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ORIGINAL PAPER

Moderate alcohol intake and cancer: the role of underreporting

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Abstract
Purpose There is compelling evidence that heavy alcohol drinking is related to increased risk of several cancer types, but the relationship of light–moderate drinking is less clear. We explored the role of inferred underreporting among light–moderate drinkers on the association between alcohol intake and cancer risk.
Methods In a cohort of 127,176 persons, we studied risk of any cancer, a composite of five alcohol-associated cancer types, and female breast cancer. Alcohol intake was reported at baseline health examinations, and 14,880 persons were subsequently diagnosed with cancer. Cox proportional hazard models were controlled for seven covariates. Based on other computer-stored information about alcohol habits, we stratified subjects into 18.4 % (23,363) suspected of underreporting, 46.5 % (59,173) not suspected of underreporting, and 35.1 % (44,640) of unsure underreporting status.
Results Persons reporting light–moderate drinking had increased cancer risk in this cohort. For example, the hazard ratios (95 % confidence intervals) for risk of any cancer were 1.10 (1.04–1.17) at <1 drink per day and 1.15 (1.08–1.23) at 1–2 drinks per day. Increased risk of cancer was concentrated in the stratum suspected of underreporting. For example, among persons reporting 1–2 drinks per day risk of any cancer was 1.33 (1.21–1.45) among those suspected of underreporting, 0.98 (0.87–1.09) among those not suspected, and 1.20 (1.10–1.31) among those of unsure status. These disparities were similar for the alcohol-related composite and for breast cancer.
Conclusions We conclude that the apparent increased risk of cancer among light–moderate drinkers may be substantially due to underreporting of intake.

Keywords Cancer · Risk factors · Alcohol drinking · Underreporting · Epidemiology

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Introduction

Heavy alcohol intake, often defined as usual or average intake of ≥3 standard-sized drinks per day in men and ≥2 standard-sized drinks per day in women, has been associated in observational studies with increased risk of cancers of the oral cavity, pharynx, larynx, esophagus, liver, large intestine, and female breast [1, 2]. Because of conflicting data the role of light–moderate drinking in cancer risk has been less clear, with evidence for increased risk probably

most consistent for female breast cancer [3, 4]. The subject has been thrust into public health consciousness by meta-analyses and reviews leading to statements that the alcohol–cancer association is continuous at all drinking levels and that there is no safe amount of alcohol intake from the viewpoint of cancer risk [5–7].

Tobacco use is a potential confounder of the alcohol–cancer associations, as alcohol drinking and smoking are associated habits in many populations [8]. Incomplete control for smoking might result in spurious associations with alcohol drinking. Other potential confounders of observed alcohol–cancer associations include chronic infections, dietary habits, adiposity, exercise, occupational exposures, air pollution, radiation, food additives, and other chemicals [9]. However, most of these probably do not have sufficiently strong associations with drinking to explain the observed alcohol–cancer associations.

Underreporting of alcohol intake is another source of bias with respect to adverse effects of light–moderate drinking. Intentionally or unintentionally, some heavy drinkers misstate the amount they drink, producing misclassifications in most survey databases. We attempted to study this aspect in a cross-sectional analysis of alcohol drinking and systemic hypertension by identifying a subgroup of "light–moderate" drinkers more likely by inference to be underreporters [10, 11]. The results suggested that observed higher prevalence of hypertension at 1–2 drinks per day was limited to suspect underreporters. We here apply similar methodology to a study of the association of alcohol drinking with incident cancer.

Materials and methods

Study population and data

Study protocols were approved by the Institutional Review Board of the Kaiser Permanente Medical Care Program. We studied a multi-ethnic cohort of 127,176 persons free of cancer history, who were members of a comprehensive prepaid health care program in Northern California. Baseline data were from special alcohol research questionnaire offered at voluntary routine health examinations from 1978 to 1985. More than 80 % of examinees completed the alcohol research questionnaire; most of those that did not complete it took the examination in the absence of a special research clerk. The examination procedure has been described in detail elsewhere [12, 13]. It included health measurements, self-classified ethnicity, and check-sheet queries about socio-demographic status, habits, medical history, and symptoms. Persons responding "yes" to "Did you drink alcohol in the past year?" were asked "How many alcoholic drinks (wine beer, whiskey, cocktails) did you usually have in the past year?" Persons responding "no" to drinking in the past year were asked "did you drink alcohol in the past?" These queries enabled baseline categorization into lifelong abstainers, ex-drinkers, and persons drinking <1 drink per day, 1–2 drinks per day, and ≥3 drinks per day. For subjects with more than one examination in 1978–1985, data from the first were used.

At baseline examinations from January, 1978 to October, 1979, 37,620 persons had aspartate aminotransferase (AST) determinations. From January, 1978 to August, 1981, 69,904 persons had alanine aminotransferase (ALT) determinations. Except for examination date, there were no other selection factors.

Cancer subjects

Cancer ascertainment was from the Health Care Program's Cancer Registry, which covers all subscribers and contributes to the local surveillance, epidemiology, and end results (SEER) program. There were 14,880 subjects (7,517 men and 7,563 women) with codes 140–209 of the International Classification of Diseases, Ninth edition, Clinical Modification (ICD-9-CM).

Table 1 presents data about selected traits of the study population and the cancer subjects.

"Likely" underreporters

Likely persons either reported heavier intake at another time or had an alcohol-related diagnosis (ARD) (death certificate, hospitalization, or outpatient) at some time. Self-reports of heavier intake were possible either on a later alcohol research questionnaire between 1978 and 1985 or on another computerized health examination questionnaire. The health plan computer bases included some alcohol intake data spanning a much longer period of time from 1964 to 1991. The alcohol-related diagnoses included the following ICD-9 codes: 291 (alcoholic psychosis), 303 (alcohol dependence syndrome), 305.0 (alcohol abuse), 790.3 (excess blood alcohol level), E860-860.1 (alcohol poisoning), 265.2 (pellagra), 357.5 (alcoholic polyneuropathy), 425.5 (alcoholic cardiomyopathy), 535.3 (alcoholic gastritis), 571.0 (alcoholic fatty liver), 571.1 (acute alcoholic hepatitis), 571.2 (alcoholic cirrhosis), and 571.2 (alcoholic liver disease, unspecified). Death certificate diagnoses in California were available from the index examination onward; health plan facility hospitalization diagnoses (primary or secondary) were available from 1972 onward; outpatient diagnoses were available at most health plan facilities from January, 1994.

There were 23,363 inferentially *Likely* underreporters. Of these 13,995 (59.9 %) reported heavier drinking on

Table 1 Selected baseline traits of study population and subjects with subsequent cancer

Group	N in study population	N any cancer	N alcohol-related cancer*
All	127,176 (100 %)	14,880 (100 %)	6,818 (100 %)
Sex—race/ethnicity			
Men	56,270 (44.3)	7,517 (50.5)	2,191 (32.1)
Women	70,906 (55.8)	7,363 (49.5)	4,627 (67.9)†
Black	34,167 (26.9)	8,996 (60.5)	4,032 (59.1)
White	70,721 (55.6)	4,076 (27.4)	1,933 (28.4)
Asian	13,452 (10.6)	1,148 (7.7)	555 (8.1)
Hispanic	5,741 (4.5)	440 (3.0)	192 (2.8)
Other race	2,860 (2.3)	201 (1.4)	98 (1.4)
Baseline age			
<40 years	70,556 (55.5)	3,056 (20.5)	1,360 (20.0)
40–49 years	21,118 (16.6)	3,148 (21.2)	1,431 (21.0)
50–59 years	18,544 (14.6)	4,359 (29.3)	2,047 (30.0)
60–69 years	12,289 (9.7)	3,289 (22.1)	1,542 (22.6)
≥70 years	4,669 (3.7)	1,028 (6.9)	438 (6.4)
Reported baseline alcohol drinking			
Never drink	15,165 (11.9)	1,708 (11.5)	837 (12.3)
Ex-drinker	4,096 (3.2)	645 (4.3)	295 (4.3)
<1 drink per day	74,476 (58.6)	7,880 (53.0)	3,661 (53.7)
1–2 drinks per day	23,048 (18.1)	3,134 (21.1)	1,334 (19.6)
≥3 drinks/day	10,391 (8.2)	1,513 (10.2)	691 (10.1)
Reported baseline cigarette smoking			
Never smoker	60,933 (47.9)	5,962 (40.1)	2,564 (37.6)
Ex-smoker	28,129 (22.1)	4,117 (27.7)	1,649 (24.2)
<1 pack per day	21,371 (16.8)	2,370 (15.9)	1,241 (18.2)
≥1 packs per day	11,722 (9.2)	1,809 (12.2)	1,069 (15.7)
Smoking unknown	5,021 (4.0)	622 (4.2)	295 (4.3)
Underreport status‡			
Likely	23,363 (18.4)	4,515 (30.3)	2,126 (31.2)
Unlikely	59,173 (46.5)	3,640 (24.5)	1,687 (24.8)
Unsure	44,640 (35.1)	6,725 (45.2)	3,005 (44.1)

* Cancers of the UAD tract, liver, colo-rectum, lung, and female breast

† 2,858 (61.6 %) female breast cancer

‡ See text for definition

another questionnaire, 14,717 (63.0 %) had an ARD and 5,349 (22.9 %) had both. The intervals between the index alcohol intake measure and the indicator of *Likely* ranged from −21 to +26 years.

Table 2 Distribution of persons according to classification of underreporting status as likely, unlikely, or unsure

Group	N likely underreporter (row %)	N unlikely underreporter (row %)	N unsure underreporter (row %)
All	23,363 (18.4)	59,173 (46.5)	44,640 (35.1)
Sex—ethnicity			
Men	14,295 (25.4)	24,642 (43.8)	17,333 (30.8)
Women	9,068 (12.8)	34,531 (48.7)	27,307 (38.5)
Black	7,525 (22.0)	14,295 (41.8)	12,347 (36.1)
White	13,714 (19.4)	31,375 (44.4)	25,632 (36.2)
Asian	773 (5.7)	8,541 (63.5)	4,138 (30.8)
Hispanic	893 (15.6)	3,164 (55.1)	1,684 (29.3)
Other race/ethnicity	458 (16.0)	1,563 (54.7)	839 (29.3)
Baseline age			
<40 years	9,034 (12.8)	43,137 (61.1)	18,385 (26.1)
40–49 years	5,058 (24.0)	7,525 (35.6)	8,535 (40.4)
50–59 years	5,013 (27.0)	4,868 (26.3)	8,663 (46.7)
60–69 years	3,306 (26.9)	2,751 (22.4)	6,232 (50.7)
≥70 years	952 (20.4)	892 (19.1)	2,825 (60.5)
Reported baseline alcohol drinking			
Never drink	663 (4.4)	10,325 (68.1)	4,177 (27.5)
Ex-drinker	968 (23.6)	2,345 (57.3)	783 (19.1)
<1 drink per day	7,645 (10.3)	35,953 (48.3)	30,878 (41.5)
1–2 drinks per day	6,025 (26.1)	8,902 (38.6)	8,121 (35.2)
Reported baseline cigarette smoking			
Never smoker	4,984 (8.2)	32,126 (52.7)	23,823 (39.1)
Ex-smoker	6,351 (22.6)	10,864 (38.6)	10,914 (38.8)
<1 pack per day	5,938 (27.8)	9,267 (43.4)	6,166 (28.9)
≥1 packs per day	5,101 (43.5)	4,185 (35.7)	2,436 (20.8)
Smoking unknown	989 (19.7)	2,731 (54.4)	1,301 (25.9)

See text for definitions

"Unlikely" and "unsure" underreporters

Persons with at least two computer-stored examinations (index measurement and at least one other before or after) and no indicator of *Likely* were classified as *Unlikely*. The actual median number of examinations for Unlikely persons was four. Persons with only one computer-stored alcohol intake report were classified as *Unsure* with respect to underreporting.

Table 2 shows selected distributions of the *Likely*, *Unlikely* and *Unsure* groups.

Analytic methods

Subjects were followed through 2008, cancer diagnosis, or termination of health plan membership. Mean follow-up was 18.2 years, yielding an estimated 2,365,000 person-years of follow-up. Analyses used the Cox proportional hazards model, yielding hazard ratio (HR), estimates, 95 % confidence intervals (CI), and p values. Alcohol drinking was studied categorically, with lifelong abstainers as referent; other categories were ex-drinkers and drinkers of <1, 1–2, or ≥3 drinks per day. In addition to alcohol categories, covariates in most models were age (continuous), ethnicity (white referent, black, Asian, Hispanic, and others), education (no college referent, some college, and college graduate), body mass index (<25 kg/m^2 referent, 25–29 kg/m^2, ≥30 kg/m^2), marital status (married referent, never married, and formerly married), cigarette smoking (never smoked referent, ex-smoker, <1 pack per day [ppd], and ≥1 ppd). We use the term "significant" for p values < 0.05. We studied risk of any cancer, risk of a composite of five cancer types with alcohol-associated risk in this cohort (UAD, lung, liver, female breast, and colorectal), and risk of female breast cancer alone.

In view of the importance of smoking as a potential confounder, we performed additional models with more smoking categories (<½ ppd, ½–1 ppd, 1–2 ppd, and ≥2 ppd). In view of the long follow-up after our measure of alcohol intake, we performed models for follow-up time strata (<10, 10–20, and ≥20 years.

Likely versus *Unlikely* underreporting was studied in relation to high AST and ALT levels, defined as the highest 5 % for each sex. Analyses were carried out for all persons and separately for persons drinking <1 and 1–2 drinks per day, controlling for age, sex, race/ethnicity, and cigarette smoking.

In this article, persons reporting <1 drink per day and 1–2 drinks per day are called "light–moderate" drinkers.

Results

Table 2 shows that prevalence of *Likely* status was higher in men, in persons 40–70 years of age, and in smokers, and *Likely* prevalence was quite low in Asians. Among light–moderate drinkers prevalence of *Likely* was 10 % among drinkers reporting <1 drink per day and 26 % among reporters of 1–2 drinks per day.

Table 3 presents the adjusted relationships at the index examination of light–moderate alcohol drinking with risk of any cancer and of the alcohol-related composite by sex and underreport status. In both sexes, there was increased risk of cancer among these drinkers but it was concentrated in the *Likely* group. In fact, there was no significant relationship in the *Unlikely* persons. The *Unsure* groups were intermediate with respect to risk of cancer.

Table 3 Risk of cancer in light–moderate drinkers versus lifelong abstainers—HR (95 % CI) by underreport status*

Group	<1 drink per day	1–2 drinks per day
Any cancer		
All in category	1.10 (1.04–1.17)[a]	1.15 (1.08–1.23)[a]
Likely*	1.36 (1.24–1.48)[a]	1.33 (1.21–1.45)[a]
Unlikely*	1.00 (0.92–1.09)	0.98 (0.87–1.09)
Unsure*	1.13 (1.05–1.22)[b]	1.20 (1.10–1.31)[a]
Alcohol-related cancer		
All in category	1.12 (1.03–1.21)[b]	1.2 0 (1.09–1.32)[a]
Likely*	1.42 (1.25–1.61)[a]	1.44 (1.26–1.65)[a]
Unlikely*	1.06 (0.94–1.20)	1.05 (0.89–1.24)
Unsure*	1.11 (0.99–1.23)	1.14 (1.00–1.30)

* "Likely" persons reported these categories on index examination but reported heavy (≥3 drinks per day) on another occasion or had an alcohol-related diagnosis (ARD) at some time. "Unlikely" persons consistently reported no heavy intake and had no ARD. "Unsure" persons had only one computer-stored alcohol intake report

[a] p value < 0.001

[b] p value < 0.01

Figure 1 presents graphically the adjusted relationships at the index examination of all alcohol drinking categories to risk of any cancer as well as the relationships of *Likely* and *Unlikely* underreporters among persons reporting light–moderate intake. Figure 2 presents similar data for the composite of alcohol-related cancer. Figure 3 presents data for female breast cancer. Among 3,200 women with breast cancer, the HRs (CI) for alcohol categories were as follows: ex-drinkers = 1.31 (1.04–1.66), <1 drink per day = 1.14 (1.02–1.28), 1–2 drinks per day = 1.23 (1.07–1.43), and ≥3 drinks per day = 1.35 (1.10–1.65). *Likely* breast cancer subjects comprised 14.8 % (281/1,898) of those reporting <1 drink per day and 30.3 % (162/534) of those reporting 1–2 drinks per day. Among *Likely*, the breast cancer HR was 1.48 (1.23–1.79, $p < 0.001$) at <1 drink per day and 1.39 (1.12–1.74, $p = 0.003$) at 1–2 drinks per day; the corresponding risks for unlikely were 1.05 (0.89–1.24) and 1.06 (0.81–1.37).

Alcohol-associated cancer risks were little affected by addition of more smoking categories. For example, for 1–2 drinks per day and ≥3 drinks per day, the HRs for alcohol-related cancer were 1.22 (1.13–1.33) and 1.48 (1.35–1.64), respectively, in the model with four smoking categories; with six smoking categories in the model, the corresponding numbers were 1.20 (1.10–1.32) and 1.45 (1.30–1.62). There was slight attenuation of alcohol-associated risk with passage of time, but it persisted beyond 20 years from baseline. For example, for alcohol-associated cancer, the HRs for persons reporting 1–2 drinks per day were 1.35 (1.16–1.56) for cancer diagnosis within

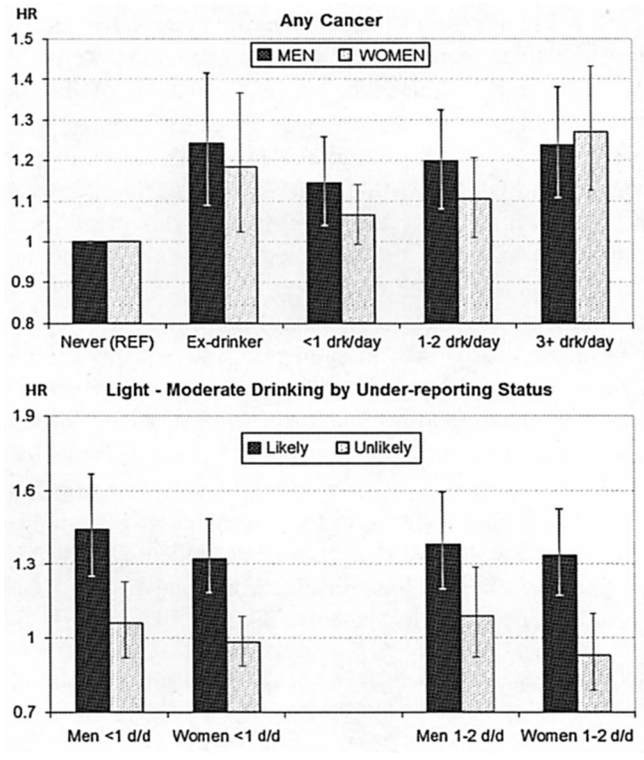

Fig. 1 *Upper panel.* Adjusted RR (95 % CI) of any cancer among non-drinkers, ex-drinkers, light–moderate drinkers, and heavy drinkers. *Lower panel.* Adjusted RR (95 % CI) of any cancer among light–moderate drinkers according to suspicion of underreporting alcohol intake. See text and footnote in Table 3 for definitions of Likely and Unlikely

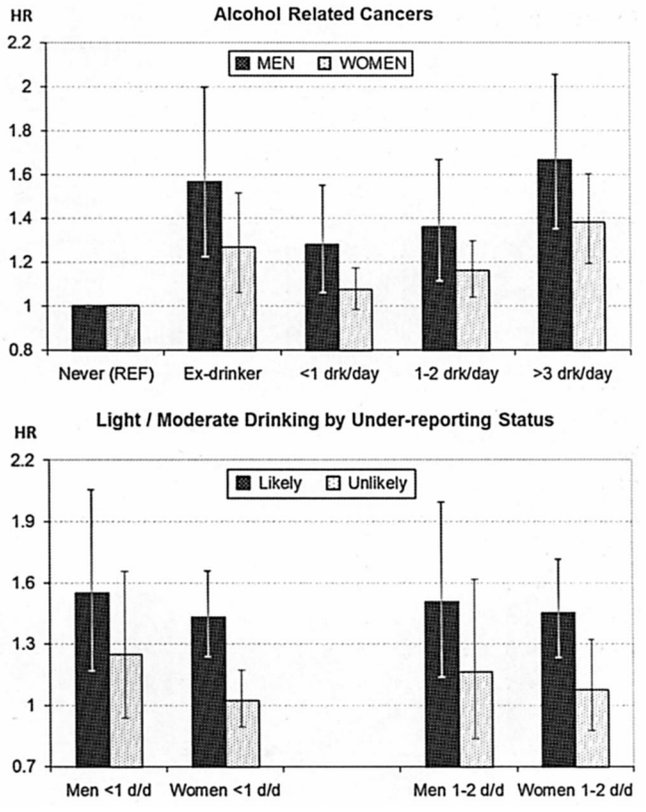

Fig. 2 *Upper panel.* Adjusted RR (95 % CI) of alcohol-related cancer (UAD, lung, liver, large intestine, breast) among non-drinkers, ex-drinkers, light–moderate drinkers, and heavy drinkers. *Lower panel.* Adjusted RR (95 % CI) of alcohol-related cancer among light–moderate drinkers according to suspicion of underreporting alcohol intake. See text and footnote in Table 3 for definitions of Likely and Unlikely

10 years, 1.24 (1.07–1.43), for cancer diagnosis at 10–20 years, and 1.21 (1.05–1.38) for cancer diagnosis at ≥20 years; for those reporting ≥3 drinks per day, these numbers were 1.69 (1.42–2.02), 1.53 (1.29–1.80) and 1.43 (1.22–1.69).

For all persons, the adjusted HR of *Likely* versus *Unlikely* for high AST was 1.42 (1.23–1.65, $p < 0.001$) and for high ALT, it was 1.43 (1.29–1.49, $p < 0.001$). Among persons reporting <1 drink per day, these values were 1.19 (0.94–1.50) for AST and 1.22 (1.04–1.44, $p = 0.01$) for ALT, and among persons reporting 1–2 drinks, they were 1.57 (1.17–2.11, $p = 0.003$) for AST and 1.60 (1.30–1.97, $p < 0.001$) for ALT.

Discussion

The main finding in these data is the absence of a relation of light–moderate drinking to risk of cancer in the *Unlikely* subgroup. These persons comprise almost half (46.5 %) of all examinees in our study population. Thus, the apparent increased risk of cancer among all light–moderate drinkers in our data and in other reports may be exaggerated or spurious.

We have previously summarized evidence supporting the substantial consensus that underreporting of alcohol intake by heavier drinkers is frequent [10]. In the data reported here, the greater likelihood of high liver transaminase enzymes in *Likely* than in *Unlikely* persons provides another line of evidence that *Likely* light–moderate drinkers were probably taking more alcohol than those in the *Unlikely* group. Misclassification consequent to underreporting can cause a true threshold level for an alcohol effect to appear as a continuous dose–response relationship [9].

Determination of health measurements and alcohol habits only at baseline is a limitation of our study, but there is known relative stability of drinking in this population [14, 15]. In a follow-up survey of a subgroup of relatively elderly subjects in 2000, approximately 20 years from baseline [15], 52 % reported identical intake to that reported in 1978–1985. Of those reporting a change, the proportions of persons reporting decreased total drinking in 2000 substantially outnumbered those for increased drinking (33 vs. 15 %, $p < 0.001$). Relevant to this issue

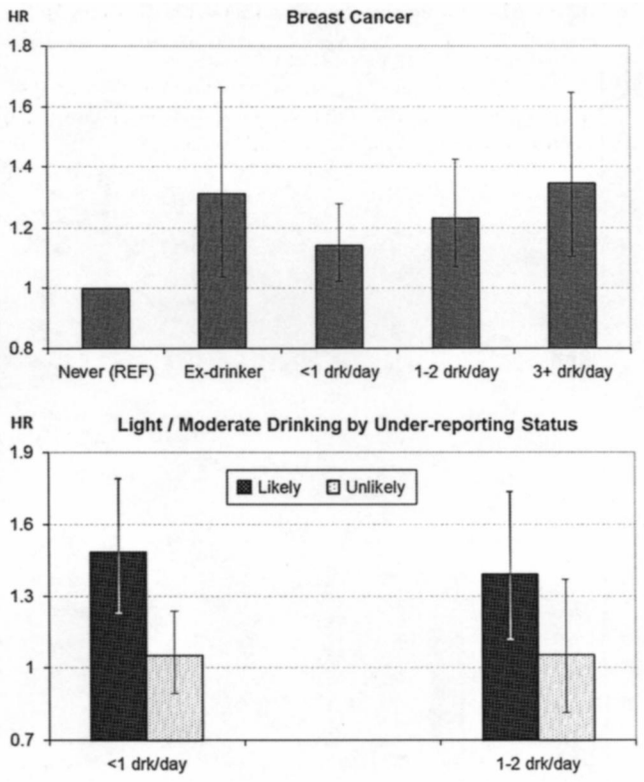

Fig. 3 *Upper panel.* Adjusted RR (95 % CI) of female breast cancer among non-drinkers, ex-drinkers, light–moderate drinkers, and heavy drinkers. *Lower panel.* Adjusted RR (95 % CI) of breast cancer among light–moderate drinkers according to suspicion of underreporting alcohol intake. See text and footnote in Table 3 for definitions of Likely and Unlikely

are the data showing persistence of alcohol–cancer associations at 10–20 years, and at ≥20 years, albeit with some weakening with longer follow-up. The most plausible explanation for this late weakening is reduced mean alcohol intake with aging.

Some proportion of persons labeled *Likely* because of reported heavier drinking on another occasion probably were telling the truth on both occasions and thus were not really underreporting. Although the greater prevalence of high liver enzymes in the *Likely* subgroup suggests that some were underreporting, the query asking for a year-long summary intake estimate left room for the existence of *Likely* status in some persons who correctly estimated their yearly average but drank more alcohol in the days or weeks before the index examination. Conversely, some *Unlikely* light–moderate drinkers were probably underreporters, but in aggregate this group did not have increased cancer risk compared to those reporting abstinence (some of whom were also probably underreporting). We cannot determine which individuals were truly underreporters, but feel certain that the *Likely* subgroup included a disproportionate number.

Our findings could plausibly be influenced by uncontrolled residual confounding by traits related to *Likely* and *Unlikely* status. Since the median number of health examinations among *Unlikely* was four, these persons with multiple examinations might be especially "health-conscious" individuals with favorable and stable life style habits. Even if this is the case, this analysis identifies a substantial subgroup of light–moderate drinkers with no apparent increased risk of cancer.

While all evidence is inferential, the simplest explanation of our data is that, in aggregate, *Likely* status among light–moderate drinkers is associated with higher prevalence of underreporting and that *Unlikely* status is less often so associated. The apparently spurious nature of increased cancer risk in consistent light–moderate drinkers with should be reassuring to these established light–moderate drinkers. In any case, advice to patients by health professionals is best individualized, keeping in mind the hazards and benefits of alcohol drinking [16].

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