Alcohol consumption and risk of prostate cancer: The Harvard Alumni Health Study

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Background	Although many studies suggest that consumption of alcohol increases the risk of several site-specific cancers, the evidence remains unclear for prostate cancer. Few data exist on beverage-specific associations as well as lifetime patterns of alcohol consumption and prostate cancer risk.
Methods	We prospectively followed 7612 Harvard alumni (mean age 66.6 years) from 1988 through 1993, during which 366 cases of incident prostate cancer occurred. Self-reported alcohol consumption was assessed at baseline from wine, beer, and liquor intake. Previous assessments during college and in 1977 were also available.
Results	Overall, the mean total alcohol consumption in 1988 was 123.1 g/week, of which 28.6% was from wine, 15.8% from beer, and 55.6% from liquor. Compared to men reporting almost never drinking alcohol in 1988, the multivariate relative risks (95% CI) for 1 drink/month to <3 drinks/week, 3 drinks/week to <1 drink/day, 1 to <3 drinks/day, and ⩾3 drinks/day were 1.33 (0.88–2.01), 1.65 (1.12–2.44), 1.85 (1.29–2.64), and 1.33 (0.86–2.05), respectively. Wine or beer consumption was unassociated with prostate cancer; however, moderate liquor consumption was associated with a significant 61–67% increased risk of prostate cancer (*P*, non-linear trend < 0.001). Men initiating alcohol consumption between 1977 and 1988 had a twofold increased risk of prostate cancer compared to men with almost no alcohol consumption at both times.
Conclusions	In contrast to the majority of previous studies, we found a positive association between moderate alcohol consumption and the risk of prostate cancer. Liquor, but not wine or beer, consumption was positively associated with prostate cancer.
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A recent review of epidemiological studies on alcohol consumption and prostate cancer risk concluded that the available evidence shows no apparent increased risk among light-to-moderate drinkers.[1] Many studies have examined the association between alcohol consumption and prostate cancer, with the majority of studies finding no association.[2–38] However, because of limited data, Breslow and Weed[1] were unable to make any conclusions regarding heavy drinking or lifetime patterns of drinking. Few data exist regarding beverage-specific effects.

Alcohol may increase prostate cancer risk through several proposed mechanisms, including increased levels of plasma sex hormones, immunosuppression, or activation of carcinogenic metabolites.[39]

We therefore examined the association of total and beverage-specific alcohol consumption with prostate cancer risk using data from the Harvard Alumni Study, a prospective cohort study of middle-aged and older men. In addition, we sought to investigate whether lifetime patterns of alcohol consumption were associated with the risk of prostate cancer.

Methods

Subjects

The Harvard Alumni Study is an ongoing cohort study of the predictors of chronic diseases in men matriculating as undergraduates at Harvard University between 1916 and 1950. We first mailed a health questionnaire to surviving alumni in either 1962 or 1966, then periodically sent questionnaires to all surviving alumni in the relevant classes to update information

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on health habits and medical history. For this study, we were interested in information from a mailed questionnaire in 1988. Of 12 805 men returning the 1988 questionnaire, we excluded 1683 men reporting any history of physician-diagnosed cancer, 1678 men with incomplete data on alcohol consumption, and 419 men with missing data on other potential risk factors for prostate cancer. Of the remaining 8935 men, we successfully followed 7612 (i.e. they returned a subsequent questionnaire in 1993, or were known to have died by the end of 1993), or 85.2%.

Assessment of alcohol consumption and other risk factors

To assess alcohol consumption in 1988, we asked alumni to report their intake of wine, beer, and liquor (or spirits, e.g. whiskey) by responding to the question, 'How many servings of the following foods do you eat?' We assumed that one serving was equivalent to one unit of alcohol. Seven responses were possible for each alcoholic beverage, including almost never, 1–3 per month, 1–2 per week, 3–6 per week, 1–2 per day, 3–5 per day, and 6+ per day. We estimated total alcohol consumption by summing wine, beer, and liquor intake using the midpoints of the first six responses (with values of 0, 0.5, 1.5, 4.5, 10.5, and 28 drinks/week, respectively) and a conservative midpoint estimate for the seventh response (42 drinks/week). Based on the distribution of total and beverage-specific intake, we *a priori* collapsed the seven categories into five categories of intake: almost never, 1/month to <3/week, 3/week to <1/day, 1 to <3/day, and ≥3/day. Self-reports of alcohol intake using food frequency questions are reasonably reliable and valid, as indicated by previous studies of male health professionals and population-based groups.[40–42] Total alcohol consumption on the 1977 questionnaire was calculated in an identical manner as done for the 1988 questionnaire, summing intake from wine, beer and liquor. Alcohol consumption from the college physical exam was measured from an overall question without regard to specific beverage intakes.

Information was additionally collected from the 1988 questionnaire on age (categorized in analyses as <60, 60–<65, 65–<70, 70–<75, and ≥75 years), cigarette smoking (categorized in analyses as never smoker, former smoker, current smoker [<20 cigarettes/day, ≥20 cigarettes/day]), parental history of cancer (none, any), and weight and height (combined into body mass index, kg/m^2). On the 1988 questionnaire, we also asked alumni to report the daily number of flights of stairs climbed and city blocks walked, as well as to list all sports or recreational activities in which they had actively participated during the past year.[43] For each sport or activity listed, we asked for details regarding the frequency (weeks/year) and duration (time/week when active). From these data, we estimated total energy expenditure. Dietary information was collected using a 23-item food frequency questionnaire, from which we estimated red meat intake (<3 or ≥3 servings/week), vegetable intake (<3 or ≥3 servings/day), any vitamin/mineral supplement use (no, yes), total caloric intake (in kcal/day), and saturated fat intake (per cent of total calories).

Ascertainment of prostate cancer occurrence

We ascertained cases of prostate cancer through self-reports on the follow-up questionnaire sent in 1993. The date of diagnosis was taken as the reported year of diagnosis. The accuracy of self-reported, physician-diagnosed prostate cancer in Harvard alumni was confirmed 91% of the time.[44] In addition, deaths were compiled continuously by the Harvard Alumni Office, which maintains a listing of deceased alumni. We traced deaths through the end of 1993. For each death reported by the alumni office, we requested and obtained death certificates from the appropriate state. We included as prostate cancer endpoints deaths with prostate cancer listed as either the underlying or a contributing cause of death. Mortality follow-up in this cohort is over 99% complete.[45]

Data analyses

We first examined the distribution of baseline characteristics according to categories of alcohol consumption. We calculated person-years of follow-up from 1988 to the year in which prostate cancer was first reported, the year of death, or 1993, whichever occurred first. Relative risks (RR) and 95% CI for prostate cancer were calculated for each alcohol consumption category using Cox proportional hazards, always using the lowest level of alcohol consumption ('almost never') as the reference group. The assumption of proportional hazards was satisfied by testing for the interaction between follow-up time and categories of alcohol consumption ($P = 0.72$). Models were first adjusted for age, and multivariate models were further adjusted for the non-dietary risk factors described above. A second multivariate model was further adjusted for the dietary factors described above that may confound the association between alcohol consumption and the risk of prostate cancer. Tests for linear trend treated the five categories of alcohol consumption as a single ordinal variable, using the median values for each category. We then tested for the presence of non-linear trends with the addition of both the ordinal term and the square of the ordinal term in the model. Parallel analyses were performed for each alcoholic beverage type. In secondary analyses, we excluded men with prostate cancer during the first 2 years of follow-up to minimize any bias due to illnesses that might have affected baseline alcohol consumption. We also examined whether the exclusion of 1145 men with prevalent cardiovascular disease on the 1988 questionnaire altered any of the results.

Subjects in the present study had been asked additionally about alcohol consumption in the past at their college physical examination and on a questionnaire mailed in 1977. We examined 6686 men (87.8%) returning both the 1977 and 1988 questionnaires with follow-up from 1988 through the end of 1993 (282 prostate cancer cases). Alcohol consumption was initially cross-classified using the five categories of intake from each questionnaire, then categories were collapsed *a priori* to improve power since few men reported large changes in alcohol consumption from the 1977 to 1988 questionnaires. We then identified 4269 men (56.1%) with data on alcohol consumption from their college physical exam, 1977 questionnaire, and 1988 questionnaire. Lifetime alcohol consumption was classified as none (almost never at all three timepoints; n = 508) or any (any amount at all three timepoints; n = 1455); remaining men with other patterns of drinking were excluded from these analyses.

Results

The mean (standard deviation) age of the 7612 men at baseline in 1988 was 66.6 (7.7) years. Overall, the mean total alcohol

consumption in 1988 was 123.1 (136.3) g/week, of which 28.6% was from wine, 15.8% from beer, and 55.6% from liquor (or spirits, e.g. whiskey). The proportions of men reporting total alcohol consumption of almost never, 1/month to <3/week, 3/week to <1/day, 1/day to <3/day, and ≥3 drinks/day were 15.6%, 17.6%, 19.4%, 33.3%, and 14.2%, respectively. Although a similar number of men consumed wine or liquor, wine consumption was more moderate whereas liquor consumption was heavier. Table 1 provides data on the baseline

characteristics according to levels of alcohol consumption in 1988. Men who almost never drank alcohol tended to be older, consume more vegetables, and take vitamins or supplements. Men who consumed greater amounts of alcohol tended to have higher physical activity levels, yet were more likely to be current or former smokers and consume more red meat.

During a median follow-up of 5.0 years, there were 366 incident cases of prostate cancer. Table 2 provides the results for the association between total alcohol, wine, beer, and liquor

Table 1 Baseline characteristics of 7612 Harvard alumni according to levels of alcohol consumption in 1988

	Total alcohol consumption[a]				
	Almost never (n = 1186)	1/month to <3/week (n = 1337)	3/week to <1/day (n = 1480)	1/day to <3/day (n = 2531)	≥3/day (n = 1078)
Age (years)	67.6 ± 8.4[b]	66.0 ± 7.8	66.0 ± 7.6	66.8 ± 7.5	66.7 ± 7.3
Body mass index (kg/m^2)	24.7 ± 3.3	24.9 ± 3.1	24.8 ± 2.9	24.6 ± 2.8	24.8 ± 2.9
Physical activity (kcal/wk)	2380 ± 2702	2441 ± 2602	2824 ± 3004	2839 ± 3107	2904 ± 3707
Smoking status (%)					
Current (<20 cigarettes/day)	2.2	1.9	2.5	3.6	4.5
Current (≥20 cigarettes/day)	5.2	3.0	2.5	4.9	9.1
Former	47.6	49.7	53.0	57.1	63.4
Never	44.9	45.4	42.0	34.5	23.1
Parental history of cancer (%)	40.0	40.2	41.2	40.3	42.1
Red meat intake (≥3 servings/week)	33.2	25.5	27.8	30.4	38.1
Vegetable intake (≥3 servings/day)	18.7	15.2	13.6	13.6	16.5
Any vitamin/mineral supplement use (%)	49.9	45.1	43.0	42.7	46.1

[a] Number of drinks estimated from wine, beer, and liquor consumption.

[b] Mean ± standard deviation.

Table 2 Relative risks (RR) (95% CI) of prostate cancer according to level of alcohol consumption in 1988

	1988 alcohol consumption					
	Almost never	1/month to <3/week	3/week to <1/day	1/day to <3/day	≥3/day	P, trend[a]
Total alcohol						
No. of men (cases)	1186 (38)	1337 (54)	1480 (76)	2531 (151)	1078 (47)	
Person-years	5447	6306	6937	11 693	5033	
Age-adjusted RR	1.00 (ref.)	1.33 (0.88–2.02)	1.67 (1.13–2.47)	1.86 (1.31–2.66)	1.35 (0.88–2.07)	0.35*
Multivariate-adjusted RR[b]	1.00 (ref.)	1.33 (0.88–2.01)	1.65 (1.12–2.44)	1.85 (1.29–2.64)	1.33 (0.86–2.05)	0.40*
Wine						
No. of men (cases)	2358 (105)	2816 (145)	1123 (55)	1152 (54)	163 (7)	
Person-years	10 772	13 193	5295	5377	779	
Age-adjusted RR	1.00 (ref.)	1.26 (0.98–1.62)	1.23 (0.88–1.70)	1.14 (0.82–1.59)	1.07 (0.50–2.31)	0.86
Multivariate-adjusted RR[b]	1.00 (ref.)	1.25 (0.97–1.62)	1.22 (0.88–1.70)	1.13 (0.82–1.58)	1.05 (0.49–2.27)	0.91
Beer						
No. of men (cases)	3849 (183)	2714 (149)	586 (15)	402 (17)	61 (2)	
Person-years	17 642	12 814	2786	1890	284	
Age-adjusted RR	1.00 (ref.)	1.26 (1.01–1.57)	0.59 (0.35–1.00)	0.97 (0.59–1.59)	0.76 (0.19–3.08)	0.22
Multivariate-adjusted RR[b]	1.00 (ref.)	1.24 (0.99–1.54)	0.58 (0.34–0.99)	0.95 (0.58–1.57)	0.72 (0.18–2.90)	0.19
Liquor						
No. of men (cases)	2590 (93)	1938 (82)	1059 (68)	1687 (108)	338 (15)	
Person-years	12 020	9179	4924	7733	1560	
Age-adjusted RR	1.00 (ref.)	1.19 (0.89–1.61)	1.68 (1.23–2.29)	1.62 (1.22–2.13)	1.11 (0.64–1.91)	0.09*
Multivariate-adjusted RR[b]	1.00 (ref.)	1.18 (0.88–1.59)	1.67 (1.22–2.29)	1.61 (1.21–2.13)	1.12 (0.64–1.94)	0.10*

[a] Linear trend across categories of alcohol consumption.

[b] Adjusted for age (categorized as <60, 60–<65, 65–<70, 70–<75, and ≥75 years), body mass index, physical activity, cigarette smoking (never, former, current [<20 cigarettes/day, ≥20 cigarettes/day]), and parental history of cancer.

* P, non-linear trend < 0.05.

consumption and the risk of prostate cancer. Age was the strongest confounder in all models of alcohol consumption and the risk of prostate cancer. Additional adjustment for potential confounders including body mass index, physical activity, cigarette smoking, and parental history of cancer had a nominal effect on the observed RR. In sensitivity analyses, adjustment for history of hypertension, diabetes, or cardiovascular disease did not appreciably alter the RR. Finally, the exclusion of men with prostate cancer during the first 1, 2, or 3 years of follow-up resulted in no difference for the RR (data not shown).

Total alcohol consumption, particularly from 3/week to <3/day, was associated with an increased risk of prostate cancer. In a model testing for non-linearity, the beta/SE for the linear and squared terms for total alcohol consumption were +0.07/0.02 ($P < 0.001$) and –0.002/0.001 ($P < 0.001$), respectively, indicating that the maximum risk of prostate cancer associated with prostate cancer was at moderate levels. With additional adjustment for dietary factors, including red meat intake, vegetable intake, any vitamin/mineral supplement use, total caloric intake, and saturated fat intake, the risk estimates were slightly attenuated, with corresponding RR of 1.29, 1.50, 1.83, and 1.18, respectively (P, linear trend = 0.57; P, non-linear trend < 0.001). Excluding 1145 men with prevalent cardiovascular disease at baseline slightly increased the magnitude of the RR for increasing total alcohol consumption to 1.51, 1.61, 1.97, and 1.45, respectively. We also considered deciles of total alcohol consumption estimated from wine (10.8 g/drink), beer (13.2 g/drink), and liquor (15.1 g/drink), using no alcohol consumption (0 g/week) as the referent. The pattern of RR for increasing deciles of total alcohol consumption followed the same pattern as found for the categorical results.

Moderate wine consumption, from 1/month to <1/day, was associated with a non-significant 22–25% increased risk of prostate cancer in multivariate models. There was little apparent effect for levels of wine consumption ≥1 drink/day (P, linear trend = 0.91; P, non-linear trend = 0.49). Few men reported beer consumption ≥3/week, resulting in smaller case counts and wider 95% CI. Liquor consumption also exhibited a similar non-linear trend ($P < 0.001$) of increased risk of prostate cancer particularly among men consuming 3 drinks/week to <3 drinks/day, with a significant 61–67% increased risk.

We next considered alcohol consumption data provided by 6686 alumni on both the 1977 and 1988 questionnaires and the risk of prostate cancer (Table 3). As before, adjustment for confounders other than age had little impact on the RR. Compared to men reporting almost never drinking alcohol on both questionnaires, men who initiated alcohol consumption by 1988 had a multivariate RR (95% CI) of prostate cancer of 2.16 (0.92–5.07). Only 12.9% of men (47 cases of prostate cancer) reported any increases in alcohol consumption from 1977 to 1988, limiting our power. Such men with any increases in alcohol consumption may have at least a twofold increase in prostate cancer risk. Men were more apt to decrease their alcohol consumption; however, only 216 men (4.7%) went from ≥3/week in 1977 to almost never in 1988. Those who reduced their alcohol consumption from 1977 to 1988 still had a somewhat elevated risk of prostate cancer compared to men reporting almost never for alcohol consumption at both timepoints.

Finally, we considered whether lifetime alcohol consumption, defined as any amount of alcohol consumption on the college physical exam, 1977, and 1988 questionnaire, was associated

Table 3 Age- and multivariate-adjusted relative risks (95% CI) of prostate cancer from 1988 to 1993 for alcohol consumption on the 1977 and 1988 questionnaires among 6686 Harvard alumni

1977 alcohol consumption	1988 alcohol consumption				
	Almost never	1/month to <3/week	3/week to <1/day	1/day to <3/day	≥3/day
Almost never	**drank the same** 519 [13][a] 1.00 (ref.)[b] 1.00 (ref.)[c]	**drank more** 178 [9] 2.12 (0.90–4.95) 2.16 (0.92–5.07)			
1/month to <3/week	**drank less** 171 [4] 0.95 (0.31–2.90) 0.95 (0.31–2.91)	**drank the same** 280 [12] 1.86 (0.85–4.09) 1.86 (0.85–4.09)		**drank more** 141 [10] 3.12 (1.37–7.12) 3.12 (1.37–7.12)	
3/week to <1/day	**drank less** 619 [30] 2.11 (1.10–4.05) 2.10 (1.09–4.02)		**drank the same** 401 [18] 1.97 (0.96–4.01) 1.97 (0.96–4.02)	**drank more** 219 [11] 2.13 (0.96–4.76) 2.16 (0.97–4.82)	
1/day to <3/day	**drank less** 350 [14] 1.65 (0.77–3.50) 1.68 (0.79–3.59)		**drank less** 670 [35] 2.18 (1.15–4.11) 2.15 (1.14–4.07)	**drank the same** 1413 [90] 2.54 (1.42–4.54) 2.51 (1.40–4.49)	**drank more** 327 [17] 2.03 (0.99–4.19) 2.02 (0.98–4.16)
≥3/day	**drank less** 255 [8] 1.30 (0.54–3.15) 1.28 (0.53–3.11)			**drank less** 547 [34] 2.54 (1.34–4.81) 2.53 (1.33–4.83)	**drank the same** 596 [19] 1.27 (0.63–2.58) 1.27 (0.62–2.58)

[a] Number of men (cases of prostate cancer).

[b] Adjusted for age (categorized as <60, 60–<65, 65–<70, 70–<75, and ≥75 years).

[c] Adjusted for age (categorized as <60, 60–<65, 65–<70, 70–<75, and ≥75 years), body mass index, physical activity, cigarette smoking (never, former, current [<20 cigarettes/day, ≥20 cigarettes/day]), and parental history of cancer.

with the risk of prostate cancer. Compared to men reporting no lifetime alcohol consumption (n = 508; 12 cases of prostate cancer), those who consistently consumed any alcohol (n = 1455; 61 cases of prostate cancer) had age- and multivariate-adjusted RR (95% CI) of 1.74 (0.93–3.23) and 1.73 (0.92–3.27), respectively. Additional adjustment for the dietary factors modestly attenuated the RR to 1.53 (0.79–2.97).

Discussion

We found a positive association between total alcohol consumption and the risk of prostate cancer in a cohort of older, male alumni. There was a dose-response effect up to <3 drinks/day that weakened among men consuming ⩾3 drinks/day. When we considered beverage-specific effects, only liquor (or spirits, e.g. whiskey) consumption exhibited a similar pattern to that for total alcohol consumption. Finally, men who maintained or increased their total alcohol consumption during an 11-year period had an approximate twofold increased risk of prostate cancer compared to men with no consumption during the same period. The consideration of alcohol consumption during college did not add information beyond the two assessments during adult years.

The present study suggests that there may be differential effects for wine, beer, and liquor consumption on the risk of prostate cancer within the moderate range of intake. Few men consumed large enough amounts of individual alcoholic beverages to reliably assess intake using higher categories than the ⩾3/day used in our analyses. Moderate liquor consumption ranging from 3/week to <3/day was associated with a 60% increased risk of prostate cancer. On the other hand, there was no significant association between wine consumption and risk of prostate cancer. We found a possible reduction in prostate cancer risk for beer, but only for intake ranging from 3 drinks/ week to <1 drink/day. The lack of a linear or non-linear trend for increasing beer consumption suggests that this may be a chance finding. However, the presence of oestrogenic substances, which may be inversely associated with prostate cancer, in beer may explain our results.[46] It is important to consider that the beverage-specific differences for the risk of prostate cancer are difficult to interpret using statistics to distinguish these associations.

The aetiology of prostate cancer remains poorly elucidated, with few well-established behavioural or dietary factors.[47] Alcohol consumption has been shown to be associated with cancers of the oropharynx, larynx, oesophagus, and liver.[20,48] Moderate alcohol intake also appears to be associated with the risk of breast cancer.[49] Several biological mechanisms may underlie an association between alcohol consumption and prostate cancer. Alcohol may increase prostate cancer risk by affecting the composition and functioning of cell membranes, causing free radical generation, affecting the metabolism of detoxification enzymes, depressing levels of DNA repair enzymes, or impairing immune function.[39] Higher levels of alcohol consumption may also be associated with insufficient dietary intake of key macronutrients or micronutrients that may increase the risk of prostate cancer.

Our follow-up period of 5 years may be inadequate to precisely capture any effect of alcohol consumption on the risk of prostate cancer. The long latent period for prostate cancer suggests that chronic patterns in diet, behaviour, and other potential preventive measures may be better suited for study. However, by starting follow-up in 1988, at which time the mean age was 66.6 years, we were better able to capture lifetime patterns of alcohol consumption. We found that increasing alcohol consumption from middle to late adulthood may increase the subsequent risk of prostate cancer. When we examined men reporting any alcohol consumption starting in college, we did not find any appreciable differences in the RR compared with the results limited to the 1988 questionnaire. This finding that alcohol may have deleterious effects on prostate cancer development in middle-aged and older men is consistent with the aetiologically relevant period of prostate carcinogenesis.

Some methodological limitations should be also considered. First, the measurement of alcohol consumption may be susceptible to misclassification. If random with respect to the occurrence of prostate cancer, this would bias our results toward the null. Alternatively, misclassification among heavier drinkers who underestimated their alcohol intake may underlie the observed increased risk of prostate cancer in moderate drinkers. However, previous validation studies suggest that self-reported alcohol consumption is reasonably reliable and valid,[40–42] so any misclassification should only modestly affect our risk estimates. In addition, our measurement of alcohol consumption in the distant past was based on information collected in the past, thereby increasing precision. Second, the increase in prostate-specifc antigen (PSA) screening during the follow-up period may explain our results if moderate drinkers tended to be screened more frequently, thus increasing their likelihood of a prostate cancer diagnosis. However, this is unlikely, since PSA screening is associated with healthier behaviours.[50] Next, we did not collect information on the stage of prostate cancer at diagnosis, which would have allowed us to determine whether alcohol has differential effects on quiescent or aggressive tumours. Finally, uncontrolled confounding may explain our results. Higher levels of alcohol consumption in male alumni may be associated with other dietary or biochemical markers, which in turn increase the risk of prostate cancer. However, given the lack of knowledge for other relevant potential confounders, it remains unclear whether residual confounding would explain our results.

Since most studies found no relation between alcohol and prostate cancer, it is important to consider why we found a positive association. The higher socioeconomic status of Harvard alumni compared to previous studies may have affected PSA screening rates and subsequent prostate cancer diagnoses. It is also possible that the relevant exposure period for alcohol in the aetiology of prostate carcinogenesis is at older ages; however, previous studies have generally investigated younger men.

In conclusion, we found a positive association between moderate alcohol consumption and the risk of prostate cancer in a cohort of older men. Liquor consumption, rather than wine or beer consumption, appeared to account for this increased risk. The consideration of past alcohol consumption as far back as college time did not appreciably alter our results. This suggests that any effect of alcohol consumption may occur later in the development of prostate cancer. Alternatively, those

who drink tend to continue drinking, so the observed effects on prostate cancer may reflect a long-term consequence of alcohol consumption. Further studies should seek to capture lifetime patterns of total and beverage-specific alcohol consumption to clarify these issues in relation to the risk of prostate cancer risk.

Acknowledgement

KEY MESSAGES

- In contrast to previous studies, we found a positive association between total alcohol consumption and the risk of prostate cancer in a prospective cohort of older, male Harvard alumni, with a dose-response effect up to <3 drinks/day that weakened among men consuming ≥3 drinks/day.

- When we considered beverage-specific effects, only liquor (or spirits, e.g. whiskey) consumption exhibited a similar pattern of a positive association with the risk of prostate cancer as that for total alcohol consumption.

- Men who maintained or increased their total alcohol consumption during an 11-year period had an approximate twofold increased risk of prostate cancer compared to men with no consumption during the same period; the consideration of alcohol consumption during college did not add information.

- Further studies should seek to capture lifetime patterns of total and beverage-specific alcohol consumption to clarify these issues in relation to the risk of prostate cancer risk.

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