Alcohol-Attributable Cancer Deaths and Years of Potential Life Lost in the United States

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Objectives. Our goal was to provide current estimates of alcohol-attributable cancer mortality and years of potential life lost (YPLL) in the United States.

Methods. We used 2 methods to calculate population-attributable fractions. We based relative risks on meta-analyses published since 2000, and adult alcohol consumption on data from the 2009 Alcohol Epidemiologic Data System, 2009 Behavioral Risk Factor Surveillance System, and 2009–2010 National Alcohol Survey.

Results. Alcohol consumption resulted in an estimated 18 200 to 21 300 cancer deaths, or 3.2% to 3.7% of all US cancer deaths. The majority of alcohol-attributable female cancer deaths were from breast cancer (56% to 66%), whereas upper airway and esophageal cancer deaths were more common among men (53% to 71%). Alcohol-attributable cancers resulted in 17.0 to 19.1 YPLL for each death. Daily consumption of up to 20 grams of alcohol (≤ 1.5 drinks) accounted for 26% to 35% of alcohol-attributable cancer deaths.

Conclusions. Alcohol remains a major contributor to cancer mortality and YPLL. Higher consumption increases risk but there is no safe threshold for alcohol and cancer risk. Reducing alcohol consumption is an important and underemphasized cancer prevention strategy. (*Am J Public Health.* 2013;103: 641–648. doi:10.2105/AJPH.2012.301199)

Alcohol use is estimated to account for about 4% of all deaths worldwide.[1] Research over several decades has consistently shown that alcohol increases the risk for cancers of the oral cavity and pharynx, larynx, esophagus, and liver.[2–5] The biological mechanisms by which alcohol induces cancer are not fully understood, but may include genotoxic effects of acetaldehyde, production of reactive oxygen or nitrogen species, changes in folate metabolism, increased estrogen concentration, or serving as a solvent for tobacco metabolites.[5]

The International Agency for Research on Cancer (IARC) and the World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR) both published comprehensive reviews of the scientific literature on alcohol and cancer risk in 2007.[5–7] In addition to confirming earlier research for the previously mentioned cancers, they concluded that alcohol increases the risk for colon, rectal, and female breast cancer. (The WCRF/AICR used the term "convincing increased risk" upon judging the strength of the evidence for alcohol use with all of these cancers, with the exception of "probable increased risk" for liver cancer [both sexes] and for colorectal cancer among women.[7])

More recent studies have also found a positive association for colorectal[8–13] and breast cancer[11–14] with alcohol use. Although some researchers report a positive association between alcohol and cancers of the stomach, ovary, prostate, pancreas, bladder, or endometrium,[14–21] this has not been found by others.[11,22–29]

There have been surprisingly few efforts to ascertain the number of cancer deaths or years of potential life lost (YPLL) attributable to alcohol in the United States. To our knowledge, Rothman et al. were the first to consider this issue,[3] estimating that alcohol caused 3% of US cancer deaths in 1974. Doll and Peto, in their seminal work on avoidable causes of cancer, came up with the same estimate of 3% (range = 2%–4%) for US cancer deaths in 1978.[4] The Harvard Center for Cancer Prevention in 1996 reported that 3% of US cancers resulted from alcohol use but included no analyses in their report.[30] Boffetta et al. estimated that for the World Health Organization region consisting of the United States, Canada, and Cuba, alcohol was responsible for 3% of cancer deaths in men and 2% in women in 2002.[31]

Extensive research has been published over the past 30 years on alcohol use and cancer risk, including more recent studies showing an increased risk of breast and colorectal cancer. In addition, new methods have been developed to better estimate population-level alcohol consumption by demographics based on both survey and sales-based data. Therefore, a comprehensive examination of the population-wide impact in the United States was long overdue. The purpose of our study was to provide current estimates of deaths and YPLL from cancer attributable to alcohol use in the United States. We did so by using sensitivity analyses based on 2 different methodologies and 2 separate nationwide surveys.

METHODS

Unlike health conditions 100% related to alcohol use (e.g., alcohol psychosis), only a portion of deaths from specific types of cancer can be attributed to alcohol. Thus, we used population-attributable fractions (PAFs) to estimate the percentage and number of alcohol-related cancer deaths and YPLL. We used the following formula[32,33] to estimate the proportion of deaths and YPLL attributable to alcohol use for each cancer type:

$$(1)\quad \mathrm{PAF} = \frac{\sum_{i=1}^{n} P_i\,(RR_i - 1)}{1 + \sum_{i=1}^{n} P_i\,(RR_i - 1)}$$

where P_i is the prevalence of alcohol consumption and RR_i is the relative risk for the type of cancer at the ith consumption level.

We limited RR estimates, and subsequent mortality data and YPLL calculations, to the 7 cancers (oral cavity and pharynx, larynx, esophagus, liver, colon, rectum, and female breast) considered causally associated with alcohol use by both the IARC and WCRF/AICR.[5,7]

Data Sources

We obtained US mortality data from 2009 based on *International Classification of Diseases, Tenth Revision* codes[34] for each of the 7 cancers from the Vital Statistics System.[35] We based YPLL on life table analysis and estimated years of remaining life expectancy and also obtained this information from the Vital Statistics System.[35,36]

We obtained RRs for alcohol use and cancer risk from meta-analyses published since 2000 and that used multivariable methods to control for potential confounding factors. To help better define risk level by alcohol exposure, we only considered meta-analyses with RRs based on 3 or more levels of alcohol exposure and, whenever possible, utilized nonlinear modeling to better quantify dose–response relationships. Several studies met these requirements and we included these in our analyses.[37–41]

We obtained sales-based data on per-capita alcohol consumption of alcohol in 2009 in the United States from the Alcohol Epidemiologic Surveillance System.[42] Sales-based data, however, cannot measure differences in patterns of alcohol use by age, sex, or other demographics, which makes it essential to use nationally representative survey data. Because of substantial underreporting, survey estimates account for only 25% to 50% of alcohol consumption compared with alcohol sales data.[43,44] Fortunately, there have been recent major methodologic advances that use both alcohol sales and survey data to provide more valid population-based estimates of alcohol use prevalence and consumption, and we used these methods for this study.[45,46]

As an aspect of our sensitivity analyses, we obtained data from 2 nationwide surveys of adults, one that collects data on several health risk factors and one that collects detailed information only on alcohol. The Behavioral Risk Factor Surveillance System (BRFSS) is an annual, random-digit-dial state-based telephone survey of adults aged 18 years or older that contains questions for many health risk factors; details are available elsewhere.[47] Data in 2009 from all states and the District of Columbia were pooled to produce nationwide estimates and weighted to be representative of the US adult population. The sample size was 432 145 and the response rate was 52% based on American Association for Public Opinion Research Response Rate Formula 4.[48]

Only a limited number of alcohol questions are included on the BRFSS. We used findings from quantity and frequency questions to calculate average number of drinks consumed per day among current alcohol users, and converted them to average grams of pure alcohol per day, assuming each drink contained 14 grams of alcohol.[49] We stratified estimates by age and sex.

The 2009–2010 National Alcohol Survey (NAS) was a nationally representative survey of adults aged 18 years or older conducted by using a random-digit-dial telephone survey; details are provided elsewhere.[50,51] There was a total of 7969 respondents and the American Association for Public Opinion Research Response Rate Formula 4 value was 52%.

The NAS contains detailed questions that focus predominantly on alcohol. Average alcohol consumption was based on responses to a set of beverage-specific graduated quantity and frequency questions.[52,53] As with the BRFSS, we used NAS data from quantity and frequency questions to calculate average number of drinks per day and average grams of alcohol per day, assuming each drink contained 14 grams of alcohol. We stratified estimates by age and sex.

Method I for Calculating Population-Attributable Fractions

Method I was developed by Rehm et al. as part of the Comparative Risk Assessment for alcohol within the recently published Global Burden of Disease Study.[45,54–56] It has been widely used to calculate alcohol-attributable PAFs in several countries,[57,58] and regionally and globally by the World Health Organization and Global Burden of Disease Study.[56,59–61]

Briefly, this method triangulates estimates of alcohol consumption from surveys and per capita consumption taking into consideration factors such as recorded sales and unrecorded consumption, abstinence, and average volume of consumption among drinkers by sex and age from surveys.[45,54,60,62] Detailed descriptions of the methodology and calculations used for method I are available.[45,54,55,62] We conducted analyses separately by sex and across 5 age groups. We derived 95% confidence intervals (CIs) for PAFs by using Monte Carlo simulation.[62,63]

Method II for Calculating Population-Attributable Fractions

As another aspect of our sensitivity analyses, we used a second method to calculate PAFs. Details about this method, developed by Rey et al. and used to estimate alcohol-attributable mortality in France, are available elsewhere.[46]

Although development of method II was influenced by the work of Rehm et al., it differs somewhat because it adjusts for survey consumption underreporting of alcohol by using comparable ratios based on average per capita sales–based alcohol consumption data by sex across 6 age groups. We calculated sex- and age-specific prevalence estimates based on 6 average daily alcohol consumption levels (none, >0 to 20, >20 to 40, >40 to 60, >60 to 80, and >80 g/day), with midpoint consumption estimates (in grams) used to determine RRs. We used the delta method to estimate variance and 95% CIs for PAFs.[46,64]

Statistical Analyses

We conducted data analyses by using SAS version 9.2 (SAS Institute, Cary, NC), SAS-callable SUDAAN version 10 (Research Triangle Institute, Research Triangle Park, NC), R version 2.15, Stata version 12 (StataCorp LP, College Station, TX), and Excel 2007 (Microsoft Corporation, Redmond, WA). We weighted survey data to be nationally representative. We estimated PAFs and 95% CIs separately by method and survey. We multiplied PAFs and 95% CIs from methods I and II by deaths and YPLL for each type of cancer by age and sex group and summed them. We divided alcohol-attributable YPLL by deaths to estimate average YPLL per death. For method II only, additional analyses were feasible and conducted to estimate alcohol-attributable cancer deaths by average daily

alcohol consumption (classified as >0 to 20, >20 to 40, or >40 g/day) using the approach described by Jones et al.[65]

We based PAFs on current alcohol survey and sales-based consumption estimates, but cancer has a latency period of many years. To try to account for this latency, we conducted additional PAF analyses by using both methods based on 1992 alcohol sales data,[42] the 1992 BRFSS,[47] and the 1990 and 1995 NAS (conducted in person in both years).[50]

RESULTS

Population-attributable fractions for alcohol consumption and cancer varied somewhat by method, survey, cancer, and sex (Table 1). Nevertheless, they were much higher for cancers of the oral cavity and pharynx, larynx, and esophagus, especially for men, with method II. Estimated PAF values based on methods I and II were generally similar for esophageal, liver, and breast cancer; however, method I resulted in higher PAF values for colon and rectal cancer. Analyses based on survey and sales data from the early 1990s resulted in PAFs that were similar to those based on 2009 data (data not shown in tables).

There was variability by method and survey for the number of alcohol-attributable cancer deaths from specific cancers; differences between men and women by method were more pronounced than survey-related differences, however (Table 2). Nevertheless, overall estimates of deaths were close, ranging from 18 178 to 21 284 (average across all 4 estimates = 19 503). This amounted to 3.2% to 3.7% of all cancer deaths in 2009 (range among men = 2.4%–4.0%; range among women = 2.7%–4.8%). Cancers of the oral cavity and pharynx, larynx, and esophagus accounted for 3790 to 8395 of alcohol-attributable deaths among men (53%–71%); breast cancer accounted for 4730 to 7310 of alcohol-attributable deaths among women (56%–66%).

Alcohol-attributable cancers accounted for a total of 310 751 to 374 250 YPLL (Table 3). This amounted to an average of 17.0 to 19.1 years per death, with slight variations based on method and survey.

Using data available only from method II (Table 4), an estimated 48% to 60% of alcohol-attributable cancer deaths occurred at average daily consumption levels of more than 40 grams of alcohol (i.e., ≥3 drinks per day). Consumption of from more than 20 grams to 40 grams (approximately 1.5 to <3 drinks per day) was responsible for 14% to 17% of deaths, whereas consumption of 20 grams or less (≤1.5 drinks per day) accounted for 25% to 35% of alcohol-attributable cancer deaths (range among men = 17%–25%; range among women = 37%–51%; data stratified by sex not shown in tables). Although cancer risk increased with higher alcohol consumption levels, the greater percentage of total alcohol-related cancer deaths occurring among those consuming 20 grams or less per day, compared with those consuming more than 20 grams and up to 40 grams per day, stems from the larger number of persons who consumed alcohol at lower levels.

TABLE 1—Population-Attributable Fractions for Alcohol-Attributable Cancers: United States, 2009

Cancer Type	BRFSS		NAS	
	Men, % (95% CI)	Women, % (95% CI)	Men, % (95% CI)	Women, % (95% CI)
	Method 1 PAF[45,54–56]			
Oral cavity and pharynx	30 (29, 32)	28 (27, 30)	27 (25, 29)	26 (24, 28)
Larynx	20 (19, 22)	22 (20, 23)	17 (16, 19)	19 (18, 21)
Esophagus	19 (18, 20)	21 (19, 22)	17 (15, 18)	18 (17, 20)
Colon	8 (7, 9)	14 (12, 15)	7 (6, 8)	12 (10, 13)
Rectum	10 (9, 11)	15 (13, 16)	8 (7, 9)	12 (11, 14)
Liver	13 (11, 14)	16 (15, 18)	11 (10, 12)	14 (13, 16)
Female breast	NA	18 (16, 20)	NA	16 (14, 17)
	Method 2 PAF[46]			
Oral cavity and pharynx	66 (63, 69)	37 (34, 41)	64 (60, 68)	38 (36, 40)
Larynx	38 (36, 41)	18 (16, 20)	32 (29, 35)	11 (9, 13)
Esophagus	34 (32, 36)	20 (18, 23)	30 (29, 33)	16 (14, 18)
Colon	5 (4, 6)	3 (2, 4)	4 (3, 5)	2 (1, 3)
Rectum	9 (8, 10)	5 (4, 6)	8 (7, 9)	4 (3, 5)
Liver	16 (15, 17)	9 (8, 10)	15 (14, 16)	8 (6, 10)
Female breast	NA	14 (12, 16)	NA	12 (10, 14)

Note. BRFSS = Behavioral Risk Factor Surveillance System; CI = confidence interval; NA = not applicable; NAS = National Alcohol Survey; PAF = population-attributable fraction.

DISCUSSION

To our knowledge, this is the first comprehensive study of alcohol-attributable cancer deaths in the United States in more than 30 years. We used updated RR estimates for alcohol use, and the 7 types of cancer for which there is scientific consensus about increased risk, in our study.[5–7] We used 2 rigorous methods that adjusted survey-based estimates to sales-based alcohol consumption data, along with 2 different nationwide surveys, to assess the robustness of our results.

Overall, we found that alcohol use accounted for approximately 3.5% of all cancer deaths, or about 19 500 persons, in 2009. It was a prominent cause of premature loss of life, with each alcohol-attributable cancer death resulting in about 18 years of potential life lost. Although cancer risks were greater and alcohol-attributable cancer deaths more common among persons who consumed an average of more than 40 grams of alcohol per day (≥3 drinks), approximately 30% of alcohol-attributable cancer deaths occurred among persons who consumed 20 grams or less of alcohol per day. About 15% of breast cancer deaths among women in the United States were attributable to alcohol consumption.

Of note was that the 4 sets of analyses produced similar overall alcohol-attributable cancer mortality findings, providing a good

TABLE 2—Estimated Alcohol-Attributable Cancer Deaths: United States, 2009

Cancer Type	BRFSS			NAS		
	Men, No. (95% CI)	Women, No. (95% CI)	Overall, No. (95% CI)	Men, No. (95% CI)	Women, No. (95% CI)	Overall, No. (95% CI)
			Method I[45,54–56]			
Oral cavity and pharynx	1656 (1567, 1745)	691 (649, 733)	2347 (2215, 2478)	1469 (1362, 1576)	640 (593, 687)	2109 (1955, 2264)
Larynx	580 (542, 618)	165 (152, 178)	745 (694, 795)	499 (463, 536)	147 (136, 159)	647 (599, 694)
Esophagus	2121 (1991, 2251)	595 (548, 642)	2716 (2540, 2893)	1822 (1696, 1947)	532 (489, 575)	2353 (2185, 2522)
Colon	1805 (1583, 2016)	2885 (2559, 3210)	4689 (4151, 5227)	1451 (1287, 1614)	2441 (2173, 2709)	3892 (3460, 4324)
Rectum	367 (330, 404)	411 (367, 455)	778 (698, 859)	303 (274, 332)	350 (313, 386)	652 (588, 717)
Liver	1662 (1490, 1830)	1037 (933, 1141)	2699 (2426, 2971)	1426 (1281, 1571)	906 (815, 998)	2332 (2096, 2568)
Female breast	NA	7310 (6644, 7976)	7310 (6644, 7976)	NA	6357 (5803, 6911)	6357 (5803, 6911)
Total	8191 (7517, 8864)	13 094 (11 852, 14 335)	21 284 (19 369, 23 200)	6970 (6364, 7576)	11 373 (10 322, 12 425)	18 343 (16 686, 20 000)
US cancer deaths, %	2.8 (2.5, 3.0)	4.8 (4.4, 5.3)	3.7 (3.4, 4.1)	2.4 (2.2, 2.6)	4.2 (3.8, 4.6)	3.2 (2.9, 3.5)
			Method II[46]			
Oral cavity and pharynx	3582 (3406, 3757)	915 (825, 1005)	4497 (4232, 4762)	3502 (3257, 3699)	936 (876, 979)	4438 (4133, 4677)
Larynx	1098 (1021, 1176)	135 (119, 150)	1233 (1141, 1326)	920 (837, 1000)	88 (70, 98)	1008 (907, 1098)
Esophagus	3715 (3480, 3951)	582 (505, 658)	4297 (3985, 4610)	3372 (3206, 3638)	475 (400, 522)	3847 (3607, 4160)
Colon	991 (874, 1107)	531 (397, 664)	1521 (1271, 1771)	876 (568, 1138)	426 (125, 710)	1302 (694, 1849)
Rectum	330 (297, 362)	136 (106, 167)	466 (403, 529)	298 (251, 353)	126 (72, 153)	424 (323, 506)
Liver	2015 (1961, 2249)	569 (480, 658)	2674 (2440, 2908)	1944 (1778, 2135)	485 (410, 598)	2430 (2188, 2733)
Female breast	NA	5518 (4693, 6343)	5518 (4693, 6343)	NA	4730 (4181, 5591)	4730 (4181, 5591)
Total	11 820 (11 039, 12 602)	8386 (7125, 9646)	20 206 (18 164, 22 248)	10 912 (8971, 11 963)	7266 (6134, 8651)	18 178 (16 032, 20 613)
US cancer deaths, %	4.0 (3.7, 4.2)	3.1 (2.6, 3.6)	3.6 (3.2, 3.9)	3.7 (3.3, 4.0)	2.7 (2.3, 3.2)	3.2 (2.8, 3.6)

Note. BRFSS = Behavioral Risk Factor Surveillance System; CI = confidence interval; NA = not applicable; NAS = National Alcohol Survey.

indication of the robustness of these results. However, there was some variability by type of cancer and sex between methods. Method I resulted in higher mortality estimates for women than men, and lower total and average YPLL compared with method II. These differences probably result from the different assumptions and approaches used in each method. By contrast, survey-associated differences in mortality estimates were relatively small.

The estimate that 3.5% of all cancer deaths are alcohol-related was slightly higher than previous estimates of 3% for the United States.[3,4,30] This probably stems from including additional cancers, especially female breast cancer. Nevertheless, our findings demonstrate there has been little, if any, progress in reducing alcohol-attributable cancer deaths in the United States. Other research has shown that the percentage of cancer deaths or cancer incidence attributable to alcohol is substantially higher in several European countries with higher per capita alcohol consumption than the United States, especially for men.[31,66–68] For example, a recent prospective study with a sample size greater than 350 000 in 8 European countries found that alcohol accounted for 10% of total cancer incidence in men and 3% in women.[67]

Several studies report PAFs for alcohol-attributable cancers by type of cancer, and our PAFs were higher in some instances and lower in others.[31,46,65,66,68] This was not surprising when one considers the broad variability in data sources, study designs, and average per capita alcohol consumption levels among studies and countries. However, our findings confirm previous research that oral cavity and pharyngeal, laryngeal, and esophageal cancers account for the majority of alcohol-related cancer deaths among men, and breast cancer for the majority of such deaths among women.[31,46,65,67–69]

Some researchers have estimated RRs for cancer associated with low or moderate alcohol use,[8,11,14,70,71] but to our knowledge, only 1 mentions specific cancer types with detailed information on average alcohol exposure data.[67] The recent study of 8 European countries by Schutze et al. found an estimated 14% of alcohol-attributable incident cancer cases occurred among men who drank alcohol and consumed 24 grams or less (≤ 2 drinks) of alcohol per day, which was lower than our estimate of 16% to 25% for men consuming 20 grams or less (≤ 1.5 drinks) per day among alcohol drinkers.[67] Similarly, Schutze et al.'s estimate that 20% of alcohol-related cancer cases occurred among women consuming 12 grams or less (≤ 1 drink) per day was lower than our estimate of 31% to 51% for female cancer deaths at 20 grams or less (≤ 1.5 drinks) per day.

The difference between the 2 studies is probably because of relatively lower alcohol consumption levels in the United States compared with most European countries,[72] resulting in a proportionally greater percentage of US alcohol-attributable cancer deaths occurring at lower consumption levels. Thus, our findings add to the growing research evidence showing that, in addition to risks at high consumption levels, regular alcohol use at low consumption levels is also associated with increased cancer risk.[71,73] In sum,

TABLE 3—Total and Average Alcohol-Attributable Years of Potential Life Lost: United States, 2009

Cancer Type	BRFSS		NAS	
	Total YPLL	Average YPLL	Total YPLL	Average YPLL
	Method I[45,54–56]			
Oral cavity and pharynx	41 143	17.5	37 031	17.6
Larynx	12 389	16.6	10 775	16.7
Esophagus	44 464	16.5	38 571	16.4
Colon	67 537	14.4	54 798	14.1
Rectum	12 494	16.0	10 324	15.9
Liver	44 798	16.6	38 652	16.6
Female breast	141 770	19.3	120 599	19.1
Total	364 003	17.1	310 751	17.0
	Method II[46]			
Oral cavity and pharynx	80 650	17.9	81 878	18.4
Larynx	21 062	17.1	18 212	18.1
Esophagus	72 698	16.9	66 969	17.4
Colon	24 554	16.1	22 065	17.0
Rectum	8403	18.0	7845	18.5
Liver	49 209	18.4	45 911	18.9
Female breast	116 748	21.3	104 586	22.1
Total	374 250	18.5	347 467	19.1

Note. BRFSS = Behavioral Risk Factor Surveillance System; NAS = National Alcohol Survey; YPLL = years of potential life lost.

there is no apparent threshold when it comes to alcohol and cancer risk.[5,6] For cancer prevention purposes, this means it is better to drink alcohol at low levels, with the lowest risk involving not drinking at all.

Limitations and Strengths

Our study had limitations. We based RRs on meta-analyses but all potential confounders were not necessarily controlled within each study included in meta-analyses. We did not examine cancer risk by alcohol-specific beverage because research on beverage-specific risks for cancer is limited and inconsistent[74–76] and beverage-specific data are not collected in the BRFSS. Survey response rates have been declining in US surveys for many years, especially for telephone-based surveys.[77–79] But the potential impact, if any, was likely to be small because PAFs were similar based on surveys with higher response rates conducted in the early to mid-1990s, and response rate is not necessarily a measure of survey validity.[79–82] We based PAFs on current prevalence estimates, not lifetime alcohol exposure; therefore, our results may be conservative because risk of cancer increases,[83] and prevalence and consumption levels of alcohol use decrease,[49] with age in the United States. Further evidence that estimates may be conservative is that survey nonrespondents and persons without landline telephones are more likely to use alcohol and to be heavier alcohol consumers.[84–89] The impact of nonresponse and noncoverage bias is probably limited, however, because the heaviest alcohol consumers missed in surveys likely represent a relatively small proportion of the total population.[90] Conversely, attributable risk calculations cannot definitely prove that all observed associations are 100% causal in nature.

There were also study strengths. We utilized methods taking into account sales and survey estimates of alcohol consumption and that have been used in other countries. Use of sensitivity analyses provides greater confidence in the validity of the results. The PAF calculations based on data from the early to mid-1990s were similar to those found with 2009 data. This suggests that temporal changes in alcohol consumption had little effect on our estimates and is consistent with

TABLE 4—Percentage of Cancer-Specific Alcohol-Attributable Cancer Deaths in the United States, by Average Daily Alcohol Consumption and Survey: 2009

Cancer Type	BRFSS, %			NAS, %		
	>0 to 20 Grams/Day	>20 to 40 Grams/Day	>40 Grams/Day	>0 to 20 Grams/Day	>20 to 40 Grams/Day	>40 Grams/Day
Oral cavity and pharynx	6.2	4.7	89.1	24.5	5.2	70.2
Larynx	5.8	7.8	86.4	7.0	11.8	81.2
Esophagus	25.2	16.3	58.6	32.1	21.5	46.4
Colon	33.1	17.3	49.6	41.5	21.6	36.9
Rectum	32.1	17.5	50.4	41.6	20.8	37.6
Liver	30.3	17.4	52.3	38.9	21.1	40.0
Female breast	32.9	17.7	49.4	49.7	22.7	27.6
Total	25.5	14.4	60.2	35.2	17.2	47.5

Note. BRFSS = Behavioral Risk Factor Surveillance System; NAS = National Alcohol Survey. Based on method II, Rey et al.[46]

findings that survey-based alcohol consumption estimates and per capita alcohol sales–based estimates have not changed appreciably over the past 2 decades.[42,47,49,91]

Implications and Conclusions

Our estimate of 19 500 alcohol-related cancer deaths is greater than the total number of deaths from some types of cancer that receive much more prominent attention, such as melanoma or ovarian cancer,[36] and it amounted to more than two thirds of all prostate cancer deaths in 2009.[36] Reducing alcohol consumption is an important and underemphasized cancer prevention strategy, yet receives surprisingly little attention among public health, medical, cancer, advocacy, and other organizations in the United States, especially when compared with efforts related to other cancer prevention topics such as screening, genetics, tobacco, and obesity.

New recommendations by the US Preventive Services Task Force on breast cancer screening among women aged 40 to 49 years in 2009,[92] for example, generated enormous attention and stimulated efforts by various individuals and organizations. Yet the task force's finding that 1904 women need to be invited for screening to save 1 life, and excluding the potential harms associated with screening for this population, would amount to saving at most 6600 lives among all women in this age group over the course of their lifetime.[92,93] Our finding that many breast cancer deaths are attributable to alcohol consumption strongly suggests that greater emphasis on the role of alcohol as an important risk factor for breast cancer is needed.

Several major health organizations in the United States and other countries that issue policy statements or recommendations have done so for alcohol, or for cancer prevention, but not for cancer risks associated with alcohol use.[94–97] The American Cancer Society[98] and AICR[99] have noted that alcohol increases the risk for several types of cancer and that its use is not recommended or should be limited. However, both organizations add a caveat about low or moderate alcohol use and decreased risk of heart disease. The 2010 National Dietary Guidelines for Americans mention that moderate alcohol use is associated with an increased risk of breast cancer, and excessive drinking increases upper gastrointestinal tract and colon cancer risk, but state that there is strong evidence for health benefits from moderate use.[100]

One likely reason more efforts have not been mustered to reduce alcohol use for cancer prevention is reluctance based upon the purported cardiovascular benefits of low-level alcohol consumption.[101] Such reluctance may not be justified, particularly in an era of declining cardiovascular mortality.[83] Studies on the effects of moderate drinking have serious limitations related to confounding, selection bias, and other factors.[102–108] When viewed in the broad context, alcohol results in 10 times as many deaths worldwide even after one considers possible beneficial effects of low-level use for cardiovascular disease and diabetes.[1] For most alcohol users, therefore, reducing alcohol consumption would likely improve their health in many ways in addition to reducing cancer risk.

Stronger and more comprehensive individual- and population-level efforts are warranted to reduce cancer risk from alcohol use. Clear and consistent statements from medical and public health organizations and providers are needed emphasizing that (1) alcohol is a known human carcinogen, (2) cancer risk increases considerably at high consumption levels but there is no safe level at which there is no cancer risk, and (3) alcohol use should be lowered or avoided to reduce cancer risk. Proven population-based strategies are also needed, which include reducing alcohol accessibility, increasing prices by raising alcohol taxes, restricting alcohol marketing and advertising, and increasing treatment opportunities for heavier alcohol users.[109–112] ■

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Contributors

D. E. Nelson directed the study. D. W. Jarman assisted with the study design and conducted data analyses. J. Rehm, T. K. Greenfield, and G. Rey assisted with study design, analysis, and interpretation. W. C. Kerr assisted with data analysis and interpretation. P. Miller, K. D. Shield, and Y. Ye conducted data analyses. T. S. Naimi helped originate, design, and direct the study.

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Human Participant Protection

No protocol approval was needed for this study because we used only de-identified data.

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