SHORT COMMUNICATION

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Nut consumption and prostate cancer risk and mortality

Weike Wang[1,6], Meng Yang[2,6], Stacey A Kenfield[3], Frank B Hu[1,2,4], Meir J Stampfer[1,2,4], Walter C Willett[1,2,4], Charles S Fuchs[4,5], Edward L Giovannucci[1,2,4,7] and Ying Bao*[,4,7]

[1]Department of Epidemiology, Harvard T.H. Chan School of Public Health, 677 Huntington Avenue, Boston, MA 02115, USA; [2]Department of Nutrition, Harvard T.H. Chan School of Public Health, 677 Huntington Avenue, Boston, MA 02115, USA; [3]Department of Urology, University of California, San Francisco, 550 16th Street, San Francisco, CA 94143, USA; [4]Channing Division of Network Medicine, Department of Medicine, Brigham and Women's Hospital, and Harvard Medical School, 181 Longwood Avenue, Boston, MA 02115, USA and [5]Department of Medical Oncology, Dana-Farber Cancer Institute, 450 Brookline Avenue, Boston, MA 02215, USA

Background: Little is known of the association between nut consumption, and prostate cancer (PCa) incidence and survivorship.

Methods: We conducted an incidence analysis and a case-only survival analysis in the Health Professionals Follow-up Study on the associations of nut consumption (updated every 4 years) with PCa diagnosis, and PCa-specific and overall mortality.

Results: In 26 years, 6810 incident PCa cases were identified from 47 299 men. There was no association between nut consumption and being diagnosed with PCa or PCa-specific mortality. However, patients who consumed nuts five or more times per week after diagnosis had a significant 34% lower rate of overall mortality than those who consumed nuts less than once per month (HR = 0.66, 95% CI: 0.52–0.83, *P*-trend = 0.0005).

Conclusions: There were no statistically significant associations between nut consumption, and PCa incidence or PCa-specific mortality. Frequent nut consumption after diagnosis was associated with significantly reduced overall mortality.

Nuts are rich in bioactive macronutrients, micronutrients, tocopherols and phytochemicals (Ros *et al*, 2010). Current epidemiological evidence has consistently linked increased nut consumption to reduced risk of several chronic conditions including cardiovascular diseases, type 2 diabetes, and inflammation (Ros, 2010; Afshin *et al*, 2014). In contrast, evidence on nut consumption and cancer risk has been insufficient and equivocal (González and Salas-Salvadó, 2007). Prostate cancer (PCa) is the leading cancer among US men (Siegel *et al*, 2015), with ~220 800 new PCa cases diagnosed in 2015 (American Cancer Society, 2015). Studies regarding nut intake and PCa incidence are limited and have reported inconsistent results (Mills *et al*, 1989; Hebert *et al*, 1998; Jain *et al*, 1999; Raimondi *et al*, 2010). Furthermore, few studies have investigated nut intake in relation to survival among PCa patients and in one that has the associations were suggestive, but not statistically significant (Richman *et al*, 2013). Thus, in the current study, we prospectively examined nut consumption in relation to PCa incidence and PCa-specific mortality in a large cohort of male health professionals.

MATERIALS AND METHODS

For details on methods see Supplementary Material.

Study population. The Health Professionals Follow-up Study (HPFS) is a prospective cohort study of US male health professionals established in 1986 (Kenfield *et al*, 2014). After exclusion criteria, the final incidence analysis included 47 299 men and the final case-only survival analysis included 4346 PCa patients without metastasis at diagnosis, followed through January 2012.

Assessment of dietary and non-dietary factors. Participants completed a validated semi-quantitative food frequency questionnaire (FFQ) at baseline and every 4 years thereafter. They were asked how

*Correspondence: Dr Y Bao; E-mail: ying.bao@channing.harvard.edu
[6]These authors contributed equally to this work and share co-first authorship.
[7]These authors contributed equally to this work and share co-senior authorship.

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often they consumed a serving (serving size, 28 g (1 oz)) of peanuts and other nuts during the preceding year. Total nut consumption was defined as the sum of peanut and other nut consumption. A validation study of the FFQ indicated that nuts were reported reasonably accurately (Salvini *et al*, 1989). We also obtained information on other lifestyle factors and medical history.

Ascertainment of PCa. PCa diagnosis was confirmed with medical records and pathology reports. Family reports and National Death Index searches were used to identify deaths. We examined hazard ratios (HR) with respect to the following categories of PCa: total (excluding T1a cancers), advanced, lethal, fatal, low-grade (Gleason score 2–6) and high-grade (Gleason score $\geqslant$7). Advanced PCa was defined as stage T3b, T4, N1, or M1 at diagnosis, or lymph node metastases, distant metastases, or PCa death during follow-up. Lethal cancer, the primary focus of our study, was defined as cases that metastasised to distant organs at diagnosis or over follow-up, or that caused PCa death. Fatal cancers were defined as those that caused PCa death. We also investigated post-diagnostic nut intake in relation to development of lethal PCa, fatal PCa, and all-cause mortality among men diagnosed with localised or regional PCa (clinical stage T1–T3a).

Statistical analysis. Cox proportional hazards models were used to estimate HR and 95% confidence intervals (CIs).

PCa incidence analysis. Men were followed from return of baseline questionnaire until diagnosis, death, or 31 January 2012. Nut consumption was presented as a cumulative average from 1986 to end of follow-up. In secondary analyses, we assessed interactions by ethnicity, age, and BMI, added a 2-year lag period between nut-intake assessment and each follow-up period, and further examined baseline nut intake in relation to being diagnosed with PCa.

Case-only survival analysis. For lethal PCa, men were followed from PCa diagnosis to metastases. For fatal PCa, men were followed from PCa diagnosis to death from PCa. For all-cause mortality, men were followed from PCa diagnosis until death by any cause or 31 January 2012. Nut consumption was presented as a cumulative

Table 1. Hazard ratios and 95% confidence intervals of prostate cancer, according to the total nut consumption

	Frequency of total nut consumption (28 g serving)					
	Less than once per month	Less than once per week	Once per week	2–4 Times per week	$\geqslant$5 Times per week	*P* for trend
Nut-intake, servings per day	0–0.033	0.034–0.10	0.11–0.20	0.21–0.60	>0.60	
All						
Event	999	1481	1615	1998	717	
Person-years	171 866	216 234	237 987	272 132	100 500	
Age-adjusted[a]	1	1.08 (1.00, 1.18)	1.11 (1.02, 1.20)	1.11 (1.03, 1.20)	1.04 (0.93, 1.16)	0.54
MV1-adjusted[b]	1	1.05 (0.97, 1.14)	1.07 (0.99, 1.16)	1.07 (0.98, 1.15)	1.00 (0.90, 1.11)	0.85
MV2-adjusted[c]	1	1.04 (0.96, 1.13)	1.06 (0.98, 1.15)	1.05 (0.97, 1.14)	0.98 (0.89, 1.09)	0.61
Advanced[d]						
Event	172	247	265	312	136	
Person-years	172 673	217 410	239 312	273 755	101 050	
Age-adjusted[a]	1	1.16 (0.95, 1.41)	1.19 (0.98, 1.45)	1.18 (0.97, 1.43)	1.23 (0.97, 1.56)	0.20
MV1-adjusted[b]	1	1.15 (0.94, 1.40)	1.18 (0.97, 1.44)	1.17 (0.96, 1.42)	1.22 (0.96, 1.55)	0.23
MV2-adjusted[c]	1	1.13 (0.93, 1.38)	1.15 (0.94, 1.40)	1.13 (0.93, 1.37)	1.18 (0.93, 1.50)	0.40
Lethal[e]						
Event	149	190	193	233	107	
Person-years	17 694	217 459	239 383	273 826	101 074	
Age-adjusted[a]	1	1.05 (0.84, 1.30)	1.02 (0.82, 1.26)	1.02 (0.83, 1.27)	1.11 (0.85, 1.43)	0.58
MV1-adjusted[b]	1	1.05 (0.84, 1.31)	1.02 (0.82, 1.27)	1.02 (0.83, 1.27)	1.11 (0.85, 1.44)	0.58
MV2-adjusted[c]	1	1.04 (0.83, 1.29)	1.00 (0.80, 1.24)	0.99 (0.80, 1.23)	1.07 (0.82, 1.40)	0.78
Fatal						
Event	124	149	159	194	84	
Person-years	172 718	217 500	239 420	273 860	101 096	
Age-adjusted[a]	1	1.00 (0.79, 1.28)	1.02 (0.80, 1.30)	1.03 (0.82, 1.31)	1.03 (0.77, 1.38)	0.76
MV1-adjusted[b]	1	1.01 (0.79, 1.29)	1.03 (0.81, 1.31)	1.05 (0.83, 1.33)	1.05 (0.78, 1.40)	0.71
MV2-adjusted[c]	1	1.00 (0.78, 1.27)	1.01 (0.79, 1.28)	1.00 (0.79, 1.27)	1.01 (0.75, 1.36)	0.94
Gleason score 2–6						
Event	465	729	731	937	311	
Person-years	172 395	216 965	238 870	273 166	100 885	
Age-adjusted[a]	1	1.10 (0.98, 1.24)	1.05 (0.93, 1.18)	1.11 (0.99, 1.25)	0.99 (0.86, 1.16)	0.93
MV1-adjusted[b]	1	1.04 (0.93, 1.18)	0.99 (0.88, 1.12)	1.04 (0.93, 1.17)	0.93 (0.80, 1.08)	0.50
MV2-adjusted[c]	1	1.03 (0.92, 1.16)	0.98 (0.87, 1.10)	1.02 (0.91, 1.15)	0.92 (0.79, 1.07)	0.43
Gleason score $\geqslant$7						
Event	264	434	526	661	233	
Person-years	172 575	217 252	239 075	273 405	100 979	
Age-adjusted[a]	1	1.15 (0.98, 1.34)	1.28 (1.10, 1.49)	1.25 (1.07, 1.45)	1.14 (0.95, 1.37)	0.33
MV1-adjusted[b]	1	1.11 (0.95, 1.30)	1.25 (1.07, 1.45)	1.19 (1.03, 1.39)	1.09 (0.91, 1.32)	0.60
MV2-adjusted[c]	1	1.10 (0.94, 1.29)	1.22 (1.05, 1.42)	1.16 (1.00, 1.35)	1.07 (0.88, 1.28)	0.86

Abbreviations: MV1 = multivariable model 1; MV2 = multivariable model 2.
[a]Age-adjusted model adjusted for age in months, time period (2-year intervals), and energy (kcal per day, quintiles).
[b]Multivariable model 1 adjusted for age in months, time period (2-year intervals), energy (kcal per day, quintiles), body mass index (<25, 25–29.9, and $\geqslant$30 kg m^{-2}), vigorous physical activity (<1, 1 to <3, and $\geqslant$3 h per week), smoking status (current, former, and never), and PSA screening history (yes, no, and unknown).
[c]Multivariable model 2 adjusted for all the variables in MV1 and family history of PCa (yes, no), ethnicity (Caucasian, African, and Asian), height (inches, quintiles), history of diabetes (yes, no), current multivitamin use (yes, no), current supplement use (yes, no), tomato sauce (servings per week, quartiles), coffee intake (servings per day, tertiles), and Mediterranean diet (score range 0–9).
[d]Advanced disease includes stage T3b-4, N1, M1, or prostate cancer-specific death.
[e]Lethal disease includes metastasis to bone or other organs at diagnosis or over follow-up or prostate cancer-specific death.

average from the date of diagnosis to end of follow-up. The FFQ immediately preceding diagnosis was used to classify the participants' diet from the diagnosis date until the next available FFQ, because it would better capture diet at the time of diagnosis without the diagnosis having affected diet. Similar secondary analyses to those above were performed. In addition, we included pre-diagnostic nut consumption from the 1986 FFQ in the multivariable models to mitigate the influence of pre-diagnostic diet.

RESULTS

Nut consumption and PCa incidence. During 26 years of follow-up, 6810 men were diagnosed with PCa. At baseline, men with higher nut consumption exercised more, took more vitamin supplements, had higher Mediterranean diet scores and drank more alcohol (Supplementary Table 1).

Nut consumption was not associated with being diagnosed with PCa (Table 1). Similarly, no significant associations were observed between peanut or other nut consumption and PCa incidence (data not shown). The null effect association remained unchanged when baseline nut intake was used as the main exposure, or after a 2-year lag period was added between nut-intake assessment and each follow-up period (data not shown). No significant interactions by age, BMI, or ethnicity were identified (all P interactions >0.05).

Nut consumption and mortality in PCa patients. Among the 4346 men diagnosed with non-metastatic PCa, 359 cases of lethal PCa, 264 cases of fatal PCa, and 1285 total deaths were identified. The mean duration of follow-up was 7.8 years for lethal PCa and 10.3 years for fatal PCa. Compared with non-consumers, patients with higher nut consumption were more likely to take vitamin supplements, less likely to have high blood pressure, consumed more alcohol, olive oil, and tomatoes, and had a higher Mediterranean diet score (Supplementary Table 2).

There were no statistically significant associations between nut consumption after diagnosis and development of lethal or fatal PCa (Table 2). But patients who consumed nuts five or more times per week had a 34% lower rate of overall mortality compared with those who consumed less than once per month (HR: 0.66, 95% CI: 0.52–0.83, P for trend $=0.0005$); (Table 2). We also observed a statistically significant difference in overall survival across nut-intake categories ($P<0.0004$, Supplementary Figure 1).

Further adjustment for pre-diagnostic baseline nut consumption or adding a 2-year lag period did not alter these findings (data not shown). No significant interactions by age, BMI, or ethnicity were identified (all P interactions >0.05). Although we observed no associations of peanuts or other nuts separately with lethal or fatal PCa, the HRs for total mortality were 0.70 (95% CI: 0.52–0.95; P for trend $=0.003$) for other nuts and 0.79 (95% CI: 0.59–1.06; P for trend $=0.01$) for peanuts, comparing five or more servings per week with less than once per month.

DISCUSSION

To our knowledge, this is the largest cohort study to prospectively assess the association of nut consumption with being diagnosed with PCa, including subtypes of aggressive PCa. Our null effect results with PCa incidence are consistent with those of the Adventists Health Study (180 PCa cases and 6-year follow-up) (Mills *et al*, 1989), although they did not examine different subtypes of PCa. In contrast, two case–control studies reported inverse associations (Jain *et al*, 1999; Raimondi *et al*, 2010). However, case–control studies are prone

Table 2. Hazard ratios and 95% confidence intervals for prostate-specific and all-cause mortality among men diagnosed with non-metastatic prostate cancer, according to the total nut consumption

	Frequency of total nut consumption (28 g serving)					
	Less than once per month	Less than once per week	Once per week	2–4 Times per week	⩾5 Times per week	*P* for trend
Nut-intake, servings per day	0–0.033	0.034–0.10	0.11–0.20	0.21–0.60	>0.60	
Lethal prostate cancer						
Event	52	66	73	122	46	
Person-years	6504	8633	9251	13 529	6706	
Age-adjusted[a]	1	0.88 (0.61, 1.27)	0.91 (0.63, 1.30)	1.03 (0.74, 1.45)	0.79 (0.52, 1.20)	0.72
MV1-adjusted[b]	1	0.87 (0.60, 1.26)	0.95 (0.66, 1.37)	1.06 (0.75, 1.49)	0.81 (0.53, 1.24)	0.83
MV2-adjusted[c]	1	0.92 (0.63, 1.33)	0.99 (0.68, 1.42)	1.13 (0.80, 1.59)	0.88 (0.57, 1.35)	0.89
Fatal prostate cancer						
Event	36	49	60	93	26	
Person-years	7726	9588	10 231	14 960	7489	
Age-adjusted[a]	1	0.87 (0.56, 1.35)	0.96 (0.63, 1.47)	1.00 (0.67, 1.48)	0.55 (0.32, 0.93)	0.09
MV1-adjusted[b]	1	0.92 (0.59, 1.43)	1.02 (0.66, 1.56)	1.10 (0.74, 1.65)	0.59 (0.35, 1.00)	0.17
MV2-adjusted[c]	1	0.91 (0.59, 1.43)	0.98 (0.64, 1.51)	1.16 (0.77, 1.74)	0.62 (0.36, 1.07)	0.38
All-cause mortality						
Event	203	266	289	380	147	
Person-years	7772	9588	10 231	14 960	7489	
Age-adjusted[a]	1	0.82 (0.68, 0.99)	0.80 (0.67, 0.97)	0.67 (0.56, 0.79)	0.52 (0.41, 0.65)	<0.0001
MV1-adjusted[b]	1	0.91 (0.75, 1.09)	0.90 (0.75, 1.08)	0.78 (0.65, 0.93)	0.58 (0.47, 0.73)	<0.0001
MV2-adjusted[d]	1	0.92 (0.76, 1.11)	0.91 (0.76, 1.10)	0.85 (0.71, 1.01)	0.66 (0.52, 0.83)	0.0005

Abbreviations: MV1 = multivariable model 1; MV2 = multivariable model 2.

[a]Age-adjusted model adjusted for age at diagnosis (years), time period (2-year intervals), time since diagnosis to FFQ (years), and energy (kcal per day, quintiles).

[b]Multivariable model 1 adjusted for age at diagnosis (years), time period (2-year intervals), time since diagnosis to FFQ (years), energy (kcal per day, quintiles), body mass index (<25, 25 to <30, and ⩾30 kg m^{-2}), vigorous physical activity (<1, 1 to <3, and ⩾3 h per week), smoking status (current, former and never), Gleason score (<7, 7, and >7), clinical T stage (T1, T2, and T3), and primary treatment (radical prostatectomy, radiation, hormonal therapy, active surveillance, and other).

[c]Multivariable model 2 for lethal/fatal PCa adjusted for all the variables of MV 1 and PSA screening history (yes, no, and unknown), family history of PCa (yes, no), ethnicity (Caucasian, African, and Asian), height (inches, quintiles), history of diabetes (yes, no), current multivitamin use (yes, no), current supplement use (yes, no), tomato sauce (servings per week, quartiles), coffee intake (servings per day, tertiles), and Mediterranean diet score (range: 0–9).

[d]Multivariable model 2 for all-cause mortality adjusted for all the variables of MV 2 for lethal/fatal PCa as well as family history of diabetes mellitus, of myocardial infarction, of cancer, and history of high blood pressure and elevated cholesterol (all defined as yes or no).

to recall bias and do not have long-term and repeated measures of dietary intake. In addition, Jain *et al*, (1999) combined nuts with beans and lentils, which could also explain the difference in results.

We observed a reasonably large, albeit non-significant, HR of 0.62 between post-diagnosis nut consumption and fatal outcome among PCa patients. This is consistent with a recent study that found an 18% lower rate of lethal PCa (HR, 0.82; 95% CI, 0.67–1.01) per daily serving increase of nut intake after diagnosis (Richman *et al*, 2013). In addition, our finding of a significant 34% rate reduction in overall mortality is consistent with other prospective studies, Richman *et al*, (2013) included, which have found inverse associations between nuts and mortality, with HRs ranging from 0.61 to 0.87 (Bao *et al*, 2013; Guasch-Ferré and Bulló, 2013; Richman *et al*, 2013; Hshieh *et al*, 2015; Luu *et al*, 2015).

Our study suggests that nuts, although not associated with being diagnosed with PCa, may still improve the overall survival of PCa patients. Among PCa patients in the HPFS, cardiovascular disease was the leading cause of death, accounting for nearly one-third of the deaths (Richman *et al*, 2013; Kenfield *et al*, 2014). Large cohort studies have consistently shown that increased nut consumption was associated with reduced cardiovascular disease incidence and mortality (Hu, 2003; Kelly and Sabaté, 2007; Kris-Etherton *et al*, 2008). Nuts are dense in nutrients and bioactive compounds that may confer cardio-protective, anti-inflammatory, and antioxidant properties (Kris-Etherton *et al*, 2008; Bao *et al*, 2013). Furthermore, nuts are rich in monounsaturated and polyunsaturated fats, and replacement of carbohydrates and animal fat with either unsaturated fats has been shown to reduce all-cause mortality and lethal outcomes among men with non-metastatic PCa (Richman *et al*, 2013).

The strengths of this study include its prospective design, large sample size, long follow-up time with excellent retention, and repeated measurement of diet and lifestyle factors. We were also able to reduce random measurement error by averaging nut intake cumulatively from multiple time points. However, there may still be residual confounding, although we adjusted extensively for risk factors for PCa development and survivorship.

In conclusion, nut consumption was not associated with PCa incidence or PCa-specific mortality in this large and prospective cohort of men. However, frequent nut consumption was associated with significantly lower overall mortality rate among men diagnosed with non-metastatic PCa.

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CONFLICT OF INTEREST

Dr Bao received a research grant from the International Tree Nut Council Nutrition Research & Education Foundation. The other authors declare no conflict of interest.

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