.1	808	0.98 (0.89–1.09)	216	0.89 (0.73–1.08)
≥3.1	780	0.94 (0.84–1.04)	227	0.89 (0.73–1.09)
p for trend[2]		0.16		0.19
Daidzein (mg/1,000 kcal)				
<0.7	1017	1.00	309	1.00
0.7–<1.3	948	1.01 (0.92–1.11)	273	0.95 (0.80–1.13)
1.3–<2.0	860	0.97 (0.88–1.07)	252	0.94 (0.78–1.12)
2.0–<3.2	804	0.97 (0.88–1.08)	221	0.89 (0.74–1.08)
≥3.2	775	0.92 (0.82–1.02)	223	0.85 (0.70–1.04)
p for trend		0.09		0.13
Glycitein (mg/1,000 kcal)				
<0.18	948	1.00	290	1.00
0.18–<0.32	869	0.95 (0.86–1.04)	263	0.91 (0.76–1.09)
0.32–<0.49	893	1.01 (0.91–1.11)	238	0.86 (0.72–1.04)
0.49–<0.80	861	0.94 (0.85–1.04)	241	0.85 (0.70–1.02)
≥0.80	833	0.91 (0.82–1.01)	246	0.84 (0.70–1.02)
p for trend		0.07		0.14
Total isoflavones (mg/1,000 kcal)				
<1.6	1009	1.00	306	1.00
1.6–<2.9	928	0.99 (0.90–1.09)	273	0.95 (0.80–1.13)
2.9–<4.5	870	0.98 (0.89–1.08)	251	0.94 (0.78–1.12)
4.5–<7.2	809	0.97 (0.87–1.07)	224	0.88 (0.73–1.07)

	Total prostate cancer		Nonlocalized or high-grade prostate cancer	
	Cases (*n* = 4,404)	RR (95% CI)[1]	Cases (*n* = 1,278)	RR (95% CI)[1]
≥7.2	788	0.93 (0.83–1.04)	224	0.85 (0.69–1.03)
p for trend		0.17		0.11

[1] Adjusted for time since cohort entry, ethnicity, family history of prostate cancer, education, BMI, smoking status and energy intake.

[2] *p*-value is based on the Wald test of a trend variable assigned the racial/ethnic-specific median values within the appropriate quintiles.

TABLE IV

RELATIVE RISKS (RR) AND 95% CONFIDENCE INTERVALS (CI) OF TOTAL PROSTATE CANCER ACCORDING TO INTAKE OF LEGUMES BY ETHNICITY IN THE MULTIETHNIC COHORT STUDY, 1993–2002[1]

	African Americans (*n* = 10,706)			Japanese Americans (*n* = 25,052)			Latinos (*n* = 19,779)			Whites (*n* = 21,090)		
	Median[2]	Cases	RR (95% CI)[3]	Median	Cases	RR (95% CI)	Median	Cases	RR (95% CI)	Median	Cases	RR (95% CI)
All legumes												
Q[1]	3.3	222	1.00	5.6	193	1.00	7.0	239	1.00	2.3	199	1.00
Q[2]	7.5	249	1.05 (0.87–1.27)	9.8	207	1.00 (0.82–1.22)	16.1	231	1.03 (0.85–1.24)	6.1	189	0.99 (0.80–1.21)
Q[3]	11.7	245	1.04 (0.86–1.26)	13.9	216	0.98 (0.80–1.20)	26.7	207	0.92 (0.75–1.12)	10.0	173	0.92 (0.75–1.14)
Q[4]	17.1	244	0.98 (0.80–1.19)	19.8	199	0.87 (0.71–1.07)	43.0	191	0.86 (0.70–1.06)	15.1	201	1.06 (0.86–1.30)
Q[5]	29.2	226	0.95 (0.78–1.16)	35.2	247	1.00 (0.82–1.21)	77.7	172	0.80 (0.65–1.00)	27.6	162	0.84 (0.68–1.04)
p for trend[4]			0.39			0.91			0.01			0.15
Soy products[5]												
Q[1]	0.0	898	1.00	2.6	292	1.00	0.0	763	1.00	0.0	561	1.00
Q[2]	0.5	141	0.81 (0.67–0.97)	6.9	382	1.03 (0.88–1.21)	0.4	149	1.02 (0.84–1.23)	1.0	199	0.98 (0.82–1.16)
Q[3]	1.9	147	0.99 (0.83–1.20)	19.0	388	0.95 (0.81–1.11)	1.6	128	0.79 (0.65–0.96)	5.5	164	1.02 (0.85–1.22)
p for trend			0.75			0.32			0.02			0.83
Legumes excluding soy products												
Q[1]	3.1	223	1.00	1.4	219	1.00	6.6	232	1.00	1.8	191	1.00
Q[2]	7.2	249	1.03 (0.85–1.25)	3.4	233	1.12 (0.92–1.35)	15.5	237	1.07 (0.89–1.30)	5.1	192	1.03 (0.83–1.26)
Q[3]	11.2	247	1.01 (0.83–1.22)	5.4	208	0.99 (0.81–1.20)	26.1	207	0.93 (0.77–1.14)	8.5	174	0.94 (0.76–1.17)
Q[4]	16.5	237	0.95 (0.78–1.15)	7.7	209	1.04 (0.85–1.26)	42.4	195	0.90 (0.74–1.11)	12.7	190	0.99 (0.80–1.22)
Q[5]	28.1	230	0.95 (0.78–1.15)	12.7	193	0.99 (0.81–1.21)	77.0	169	0.80 (0.65–1.00)	23.0	177	0.90 (0.73–1.11)
p for trend			0.36			0.62			0.01			0.26

[1] Native Hawaiians are not included in this table as the number of cases was too small for stable risk estimates.

[2] g/1,000 kcal.

[3] Adjusted for time since cohort entry, family history of prostate cancer, education, BMI, smoking status and energy intake.

[4] *p*-value is based on the Wald test of a trend variable assigned the racial/ethnic-specific median values within the appropriate quantiles.

[5] Soy product intake was calculated from tofu, miso and vegetarian meat.