18 Tsushima WT, Shimizu AA. Effects of a no-smoking policy upon medical center employees. *Int J Addict* 1991;26:23-8.
19 Baile WF, Gilbertini M, Ulschak F, Snow-Antle S, Hann D. Impact of a hospital smoking ban: changes in tobacco use and employee attitudes. *Addict Behav* 1991;16:419-26.
20 Daughton DM, Andrews CE, Orona CP, Patil KD, Rennard SI. Total indoor smoking ban and smoker behavior. *Prev Med* 1992;21:670-6.
21 Olive KE, Ballard JA. Changes in employee smoking behavior after implementation of restrictive smoking policies. *South Med J* 1996;89:699-706.
22 Stave GM, Jackson GW. Effect of a total work-site smoking ban on employee smoking and attitudes. *J Occup Med* 1991;33:884-90.
23 Rosenstock IM, Stregachis A, Heany C. Evaluation of a smoking prohibition policy in a health maintenance organization. *Am J Public Health* 1986;76:1014-5.
24 Brenner H, Mielck A. Smoking prohibition in the workplace and smoking cessation in the Federal Republic of Germany. *Prev Med* 1992;21:252-61.
25 Farelly MC, Evans WN, Sfekas AE. The impact of workplace smoking bans: results from a national survey. *Tob Control* 1999;8:272-7.
26 Glasgow RE, Cummings KM, Hyland A. Relationship of worksite smoking policies to changes in employee tobacco use: findings from COMMIT. *Tob Control* 1997;6(suppl 2):S44-8.
27 Kinne S, Kristal A, White E, Hunt J. Work-site smoking policies: their population impact in Washington State. *Am J Public Health* 1993;83:1031-3.
28 Woodruff TJ, Rosbrook B, Peirce J, Glantz SA. Lower levels of cigarette consumption found in smoke-free workplaces in California. *Arch Intern Med* 1993;153:1485-93.
29 US Department of Health and Human Services. *Reducing tobacco use: a report of the surgeon general*. Atlanta, GA: US Deparment of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office of Smoking and Health, 2000.
30 US Bureau of the Census. Resident population of the United States by age and sex. www.census.gov/population/estimates/nation/intfile2-1.txt (accessed 17 Aug 2001).
31 US Bureau of Labor Statistics. National employment and wage estimates, www.bls.gov/oes/1999/oes_nat.htm (accessed 17 Aug 2001).
32 Shopland DR, Gerlach KK, Burns DM, Hartman AM, Gibson JT. State-specific trends in smoke-free workplace policy coverage. The current population survey tobacco use supplement 1993 to 1999. *J Occup Environ Med* 2001;43:680-6.
33 Lader D, Meltzer H. *Smoking related behaviour and attitudes, 1999*. London: Office for National Statistics, 2000.
34 Orzechowski W, Walker R. The tax burden on tobacco. Arlington, VA: Orzechowski and Walker, 2000.
35 Office for National Statistics. Mid-2000 UK population estimates. www.statistics.gov.uk/pdfdir/pop0801.pdf (accessed 21 August 2001).
36 Chaloupka F. Clean indoor air laws, addiction and cigarette smoking. *Appl Econ* 1992;24:193-205.
37 Moskowitz JM, Lin Z, Hudes ES. The impact of workplace smoking ordinances in California on smoking cessation. *Am J Public Health* 2000;90:757-61.
38 Emont S, Choi W, Novotny T, Giovina G. Clean indoor air legislation, taxation and smoking behavior in the United States: an ecological analysis. *Tob Control* 1992;2:13-7.
39 Yurekli AA, Zhang P. The impact of clean indoor air laws and cigarette smuggling on demand for cigarettes: an empirical model. *Health Econ* 2000;9:159-70.
40 Wasserman J, Manning WG, Newhouse JP, Winkler JD. The effects of excise taxes and regulations on cigarette smoking. *J Health Econ* 1991;10:43-64.
41 National Cancer Institute. *Major local tobacco control ordinances in the United States*. Bethesda, MD: USDHHS, 1993 (Smoking and Tobacco Control Monograph 3).
42 Stephens T, Pederson LL, Koval JJ, Kim C. The relationship of cigarette prices and no-smoking bylaws to the prevalence of smoking in Canada. *Am J Public Health* 1997;87:1519-21.
43 Heloma A, Jaakola M, Kahkonen E, Reijula K. The short-term impact of national smoke-free workplace legislation on passive smoking and tobacco use. *Am J Pub Health* 2001;91:1416-8.
44 Farkas AJ, Gilpin EA, White MM, Pierce JP. Association between household and workplace smoking restrictions and adolescent smoking. *JAMA* 2000;284:717-22.
45 Lewit E, Hyland A, Kerrebrock N, Cummings K. Price, public policy and smoking in young people. *Tob Control* 1997;6(suppl 2):S17-24.
46 Chaloupka FJ, Pacula RL. Sex and race differences in young people's responsiveness to price and tobacco control policies. *Tob Control* 1999;8:373-7.
47 Wakefield MA, Chaloupka FJ, Kaufman NT, Orleans CT, Barker DC, Ruel EE. Effect of restrictions at home, at school, and in public places on teenage smoking: cross sectional study. *BMJ* 2000;321:333-7.
48 Glantz SA, Jamieson P. Attitudes toward secondhand smoke, smoking, and quitting among young people. *Pediatrics* 2000;106:E82.
49 Fichtenberg CM, Glantz SA. Youth access interventions do not affect youth smoking. *Pediatrics* 2002;109:1088-92.
50 US Department of Health and Human Services. *Reducing the health consequences of smoking: 25 years of progress. A report of the surgeon general*. Rockville, MD: US Deparment of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office of Smoking and Health, 1989.
51 Hymowitz N, Cummings KM, Hyland A, Lynn WR, Pechacek TF, Hartwell TD. Predictors of smoking cessation in a cohort of adult smokers followed for five years. *Tob Control* 1997;6(suppl 2):S57-62.
52 Glantz S, Balbach E. *Tobacco war: inside the California battles*. Berkeley, CA: University of California Press, 2000.

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Alcohol consumption and mortality: modelling risks for men and women at different ages

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ELPS

The full version of this article appears on bmj.com

Abstract

Objective To estimate the relation between alcohol consumption and risk of death, the level of alcohol consumption at which risk is least, and how these vary with age and sex.
Design Analysis using published systematic reviews and population data.
Setting England and Wales in 1997.
Main outcome measures Death from any of the following causes: cancer of lip, oral cavity, pharynx, oesophagus, colon, rectum, liver, larynx, and breast, essential hypertension, coronary heart disease, stroke, cirrhosis, non-cirrhotic chronic liver disease, chronic pancreatitis, and injuries.
Results A direct dose-response relation exists between alcohol consumption and risk of death in women aged 16-54 and in men aged 16-34. At older ages the relation is U shaped. The level at which the risk is lowest increases with age, reaching 3 units a week in women aged over 65 and 8 units a week in men aged over 65. The level at which the risk is increased by 5% above this minimum is 8 units a week in women aged 16-24 and 5 units a week in men aged 16-24, increasing to 20 and 34 units a week in women and men aged over 65, respectively.
Conclusions Substantially increased risks of all cause mortality can occur even in people drinking lower than recommended limits, and especially among younger people.

Introduction

Alcohol consumption increases the risk of various cancers, hypertension, liver disease, unintentional injuries, and violence.[1 2] Definitions of light and moderate alcohol consumption vary, but these levels of consumption are generally found to decrease the risk of ischaemic heart disease.[2–6] For all cause mortality the relation is typically U shaped, with non-drinkers and heavier

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drinkers having higher risks than light and moderate drinkers.[2 7–9] The royal colleges of physicians, psychiatrists, and general practitioners have therefore advised men and women to drink less than 21 and 14 units a week, respectively, whereas the UK government has recommended no more than 4 and 3 units a day, respectively; 1 unit is 8-10 g of alcohol.[10 11] However, the levels giving the lowest or a low risk are likely to vary with age as well as sex and have not been systematically quantified.[12] We used statistical models relating alcohol consumption to the risk of death from single causes to estimate the all cause risk for men and women of different ages in England and Wales.

Methods

Relative risks

Three reviews have quantitatively related alcohol consumption to comprehensive lists of causes of death.[1 13 14] The most appropriate review for our study was by Corrao and others because it included more recent studies, assessed study quality, and estimated risk as a function of alcohol consumption.[14 15]

Corrao and others described each cause specific risk by way of a linear, quadratic, or cubic function of alcohol consumption, or, for ischaemic heart disease, a model involving linear and square root terms. Where appropriate they excluded studies of lower quality. They reported results separately when significant differences were found between Mediterranean and non-Mediterranean countries, case-control and cohort studies, incident disease and death, or men and women: we used the results for non-Mediterranean countries, cohort studies, and deaths. Otherwise we used the pooled results. We expressed alcohol consumption in units a week, taking 1 unit as 9 g of alcohol.[16] The risk functions for each cause of death by alcohol consumption are given on bmj.com.

Alcohol consumption

Alcohol consumption was reported by respondents aged 16 and over in the 1996-7 general household survey.[17] We computed the proportions of men and women in England and Wales drinking 0 units of alcohol a week, drinking occasionally (taken as 0.25 units a week), drinking from 1 up to 100 in increments of 1 unit a week, and drinking more than 100 units a week, for age bands 16-24, 25-34, 35-44, and so on up to over 85.

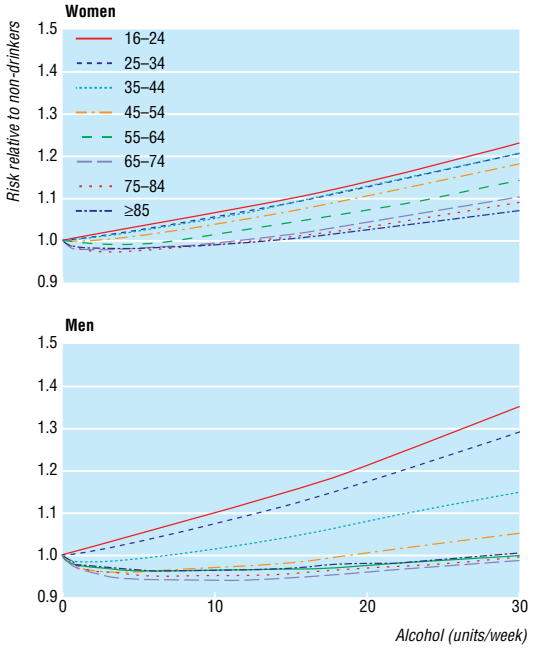

Fig 1 Risk of all cause mortality (relative to non-drinkers) by level of alcohol consumption in women and men

Mortality

We obtained data on mortality for England and Wales in 1997 from the Office for National Statistics.[18]

Pooling causes

We estimated absolute risk functions from the relative risk functions for each sex, age band, and cause by using the observed number of deaths and the distribution of alcohol consumption (see bmj.com). We summed the absolute cause specific risks to get all cause mortality for each sex and age band. The nadir is the level of alcohol consumption at which all cause risk is lowest.[16]

Results

Alcohol risk relations

Figure 1 shows the relation between all cause mortality and alcohol consumption, by age and sex. The absolute risks vary widely (table), so we show all risks relative to non-drinkers. For women there is a positive relation up

Nadirs and 5% bounds (units a week) with 95% confidence intervals showing uncertainty owing to relative risk functions and percentage annual risk

	Age (years)							
	16-24	25-34*	35-44	45-54	55-64	65-74	75-84	⩾85
Women								
Nadir	0	0	0.2 (0.1 to 0.3)	0.5 (0.4 to 0.6)	1.6 (1.3 to 1.9)	2.7 (2.3 to 3.1)	3.2 (2.7 to 3.7)	3.1 (2.4 to 3.8)
5% upper bound	7.9 (7.2 to 8.6)	8.7 (7.8 to 9.6)	9.3 (8.0 to 10.6)	11.5 (9.8 to 13.2)	14.5 (12.7 to 16.3)	17 (14.8 to 19.2)	18 (15.4 to 20.6)	20
Annual risk (%):								
Non-drinkers	0.03	0.04	0.10	0.25	0.69	2.03	5.48	15.3
Nadir	0.03	0.04	0.10	0.25	0.68	1.98	5.35	15.0
Men								
Nadir	0	0.1 (0.1 to 0.1)	1.8 (1.5 to 2.1)	5.1 (4.5 to 5.7)	7.2 (6.4 to 8.0)	8.4 (7.4 to 9.4)	8.0 (6.7 to 9.3)	7.7 (6.0 to 9.4)
5% upper bound	5.3 (4.8 to 5.8)	6.9 (6.3 to 7.5)	13.3 (11.9 to 14.7)	21 (18.6 to 23.4)	26 (23.3 to 28.7)	30 (26.4 to 33.6)	31 (26.5 to 35.5)	34 (27.5 to 40.5)
Annual risk (%):								
Non-drinkers	0.06	0.08	0.14	0.39	1.17	3.39	8.47	19.4
Nadir	0.06	0.08	0.14	0.37	1.10	3.21	8.05	18.6

*0.025 rounded to 0.

to age 35-44, but the U shape appears from age 45-54. For men aged below 35 the curve is steeper than it is for women, but the U shape appears at age 35-44, and the reduction in mortality in the lightest drinkers is larger and is sustained up to higher levels of consumption than for women. Drinking at the royal colleges' recommended limit increases risk by 9% in women aged 16-24 and by 23% in men aged 16-24. For government limits these figures are 15% and 32%, respectively.

Estimated nadirs

Figure 2 and the table show the estimated nadirs. The nadir increases from 0 at ages 16-34 to around 3 units a week in women and around 8 units a week in men aged over 65. The 95% confidence intervals around the estimated nadirs are narrow, but they account only for uncertainty in the relative risks.[19]

The 5% bounds are the levels of alcohol consumption at which risk is no more than 5% greater than the risk at the nadir. For women the 5% bound increases from 8 units a week at ages 16-24 to around 20 units a week over age 85. For men it increases from only 5 units a week at ages 16-24 (reflecting the steeper slope of the risk curve in young men compared with young women) to 30-35 units a week over age 65.

Sensitivity analyses

An alternative risk function for ischaemic heart disease lowers the nadirs, for example from 8 to 5 units a week in men aged over 65. An alternative way of splitting deaths from stroke lowers the nadir for men aged over 75. Assuming ischaemic stroke to be unrelated to alcohol consumption slightly increases the nadirs, whereas assuming breast cancer to be unrelated to alcohol consumption has no appreciable effect.

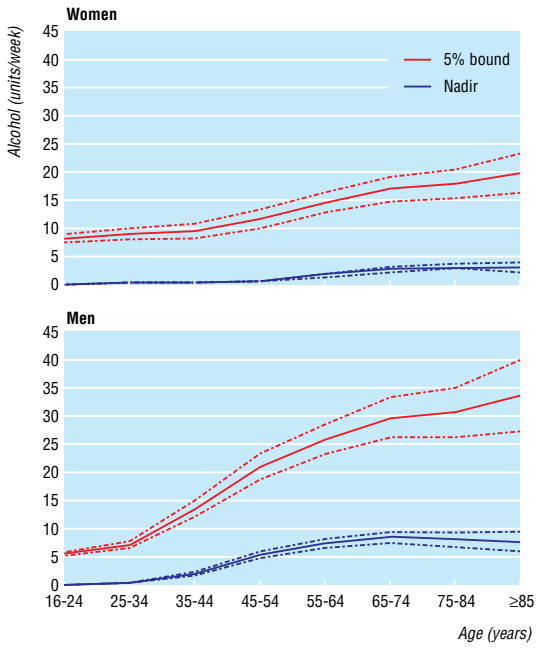

Fig 2 Level of alcohol consumption at which mortality is least (nadir) and level at which risk is raised by 5% above this minimum risk in women and men (95% confidence intervals show uncertainty due to relative risk functions only)

What is already known on this topic

Non-drinkers and heavy drinkers have higher all cause mortality rates than light drinkers—the U shaped curve

The precise shape and location of the U are likely to depend on age and sex, but this has not been quantified

What this study adds

The level of alcohol consumption that carries the lowest mortality ranges from 0 in men and women aged under 35 to 3 units a week in women aged over 65 and 8 units a week in men aged over 65

The level of alcohol consumption that carries a 5% increase in mortality increases with age from 8 to 20 units a week in women and from 5 to 34 units a week in men

Our calculations were for England and Wales in 1997: nadirs are likely to be lower in the future and in countries with less ischaemic heart disease

Discussion

If our results are not subject to bias or confounding (see bmj.com) and if the effects of alcohol consumption act over no more than 5-10 years, then the average person can decrease his or her risk of mortality by drinking at a level nearer the nadir.

Possible evidence based guidelines for sensible drinking can be derived from figure 2 and the table if no more than a 5% increase in risk of mortality is considered acceptable. Women would be advised to limit their drinking to 1 unit a day up to age 44, 2 units a day up to age 74, and 3 units a day over age 75. Non-drinking men aged 55-84 have a risk slightly more than 5% above the minimum risk, but we would not encourage these men to drink, because this might increase the overall public health burden of heavier drinking. Men would be advised to limit their drinking to 1 unit a day up to age 34, 2 units a day up to age 44, 3 units a day up to age 54, 4 units a day up to age 84, and 5 units a day over age 85. These levels are similar to current limits at older ages but considerably lower at younger ages.

Alternatively, the 9-32% increase in risk for younger people at the current limits of sensible drinking might be considered acceptable because it implies a smaller absolute increase than the same percentage at older ages, even allowing for additional years of life lost through deaths at a younger age. Public health must also take account of morbidity and social harm, which are harder to measure than mortality but much more adversely affected by alcohol consumption.[20] Finally, as most deaths attributable to alcohol at younger ages are due to injuries, a greater focus could be placed on avoiding risky patterns of drinking rather than on reducing average alcohol consumption.[21 22]

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1 English DR, Holman CDJ, Milne E, Winter MG, Hulse GK, Codde JP, et al. *The quantification of drug caused morbidity and mortality in Australia 1995*. Canberra: Commonwealth Department of Human Services and Health, 1995.
2 Anderson P, Cremona A, Paton A, Turner C, Wallace P. The risk of alcohol. *Addiction* 1993;88:1493-1508.
3 Marmot M, Brunner E. Alcohol and cardiovascular disease: the status of the U shaped curve. *BMJ* 1991;303:565-8.
4 Maclure M. Demonstration of deductive meta-analysis: ethanol intake and risk of myocardial infarction. *Epidemiol Rev* 1993;15:328-51.
5 Klatsky AL. Epidemiology of coronary heart disease—influence of alcohol. *Alcohol Clin Exp Res* 1994;18:88-96.
6 Chick J. Alcohol, health, and the heart: implications for clinicians. *Alcohol Alcohol* 1998;33:576-91.
7 Beaglehole R, Jackson R. Alcohol, cardiovascular diseases and all causes of death: a review of the epidemiological evidence. *Drug Alcohol Rev* 1992;2:275-90.
8 Poikolainen K. Alcohol and mortality: a review. *J Clin Epidemiol* 1995;48:455-65.
9 Rehm J, Bondy S. Alcohol and all-cause mortality: an overview. In: Novartis Foundation Symposium, ed. *Alcohol and cardiovascular diseases*. Chichester: Wiley, 1998.
10 Royal Colleges of Physicians, Psychiatrists, and General Practitioners. *Alcohol and the heart in perspective: sensible limits reaffirmed*. London: Royal Colleges, 1995.
11 Inter-Departmental Working Group. *Sensible drinking*. London: Department of Health, 1995.
12 Holman CDJ, Armstrong BK. The quantification of alcohol-caused morbidity and mortality in Australia: the authors respond. *Med J Aust* 1992;157:560-1.
13 Holman CDJ, Armstrong BK, Arias LN, Martin CA, Hatton WM, Hayward LD, et al. *The quantification of drug caused morbidity and mortality in Australia* (appendix 1). Canberra: Commonwealth Department of Community Services and Health, 1988.
14 Corrao G, Bagnardi V, Zambon A, Arico S. Exploring the dose-response relationship between alcohol consumption and the risk of several alcohol-related conditions: a meta-analysis. *Addiction* 1999;94:1551-73.
15 Corrao G, Rubbiata L, Bagnardi V, Zambon A, Poikolainen K. Alcohol and coronary heart disease: a meta-analysis. *Addiction* 2000;95:1505-23.
16 White IR. The level of alcohol consumption at which all-cause mortality is least. *J Clin Epidemiol* 1999;52:967-75.
17 Office for National Statistics, Social Survey Division. *General household survey, 1996-1997 (computer file SN 3804)*, 2nd edn. Colchester, Essex: Data Archive [distributor], 5 Oct 1998.
18 Office for National Statistics. *Twentieth century mortality: 95 years of mortality data in England and Wales by age, sex, year and underlying cause*. [CD ROM and 1997 Update disk]. London: ONS, 1997.
19 White IR, Nanchahal K. *The number of deaths and person-years of life lost attributable to alcohol consumption in England and Wales in 1995*. London: London School of Hygiene and Tropical Medicine, 1997. [Technical report.]
20 Murray JL, Lopez AD, eds. *The global burden of disease: a comprehensive assessment of mortality and disability from diseases, injuries and risk factors in 1990 and projected to 2020*. Cambridge, MS: Harvard University Press, 1996.
21 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*. London: London School of Hygiene and Tropical Medicine, 2000. [Technical report.]
22 Britton A, McPherson K. Mortality in England and Wales attributable to current alcohol consumption. *J Epidemiol Community Health* 2001;55:383-8.

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Analysis of predicted coronary heart disease risk in England based on Framingham study risk appraisal models published in 1991 and 2000

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In 2000 the UK government launched the national service framework for coronary heart disease, setting national standards for improving prevention, diagnosis, and treatment. In agreement with recent recommendations on preventing coronary heart disease[1] and managing hypertension,[2] this programme includes use of coronary risk appraisal models from the Framingham study published in 1991[3] to help identify patients eligible for drug treatment. These models were updated in 2000,[4] incorporating further follow up and additional risk factors. We compare the predicted risks calculated using the two models and assess the implications for preventing heart disease.

Methods and results

The health survey for England is an annual, nationwide, household based, cross sectional survey of a representative sample of the population. We used the 1998 survey data for 5518 (62.3% of 8852) participants aged 35-74 with complete information on factors needed for assessment of coronary disease risk, after exclusion of 738 (7.7% of 9590) participants reporting angina, heart attack, or stroke diagnosed by a doctor.[5] The 2000 models allow calculation of risk over a period of four years,[4] whereas the 1991 models permit estimation of risk over 4-12 years.[3] We estimated the 10 year and four year probabilities of developing heart disease predicted using the 1991 equations and the four year risk predicted using the 2000 equations.

Summary statistics for four year coronary disease risk per 100 population based on the 1991 and 2000 models within a range of risk categories show that both models generally produce similar distributions (table). Although substantial statistical agreement exists between classification of participants into risk categories based on the two models, participants within each category based on the 1991 models were distributed across a wide range of risk categories based on the 2000 models.

Comment

Although population distributions of coronary risk calculated with the two models are generally similar, a significant number of people meeting criteria for drug treatment on the basis of the 1991 models would not meet the equivalent criteria on the basis of the 2000 models. Current UK guidelines generally recommend offering drug treatment for hypertension or hypercholesterolaemia to patients with a 10 year risk $\geq 15\%$.[1 2] We used a 5% risk of a coronary event in four years as being equivalent to a 10 year risk of 15%, rather than 6% over four years, because risk increases exponentially rather than linearly with age. Had we used 6%, the discrepancy between the 1991 and 2000 models would have been even greater.