Mortality in England and Wales attributable to current alcohol consumption

A Britton and K McPherson

J. Epidemiol. Community Health 2001;55;383-388
doi:10.1136/jech.55.6.383

Mortality in England and Wales attributable to current alcohol consumption

A Britton, K McPherson

Abstract

***Study objective*—To estimate the number of deaths attributable to current alcohol consumption levels in England and Wales by age and sex.**

***Design*—Epidemiological approach using published relative risks and population data.**

***Setting*—England and Wales.**

***Main outcome measures*—Numbers of deaths by age and sex and years of life lost for alcohol related conditions.**

***Results*—Because of the cardioprotective properties of alcohol, it is estimated that there are approximately 2% fewer deaths annually in England and Wales than would be expected in a non-drinking population. This proportion varies greatly by age and sex and only among men aged over 55 years and women aged over 65 years is there likely to be found a net favourable mortality balance. It is also estimated that there were approximately 75 000 premature years of life lost in England and Wales in 1996 attributable to alcohol consumption. The main causes of alcohol attributable mortality among the young include road traffic fatalities, suicide and alcoholic liver disease.**

***Conclusions*—At a population level, current alcohol consumption in England and Wales may marginally reduce mortality. However, the benefit is disproportionately found among the elderly. Estimating alcohol attributable mortality by age and sex may be a useful indicator for developing alcohol strategies. More research into the possible effect modifications of pattern of consumption, beverage type, age and gender will enable these estimates to be improved.**

(*J Epidemiol Community Health* 2001;**55**:383–388)

Health Promotion Research Unit, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT, UK
A Britton

Cancer and Public Health Unit, London School of Hygiene and Tropical Medicine
K McPherson

Correspondence to:
Annie Britton
(annie.britton@lshtm.ac.uk)

Accepted for publication
1 February 2001

From the public health perspective, alcohol provides many challenges. Unlike most other mood altering substances, it is legal, socially acceptable and extremely widely used, thus making any control policies complicated. Moreover, it is widely recognised that alcohol can bring significant benefits as well as harm to individuals and societies. It can not only be a life enhancing convivial habit, but when consumed in moderation, it has a protective effect against coronary heart disease[1] and possibly ischaemic stroke.[2 3] Its benefits may, however, be outweighed by an increased risk of other diseases (including cirrhosis of the liver, pancreatitis, upper aerodigestive cancers, and alcohol psychoses) and an increased risk of violence and accidents.[4 5]

For certain sections of society the balance of risks and benefits, measured here in terms of deaths attributable to alcohol consumption, may result in a net number of deaths "prevented" or "delayed". This is most likely to be found among those with an underlying higher risk of heart disease, such as middle aged and elderly men. Younger men, on the other hand, may experience a net aggregate excess of alcohol attributable deaths largely because of their higher rates of weekly alcohol consumption and also their higher rates of accidents and violence compared with older men. For women the balance is complicated by the addition of a possible association between alcohol consumption and the incidence of breast cancer.[6] If at all, net positive balances will be found among postmenopausal women when their background coronary heart disease risks are increased.

Such calculations on aggregated deaths are useful to inform sensible drinking guidelines for different sections of society. Clearly, the estimation of alcohol attributable deaths is only part of the picture as the toll of alcohol related harm includes additional damage to the physical and mental health of the drinker, as well as to their families, friends and others in society through accidents, crimes, violence and impaired working capacity. On the other hand quality of life improvement resulting from moderate drinking may be a very important social benefit.[7]

Methods

Mortality data in England and Wales for 1996 were obtained from the Office for National Statistics.[8] The numbers of deaths for 26 relevant conditions, using the International Classification of Diseases version 9 (ICD-9), were extracted by age and sex. The relative risks used in this analysis, shown in tables 1 and 2, were mainly derived from a large meta-analysis (123 studies) of available international epidemiological evidence by Corrao *et al*.[9] Studies were assessed for quality (study design, alcohol consumption measurement, data analysis methods and adjustment for confounders) and separate risk functions were given by gender, area (Mediterranean versus non-Mediterranean), study design (case-control versus cohort studies) and outcome (incidence versus deaths) where appropriate. The relation between alcohol and ischaemic heart disease (IHD) was not reported in this meta-analysis as the authors were only concerned with harmful consequences. However, a subsequent meta-analysis by the same authors was published the following year.[10]

Alcohol consumption data were collected in the 1998 Health Survey for England.[11] This

Table 1 The alcohol risk functions based on meta-analyses by Corrao et al and English et al

Condition	*Effects of alcohol*
Rectal cancer	Log RR = 0.0042.alc* (+0.0282.alc if female†)
Oropharyngeal cancer	Log RR = 0.0348.alc—0.0001.alc^2 (+0.0015.alc if female −0.0067.alc if non-Med. country‡)
Oesophageal cancer	Log RR = 0.0186.alc—0.0000001.alc^3 (−0.0028.alc if non-Med. country)
Liver cancer	Log RR = 0.0074.alc—0.00001.alc^2
Laryngeal cancer	Log RR = 0.0197.alc—0.00000002.alc^3 (−0.0120.alc if non-Med. country)
Breast cancer	Log RR = 0.0123.alc (−0.0046.alc if non-Med. country)
Colon cancer	Log RR = 0.0014.alc (+0.0116.alc if cohort studies§)
IHD	Log RR = 0.0011.alc—1.0650√alc (+0.0062.alc if female)
Haemorrhagic stroke	Log RR = 0.0150.alc
Essential hypertension	Log RR = 0.0142.alc
Chronic pancreatitis	Log RR = 0.0116.alc
Alcholic liver disease	100% deaths attributable to alcohol consumption
Non-dependent abuse of alcohol	100% deaths attributable to alcohol consumption
Alcohol dependence syndrome	100% deaths attributable to alcohol consumption
Alcoholic psychosis	100% deaths attributable to alcohol consumption
Alcoholic cardiomyopathy	100% deaths attributable to alcohol consumption
Alcoholic gastritis	100% deaths attributable to alcohol consumption
Assaults	47% deaths attributable to alcohol consumption
Accidental drowning	31–50% deaths attributable to alcohol consumption (varies by age and sex**)
Accidental falls	31–50% deaths attributable to alcohol consumption (varies by age and sex)
Accidents caused by fire and flames	44% deaths attributable to alcohol consumption
Inhalation and ingestion	5% deaths attributable to alcohol consumption††
Occupational and machine injuries	0–9% deaths attributable to alcohol consumption (varies by age and sex)
Accidental poisoning by alcohol	100% deaths attributable to alcohol consumption
Motor vehicle accidents	5–40% deaths attributable to alcohol consumption (varies by age and sex)
Suicide	Relative risks of 1.40 for "low consumption", 2.32 for "hazardous consumption" and 2.52 for "harmful consumption"‡‡

*alc = alcohol dose in grams per day. †Corrao *et al* tested for interactions due to gender and where significant effects were found we have used the separate risk functions for men and women. ‡Corrao *et al* tested for interactions due to the area in which the study was performed. Where area effects were found, we used the non-Mediterranean coefficient. §Corrao *et al* tested for study design interactions and when found we have used the cohort design model. **Further details are available from the authors upon request. ††Based on an estimate from the Office for National Statistics, personal communication. ‡‡See paper by English *et al* for details.[13]

survey was based on a sample of approximately 16 000 nationally representative men and women in which they were asked a series of questions designed to provide an estimate of average weekly consumption. Consumption among the Welsh population is very similar to the English population.[12]

The meta-analysis risk functions are given for an exposure measurement in grams of alcohol per day. It was therefore necessary to convert the Health Survey for England data (given in units of alcohol per week) by fitting a log normal distribution assuming one unit to be 8 grams of alcohol. This is shown in table 3.

Table 2 Alcohol consumption levels and relative risk of mortality from key alcohol related conditions

	None	*1–10 g/day**	*10–20 g/day*	*20–30 g/day*	*30–40 g/day*	*40–50 g/day*
IHD men	1	0.832	0.778	0.768	0.775	0.793
IHD women	1	0.857	0.853	0.896	0.962	1.047
Colon cancer	1	1.067	1.215	1.384	1.575	1.794
Breast cancer	1	1.039	1.122	1.211	1.308	1.412
Haemorrhagic stroke	1	1.078	1.252	1.455	1.690	1.964

*Midpoint in each category used to calculate relative risks.

Table 3 Distribution of alcohol consumption by age and sex, England 1998

Gram/day	*16–24*	*25–34*	*35–44*	*45–54*	*55–64*	*65–74*	*75+*
Men							
None	8	7	6	5	8	10	15
1 to 10	25.1	29.3	29.4	29.6	33.9	39.6	48.8
10 to 20	21.5	25.8	25.6	24.4	25.9	25.4	19.6
20 to 30	13.7	14.5	14.5	14.5	13.5	11.7	8
30 to 40	8.8	8.2	8.3	8.6	7.2	5.7	3.7
40 to 50	5.8	5	5.1	5.4	4.1	3	1.9
50 to 60	4	3.1	3.2	3.5	2.4	1.7	1.1
60+	13.1	7.1	7.9	9	5	2.9	1.9
Women							
None	12	9	8	9	13	20	26
1 to 10	48.2	56.1	59.7	58.7	68.9	62.1	60.7
10 to 20	19.5	21	19.3	19.2	10.8	12.1	8.8
20 to 30	8.5	7.4	6.8	6.9	4.4	3.5	2.5
30 to 40	4.3	3.1	2.9	2.9	1.5	1.2	1
40+	7.5	3.4	3.3	3.3	1.4	1.1	1

Data shown as percentages. Source: Adapted from Health Survey for England 1998 (Department of Health). Weekly amounts are averaged over drinking and non-drinking days.

The relative risks were applied to the consumption levels in each sex and age group and an attributable fraction obtained using:

Population attributable fraction (PAF) for exposures with multiple levels

$$\mathrm{PAF} = \frac{\sum_{i=1}^{k} pi(RRi - 1)}{\sum_{i=0}^{k} pi(RRi - 1) + 1}, \qquad (1)$$

where RR_i = relative risk of mortality in exposed groups compared with unexposed group

p_i= proportion of the population exposed in each group

i = 0 to K, where i = 0 represents non-drinkers

The attributable fraction for a particular cause of death is the proportion of all deaths from that cause that can be attributed to current alcohol consumption (assuming the relative risks describe a causal relation)—that is, the proportion by which the mortality would be reduced if everybody became an abstainer from alcohol. Where there is a protective effect of alcohol the attributable fraction will be the increase as a proportion of the total that would result if everybody became an abstainer.

Where the development of a condition necessarily requires the consumption of alcohol (alcoholic liver disease ICD-9 571.0–571.3; alcoholic gastritis ICD-9 535.3; accidental poisoning by alcohol ICD-9 E860.0, E860.1, E860.2; non-dependent abuse of alcohol 305.0; alcohol dependence syndrome 303; alcoholic psychosis 291; alcoholic cardiomyopathy 425.5) all reported deaths were assumed to be attributable to alcohol consumption.

A single risk function was estimated by Corrao *et al* for all injuries and adverse effects combined (ICD-9 800–999). We felt that it would be more informative from a public health perspective if separate component estimates were given for the proportion of alcohol

Table 4 Alcohol attributable deaths by cause (>15 years), England and Wales, 1996

		Deaths attributable to alcohol/all deaths	
	Conditions	*Men*	*Women*
Neoplasms	Rectum cancer	174/2819	358/2136
	Colon cancer	934/5001	401/5491
	Oropharyngeal cancer	356/874	97/462
	Laryngeal cancer	74/635	9/174
	Liver cancer	108/1049	36/766
	Oesophageal cancer	829/3567	233/2268
	Breast cancer	—	667/12179
Circulatory	Haemorrhagic stroke*	598/2848	426/4465
	Essential hypertension	12/64	15/177
	IHD	−14485/70488	−6813/58556
	Alcoholic cardiomyopathy	116/116	24/24
Gastrointestinal	Alcoholic liver disease	1370/1370	689/689
	Chronic pancreatitis	10/46	2/32
	Alcoholic gastritis	3/3	2/2
Injuries and adverse effects	Assaults	75/160	24/50
	Accidental drowning	50/131	16/43
	Accidental falls	486/1439	690/2177
	Accidents caused by fire/flames	101/229	77/176
	Inhalation and ingestion	6/127	6/126
	Occupational injuries	4/63	0/2
	Accidental poisoning by alcohol	13/13	9/9
	Motor vehicle traffic accidents	654/2145	104/803
	Suicide	797/2648	200/794
Other alcohol essential	Non-dependent abuse of alcohol	100/100	44/44
	Alcohol dependence syndrome	172/172	78/78
	Alcoholic psychosis	10/10	4/4
Total		−7 433	−2 602
Deaths from all causes		261 571	287 796
% Alcohol attributable		−2.8%	−0.9%

*The ICD-9 code for stroke is often "unspecified", therefore we have estimated the proportion of deaths that are of haemorrhagic origin using proportions form the Oxford Stroke Study (Bamford J *et al*. A prospective study of acute cerebrovascular disease in the community: the Oxfordshire Community Stroke Project 1981–1986. *J Neurol Neurosurg Psychiatry* 1990;**53**:16–22).

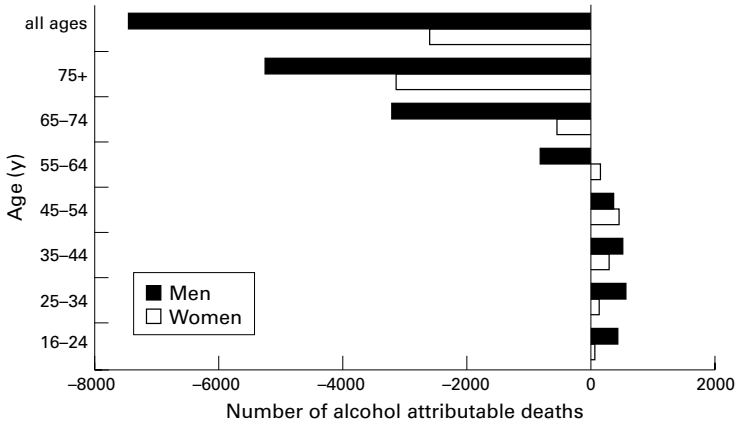

Figure 1 Net alcohol attributable deaths by age for adults in England and Wales 1996.

Table 5 Top three causes of alcohol related death by age in England and Wales, 1996

Age group (y)	*Men (number of alcohol attributable deaths)*	*Women (number of alcohol attributable deaths)*
16–24	Road traffic accidents (249)	Road traffic accidents (30)
	Suicide (99)	Suicide (20)
	Assaults (21)	Assaults (4)
25–34	Suicide (214)	Suicide (42)
	Road traffic accidents (198)	Road traffic accidents (36)
	Alcoholic liver disease (49)	Alcoholic liver disease (18)
35–44	Alcoholic liver disease (233)	Alcoholic liver disease (127)
	IHD (199 prevented)	Breast cancer (49)
	Suicide (166)	Suicide (38)
45–54	IHD (847 prevented)	Alcoholic liver disease (213)
	Alcoholic liver disease (445)	Breast cancer (129)
	Suicide (152)	IHD (96 prevented)
55–64	IHD (2080 prevented)	IHD (389 prevented)
	Alcoholic liver disease (357)	Alcoholic liver disease (161)
	Colon cancer (190)	Breast cancer (114)
65–74	IHD (4609 prevented)	IHD (1343 prevented)
	Colon cancer (314)	Breast cancer (144)
	Oesophageal cancer (286)	Alcoholic liver disease (126)
75+	IHD (6722 prevented)	IHD (4960 prevented)
	Colon cancer (328)	Accidental falls (564)
	Haemorrhagic stroke (283)	Haemorrhagic stroke (302)

attributable injury deaths. Therefore we based our estimates on those from an earlier meta-analysis of international literature by English *et al* [13] for assaults (ICD-9 E960, E965, E966, E968, E969), accidental drowning (E910), accidental falls (E880–888), accidents caused by fire and flames (E890–899), inhalation and ingestion (E911), occupational and machine injuries (E919, 920), motor vehicle traffic accidents (E810–819) and suicide (E950–959).

The attributable fractions were then applied to the mortality data and an estimate was made of the number of alcohol attributed deaths "caused" or "prevented" by age and sex. These analyses adopt the most straightforward estimates of population attributable risk and ignore, in the interests of simplicity, any joint distribution of risk factors between several diseases. Data on these distributions are often sparse and varied. Moreover, as we are dealing with several different diseases that inevitably share some risk factors, for our purposes concentrating on just one risk factor, this simple univariate approach was considered to be an adequate compromise.

Results

The balance in terms of estimated numbers of deaths "prevented" and deaths "caused" by conditions associated with current levels of alcohol consumption, for all adults in England and Wales, is shown in table 4. For men, the net balance shows that, on aggregate, more deaths were "prevented" than were "caused" by alcohol consumption. This is because of the protective effect conferred by alcohol consumption on ishaemic heart disease. The risk function described by Corrao *at al* in their meta-analysis was such that the maximum benefit of about a 20% reduction of CHD risk was found in both men and women, but at lower consumption level for women (about 10 g/day for women and 25 g/day for men—see table 2). Harmful effects of alcohol on CHD risk were reported at about 40 g per day for women and 120 g per day for men, but the majority of the population do not drink at these levels (see table 3).

For both men and women, the net balance shows that there were slightly more deaths "prevented" than "caused". We estimate that there were approximately 2.8% fewer male deaths and 0.9% fewer female deaths in 1996 than would be expected in a non-drinking population.

For women of all ages combined, most alcohol attributable deaths are from accidental falls, alcoholic liver cirrhosis, breast cancer, haemorrhagic stroke and colon cancer. For men most alcohol related deaths are from alcoholic liver cirrhosis, colon cancer, oesophageal cancer, suicide and road traffic accidents.

The net balance of alcohol attributable deaths for each age group and by sex is shown in figure 1. It can be seen that not until age band 55–64 years (men) does the balance start to favour alcohol consumption.

Clearly the leading causes of alcohol associated deaths ("caused" or "prevented") change

with age. The top three contributors for each age band are shown in table 5.

Unsurprisingly, by far the majority of alcohol related deaths occur in the oldest age groups, reflecting the much higher incidences of such conditions. We are less confident of the accuracy of the relative risks in these age groups as the elderly are often excluded from studies. It could also be argued that the deaths "caused" or "prevented" by alcohol consumption at this age are of less interest in terms of population health than premature deaths. Therefore in addition to a quantification of deaths by age, the potential years of life lost or gained can provide a useful measure of the public health impact of alcohol consumption. The deaths at younger ages, mainly from injuries (accidental and intentional), contribute more to the total years of life lost, while the extra years gained from a protection from ischaemic heart disease contribute comparatively less. We estimate that there were 52 412 male and 22 724 female years of life lost up to age 65 (simply subtracting the age at which each death occurred from 65). The majority of these, particularly for the men, were attributable to premature deaths under age 44 years.

KEY POINTS

- In England and Wales alcohol consumption marginally reduces mortality at a population level.
- The favourable mortality balance from alcohol consumption is only found among men aged over 55 years and women aged over 65 years.
- More research is needed into the possible modifications by pattern and type of consumption, age and sex.
- Estimating alcohol attributable mortality by age and sex may be a useful indicator for developing alcohol strategies.

Discussion

We estimated that there were approximately 1.8% fewer deaths in England and Wales than would be expected in a non-drinking population. This estimate is lower than many of those obtained in previous attempts to quantify mortality attributable to alcohol consumption. In some instances this is because of the use of moderate drinking as the reference category and the calculation of deaths attributable to drinking more or drinking less than this amount.[14 15]

More often it is simply because most previous estimates did not include a protective role of alcohol. If the deaths prevented were ignored in this analysis, then approximately 2.0% of all mortality would be attributable to current alcohol consumption levels. This estimate is still lower than the estimate of 4.9% given by Schultz *et al* for the US population[16] or 6.2% estimated by Sutocky *et al* for Californians.[17] The WHO Global Burden of Disease report did incorporate the protective role of alcohol, but the authors still estimated that 1.2% of all deaths in the Established Market Economies are attributable to alcohol use—that is, a net protective estimate was not found.[18] Single *et al*, in their study of the economic costs of drug use in Canada[19] chose to use the relative risks derived by English *et al*'s earlier meta-analysis[13] and estimated that there was a higher number of deaths averted by the use of alcohol than the number of deaths caused by alcohol use. Duffy applied the all cause risk estimates from the British Regional Heart Study[20] to 1987 population consumption data for men aged 45 to 64 years and also estimated that there were fewer deaths than would have occurred in an abstinent population.[21] White *et al* recently applied the risk functions from Corrao *et al's* meta-analyses[9 10] for 16 causes of death to the English and Welsh 1997 population and estimated that 0.8% of all deaths in men were prevented by alcohol consumption and 0.1% of all deaths in women were attributed to alcohol consumption.[22] Therefore there is clearly still considerable controversy surrounding the quantification of alcohol attributable mortality, but the net protective estimate found in this analysis has been reported previously by others. The UK Government's green paper, *Our Healthier Nation*,[23] quotes an estimated 40 000 annual alcohol deaths (derived by the Royal College of General Practitioners, 1986[24]), which we believe to be an inaccurate and incomplete representation of the mortality consequences of alcohol consumption.

The deaths prevented occurred largely among those individuals in the oldest age groups, who had a much higher risk of ischaemic heart disease. The high burden of potential life lost among the young is of particular interest and emphasis should be placed on reducing the mortality in this section of the population, as well as reducing the larger numbers of deaths occurring among the older age groups. From this initial overall study of alcohol attributable mortality in England and Wales, it can be concluded that only for certain subgroups of the population, namely men aged over 55 years and women over 65 years, is there a net favourable mortality outcome from current consumption levels.

In the analyses described here we assume a causal relation between alcohol consumption and the various conditions. In a sensitivity analysis we excluded the large bowel cancers and breast cancer, as there is probably most controversy over the causal link with alcohol.[25 26] This restriction leads to an estimate of 12 569 or 2.3% fewer deaths in 1996 than would be expected in a non-drinking population. In terms of person years of life lost this is 66 400 years—that is, 8735 fewer years of life lost than when breast, colon and rectal cancer were included. The relation between alcohol and ischaemic stroke remains unclear and as Corrao *et al's* meta-analysis found a non-significant relation, we have not included ischaemic stroke in our estimates. Gastric and duodenal ulcers and acute pancreatitis are also not included in our calculations because the

meta-analyses either failed to find any epidemiological evidence, or a non-significant relation with alcohol. The inclusion of these comparatively rare conditions would not change our overall conclusions.

The complete eradication of alcohol consumption will not be a target for any rational government. Therefore, in terms of a sensible drinking policy, it is useful to estimate the most beneficial drinking level in terms of lowest risk of death. This was recently estimated by White *et al* to be zero consumption at ages 16 to 34 years in men and at ages 16 to 54 years in women, rising with age to eight units (72 grams alcohol) per week in men aged over 65 years and three units (27 grams alcohol) per week in women aged over 65 years.[22]

LIMITATIONS AND FURTHER AREAS OF RESEARCH

In this analysis we did not explore the consequences of different drinking patterns and behaviours that exist within a population. There is epidemiological and physiological evidence to suggest that the amount of alcohol consumed in one drinking session has an important impact on the subsequent health consequence.[27] This is most apparent when considering alcohol and its relation with violence and accidents where an element of intoxication is assumed. However, "binge drinking" can have implications for the development of alcohol related diseases. For example, while the cardioprotective effect of alcohol is associated with moderate regular consumption, cardiovascular disease and sudden cardiac death have been shown to be associated with drinking in heavy, sporadic episodes.[28 29]

Another area of interest and some controversy, is the possible effect modification of beverage type. There has been considerable debate on whether the type of alcoholic drink confers different risks, for example, consumption of spirits or beer has been linked with an increased risk of cancer of the upper digestive tract compared with consumption of wine.[30] However, an alternative explanation for the increase is that spirit and beer drinkers themselves have a different health profile or health behaviours to wine drinkers.[31] Unfortunately neither the consumption data nor the relative risks used in this analysis were separated into beverage type.

The majority of the relative risks used in this analysis have not been modified according to age. This is largely because the data on which such modifications would be based are sparse. To assume that the relative risks apply to all ages for each disease equally may be an unreasonable assumption, but one commonly assumed in epidemiology. Where possible the relative risks used in this analysis incorporated gender effects. The gender effects, when included, showed positive coefficients, suggesting that women tend to experience consequences at lower doses than men.

The risk functions were derived from recent meta-analyses of the international literature. Caution arises when applying risk estimates to England and Wales based on other populations,[32] however, using just evidence from the UK would be severely restricting. Corrao *et al* tested for area effect modifications by separating studies into those from Mediterranean and non-Mediterranean countries. Significant effects were found for cancers of the oropharynx, oesophagus, larynx and breast and therefore for these deaths we used the risk function applicable to non-Mediterranean countries.

Where possible Corrao *et al* used studies in their meta-analysis that were adjusted for known confounders, particularly smoking for upper aerodigestive tract cancers, breast cancer and stroke. It is not possible to completely rule out the interaction between alcohol and other risk factors, however we have used the best estimates to our knowledge.

The population data on alcohol consumption are self reported and are therefore at risk of under-estimating or over-estimating actual consumption. It has been suggested that self reported alcohol use in population surveys covers only 40%–60% of alcohol sales,[33] and individuals who are very heavy drinkers are unlikely to participate in the surveys. If the Health Survey for England underestimates the actual consumption, we will have underestimated the alcohol attributable deaths in this analysis. However, the estimated number of deaths from disorders such as alcoholic liver disease, alcoholic gastritis, poisoning by alcohol, etc, are taken from the Office for National Statistics and do not rely upon consumption estimates. On the other hand, it is possible that these official figures are affected by under-reporting, for example by coroners to protect families.

This analysis focuses on current consumption and does not consider previous levels or patterns of drinking. It is probable that an exposure over several years is needed for some conditions to develop and more longitudinal studies are needed in which changes in alcohol consumption are measured. However, this study does identify the sex and age groups for which alcohol has substantial negative consequences in terms of mortality and years of life lost and this information could be used to target prevention strategies.

While the debate on the relation between alcohol consumption and mortality from certain causes is still on the agenda, and particularly in light of recent epidemiological evidence that challenges alcohol's cardioprotective properties,[34] we believe our estimations could help inform policy decisions for the forthcoming English national alcohol strategy.[35]

The authors are grateful for helpful comments from Franco Cavallo, Morten Grønbæk, Ian Macdonald, John Powles, Ron Simpson and Ian White.

Funding: unconditional grant from International Life Sciences Institute (ILSI-Europe).
Conflicts of interest: none.

1 Doll R, Peto R, Hall E, *et al*. Mortality in relation to consumption of alcohol: 13 years' observations on male British doctors. *BMJ* 1994;**309**:911–18.
2 Camargo CA. Moderate alcohol consumption and stroke: the epidemiological evidence. *Stroke* 1989;**20**:1611–26.
3 Stampfer MJ, Colditz GA, Willett WC, *et al*. A prospective study of moderate alcohol consumption and the risk of coronary disease and stroke in women. *N Engl J Med* 1988;**319**:267–73.

4 Thun MJ, Peto R, Lopez AD, *et al*. Alcohol consumption and mortality among middle-aged and elderly U.S. adults. *N Engl J Med* 1997;**337**:1705–14.
5 Fuchs CS, Stampfer MJ, Colditz GA, *et al*. Alcohol consumption and mortality among women. *N Engl J Med* 1995;**332**:1245–50.
6 Smith-Warner SA, Spiegelman D, Yaun SS, *et al*. Alcohol and breast cancer in women: a pooled analysis of cohort studies. *JAMA* 1998;**297**:535–40
7 Brodsky A, Peele S. Psychosocial benefits of moderate alcohol consumption: alcohol's role in a broader conception of health and well-being. In: Peele S, Grant M, eds. *Alcohol and pleasure: a health perspective*. Philadelphia: Taylor and Francis, 1999:187–207.
8 Office for National Statistics. *Twentieth century mortality CD ROM*. London: 1997.
9 Corrao G, Bagnardi V, Zambon A, *et al*. Exploring the dose-response relationship between alcohol consumption and the risk of several alcohol-related conditions: a meta-analysis. *Addiction* 1999;**94**:1551–73
10 Corrao G, Rubbiati L, Bagnardi V, *et al*. Alcohol and coronary heart disease: a meta-analysis. *Addiction* 2000;**95**:1505–23
11 Erens B, Primatesta P, eds. *Health survey for England 1998*. London: The Stationery Office, 1999.
12 Bridgwood A, Lilly R, Thomas M, *et al. Living in Britain: results from the 1998 General Household Survey*. London: The Stationery Office, 2000.
13 English DR, Holman CCDJ, Milne E, *et al*. *The quantification of drug caused morbidity and mortality in Australia*. Canberra: Commonwealth Department of Human Services, AGPS, 1995.
14 Anderson P. Excess mortality associated with alcohol consumption. *BMJ* 1988;**297**:824–6.
15 White I, Britton A, Nanchahal K, *et al*. Mortality attributable to drinking, drinking too much, or drinking too little: a comparison of methods. *J Public Health Med* 1999;**21**:407–11.
16 Schultz JM, Rice DP, Parker DL. Alcohol-related mortality and years of potential life lost - United States, 1987. *Morbidity and Mortality Weekly Report* 1990;**39**:173–8.
17 Sutocky JW, Schultz JM, Kizer KW. Alcohol-related mortality in California, 1980 to 1989. *Am J Public Health* 1993;**83**:817–23.
18 Murray CJL, Lopez AD, eds. *Global burden of disease*. Boston: Harvard School of Public Health on behalf of World Health Organisation and World Bank, 1996.
19 Single E, Robson L, Xie X, *et al*. The economic costs of alcohol, tobacco and illicit drugs in Canada, 1992. *Addiction* 1998;**93**:991–1006.
20 Shaper AG, Wannamethee G, Walker M. Alcohol and mortality in British men: explaining the U-shaped curve. *Lancet* 1988;2:1267–73.
21 Duffy JC. Alcohol and All-cause Mortality. In: Duffy JC, ed. *Alcohol and illness: the epidemiological viewpoint*. Edinburgh, Scotland: Edinburgh University Press, 1992:26–35.
22 White IR, Altmann DR, Nanchahal K. *'Optimal' levels of alcohol consumption for men and women at different ages, and the all-cause mortality attributable to drinking.* Technical report. London: London School of Hygiene and Tropical Medicine, 2000.
23 Department of Health. *Our healthier nation: A contract for health*. London: The Stationery Office, 1998.
24 Royal College of General Practitioners. *Alcohol—a balanced view.* London: RCGP, 1986.
25 McPherson K, Cavallo F, Rubin E. Alcohol and breast cancer. In: Macdonald I, ed. *Health issues related to alcohol consumption*. London: Blackwell, 1999:214–42.
26 Plant ML. Alcohol and breast cancer: a review. *International Journal of Addictions* 1992;**27**:107–28.
27 Grant M, Litvak J, eds. *Drinking patterns and their consequences*. Washington: Taylor and Francis, 1988.
28 McKee M, Britton A. The positive relationship between alcohol and heart disease in Eastern Europe: potential physiological mechanisms. *J Roy Soc Med* 1998;**91**:402–7.
29 Britton A, McKee M. The relation between alcohol and cardiovascular disease in Eastern Europe: explaining the paradox. *J Epidemiol Community Health* 2000:**54**:328–32.
30 Grønbaek M, Becker U, Johansen D, *et al*. Population based cohort study of the association between alcohol intake and cancer of the upper digestive tract. *BMJ* 1998;**317**:844–8.
31 Tjønneland A, Grønbaek M, Stripp C, *et al*. Wine intake and diet in a random sample of 48763 Danish men and women. *Am J Clin Nutr* 1999;**69**:49–54.
32 Duffy JC. Alcohol and all-cause mortality. In: *Alcohol and illness: the epidemiological viewpoint*. Edinburgh, Scotland: Edinburgh University Press, 1992:28–9.
33 De Fries JHM, Lemmens PHHM, Pietinen, P, *et al*. Assessment of alcohol consumption. In: Macdonald I, ed. *Health issues related to alcohol consumption*. London: Blackwell, 1999:27–62.
34 Hart CL, Davey Smith G, Hole DJ, *et al*. Alcohol consumption and mortality from all causes, coronary heart disease, and stroke: results from a prospective cohort study of Scottish men with 21 years of follow up. *BMJ* 1999;**318**:1725–9
35 Department of Health. *Saving lives: our healthier nation*. London: The Stationery Office, 1999.