RESEARCH REPORT

Changing drinking pattern does not influence health perception: a longitudinal study of the atherosclerosis risk in communities study

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Objective: To investigate if dynamic changes in the pattern of alcoholic beverages consumption are associated with modifications in health perception.

Design, setting, and participants: This study investigated 12 332 middle aged men and women from the atherosclerosis risk in communities study who reported drinking status and perceived health triennially from 1987 to 1995. Crude and adjusted risks for change in health perception between visits two and three by change in drinking status between visits one and two were computed. In the multivariate analysis the sample was restricted to participants with stable drinking status between visit two and three and stable health perception between visits one and two, to assure that exposure and outcome were not temporary. Covariates included age, sex, race, income, smoking status, educational level, and obesity.

Results: Health for persons who stopped or started drinking, or continued to abstain was more likely to decline than was health for persons who continued to drink even after adjustment and restrictions (drinking cessation: OR=1.6, 95% CI=1.1, 2.3; started drinking; OR= 1.4, 95% CI=0.9, 2.2; continued abstaining from alcohol: OR= 1.5, 95% CI=1.3, 1.9). Among participants with poor perceived health, starting, stopping, or continuing to abstain from alcohol did not improve health in relation to participants that continued to drink.

Conclusion: Increasing and decreasing drinking patterns and continuous abstinence were associated with declining health perception in comparison with continuous drinking, while starting or stopping drinking did not improve health perception of persons with poor perceived health. These findings suggest that change in health perception was not biologically related to alcohol consumption.

The relative consistency of a protective effect from light to moderate alcohol consumption against cardiovascular disease is seen in multiple population studies.^{1–16} These studies, the plausible biological mechanisms,^{17–18} and experimental animal studies showing a beneficial effect of alcohol on atherosclerosis^{19–20} support the idea that alcohol has a protective effect on all cause mortality through a reduction in coronary artery disease. However, other studies continue to suggest that biases, such as not adjusting for socioeconomic or psychological factors associated with drinking status^{5–7 21–23} or including former drinkers within the control group,^{24–25} may contribute to the putative beneficial effect of alcohol consumption. Some benefit has been ascribed to even very occasional drinking,²⁶ which may suggest that the pattern of alcohol consumption is really a marker of other beneficial factors.²³ The associations between alcohol consumption and health outcomes were mostly studied in cohort studies that measured the pattern of consumption just at one point in time. Change in the amount of alcohol consumption throughout the cohort follow up may influence the association between the baseline amount of consumption and health outcomes,²⁷ such as perceived health, which has been repeatedly shown to be associated with overall mortality.^{28–29} Despite the absence of a direct evidence linking perceived health and incidence of coronary heart disease, changes in perceived health status may be secondary to subtle modifications in health secondary to coronary heart disease or other diseases that lead to death. Self perceived health and the pattern of consumption of alcoholic beverages were repeatedly measured in follow up interviews of the atherosclerosis

risk in communities (ARIC) study, permitting an evaluation of the association between change in the pattern of consumption and health outcomes. In this report we evaluated the association between the modification in the pattern of alcohol consumption between visit one and two (exposure) and perceived health in visit three (outcome) of the ARIC cohort.

METHODS

Study population

A detailed description of the design of the ARIC study, a prospective investigation of atherosclerosis, has been published.³⁰ The study protocol was approved by the institutional review board at each study centre, and each participant signed an informed consent before participating in each examination of the study. The closed, middle aged cohort was recruited between 1987 and 1989 and was seen at about three year intervals from four centres: 14% from Forsyth County, NC, were black; 100% from Jackson, MS, were black; and essentially all participants from the suburbs of Minneapolis, MN, and Washington County, MD, were white. The baseline examination (examination 1) was completed by 15 792 participants, which represented a 65%–67% response rate except at Jackson where the response rate was 46%.³⁰ Return rates for examinations two and three were 93% and 86% respectively³¹ for persons still living. From the 15 425 black and white participants who provided information on alcohol consumption and were 45–64 years of age at baseline, there were 12 332 persons who provided information on drinking and health at examinations two and three. For evaluation of

Table 1 Baseline characteristics of the study sample, the subset with stable health status between examinations one and two and stable drinking status between examinations two and three (analytical sample), and those excluded to form the analytical sample, the ARIC cohort, 1987–95 (mean (SD) or number and percentage as indicated)

Characteristic	Study population	Analytical sample	Excluded from study population	
	n = 12332*	n = 9272*	n = 3060*	p Value
Age, mean (SD)	54.0 (5.6)	53.9 (5.6)	54.3 (5.7)	0.004
Black participants, n (%)	2709 (22.0)	1707 (18.4)	1002 (32.8)	<0.0001
Female, n (%)	6876 (55.8)	4122 (44.5)	1334 (43.6)	0.4
Good perceived health,† n (%)	10385 (84.2)	8374 (90.3)	2011 (65.7)	<0.0001
Chronic disease,‡ n (%)	5418 (44.4)	3776 (41.1)	1642 (54.4)	<0.0001
Income tertiles,§ n (%)				<0.0001
Lowest	3120 (26.2)	2139 (23.8)	981 (33.4)	
Middle	4702 (39.4)	3507 (39.0)	1195 (40.7)	
Highest	4099 (34.4)	3341 (37.2)	758 (25.8)	
Education level, n (%)				<0.0001
<High school	2432 (19.7)	1549 (16.7)	883 (28.9)	
High school	5193 (42.2)	3925 (42.4)	1268 (41.5)	
>High school	4693 (38.1)	3785 (40.9)	908 (29.7)	
Examination centre, n (%)				<0.0001
Forsyth Co, NC	3210 (26.0)	2400 (25.9)	810 (26.5)	
Jackson, MS	2397 (19.4)	1513 (16.3)	884 (28.9)	
Minneapolis, MN	3425 (27.8)	2865 (30.9)	560 (18.3)	
Washington Co, MD	3300 (26.8)	2494 (26.9)	806 (26.3)	
Drinking status,¶ n (%)				<0.0001
Never	3036 (24.6)	2272 (24.5)	764 (25)	
Former	2118 (17.2)	1463 (15.8)	655 (21.4)	
Drinker	7178 (58.2)	5537 (59.7)	1641 (53.6)	
Occasional** (<weekly)	2271 (31.6)	1573 (28.4)	698 (42.5)	<0.0001
Regular** (≥weekly)	4907 (68.4)	3964 (71.6)	943 (57.5)	<0.0001
Moderate** (2 drinks/week)	4190 (58.4)	3404 (61.5)	786 (47.9)	<0.0001
Current smoker, n (%)	2789 (22.6)	1988 (21.5)	801 (26.2)	<0.0001
Former smoker, n (%)	4118 (33.4)	3141 (33.9)	977 (32)	0.05
BMI ≥30, n (%)	3233 (26.2)	2285 (24.7)	948 (31)	<0.0001

*Maximum number of subjects available, but the numbers available are less for some variables because of missing data. †Good perceived health refers to self reported health as excellent or good relative to others of the same age. ‡Chronic disease refers to prevalence of self reported physician diagnosed hypertension, heart attack, stroke, diabetes, cancer, and/or chronic lung disease at baseline. §Ethnic-sex specific income tertiles at examination three. ¶Regular drinkers reported at least one drink/week; occasional drinkers reported less than one drink/week; former drinkers reported no consumption of alcoholic beverages in the past six months, lifetime abstainers reported never consuming alcohol. **Percentage of those who reported drinking. Moderate and regular drinking are not mutually exclusive. p Value: for χ^2 or t test for comparison of analytic subset to those excluded from the study population.

the impact of a change in drinking status between examination one and two on health change by examination three, separate analyses evaluated either all persons reporting good health at examination 2 (n = 10 470) or all persons reporting poor health at examination two (n = 1862) for evaluation of health decline and health improvement respectively.

Study variables

Ethnicity, age, sex, and education were self reported at baseline. Education was categorised for this study into three levels: having completed less than high school, high school, and greater than high school. Self reported total family

income from examination three was used to determine ethnic/sex specific income tertiles. Baseline self report was used to classify persons as current, former, or never smokers. A history of physician diagnosed chronic disease was elicited at baseline and at examinations two and three by asking whether a doctor had ever told the participant they had high blood pressure, heart attack, stroke, diabetes, cancer, or chronic lung disease. Chronic disease status was coded for each examination: yes if the participant said yes to any of the previously mentioned diseases, and no if the participant said no to all of the diseases. Perceived health was determined from questions asked at baseline and from the annual follow

Table 2 Among participants with good perceived health at examination two, the odds ratios (95% CI) for perceived health decline between examinations two and three for persons who stopped drinking, started drinking, or continued to abstain between examinations one and two relative to persons who continued to drink, ARIC study, 1987–95

Drinker status examinations 1 to 2 (n)	Perceived health decline between examinations 2–3*				
	Study population		Analytical sample†		
	Health declined (n and %)	n = 10470 Unadjusted	All analytical n = 8134 Adjusted OR (95% CI)	Smokers n = 1664 Adjusted OR (95% CI)	Non-smokers n = 6470 Adjusted OR (95% CI)
Continued to drink (5788)	444 (7.8)	1.0	1.0	1.0	1.0
Stopped drinking (649)	90 (13.9)	1.9 (1.5, 2.5)	1.6 (1.1, 2.3)	1.7 (0.9, 3.0)	1.6 (1.03, 2.5)
Started drinking (576)	80 (13.9)	1.9 (1.5, 2.5)	1.4 (0.9, 2.2)	0.8 (0.4, 1.9)	1.8 (1.1, 3.0)
Continued to abstain (3457)	488 (14.1)	2.0 (1.7, 2.3)	1.5 (1.3, 1.9)	1.0 (0.7, 1.5)	1.7 (1.4, 2.2)

*Among persons with good health at examination 2. †Analysis restricted to participants with good health between examinations 1 and 2 and stable drinking status between examinations 2 and 3, adjusted for age, sex, race, income (3 levels), education (3 levels), current smoking status, and obesity.

up interviews preceding examinations two and three. At each encounter the participant was asked to compare their health to the health of other people of the same age. For this study, perceived health was collapsed from four levels into two: excellent or good as good, and fair or poor as poor. Health decline was defined as changing from good to poor perceived health between study examinations and health improvement was changing from poor to good perceived health.

Alcohol consumption was determined by asking participants at each examination if they currently drank alcoholic beverages. Non-current drinkers who reported ever consuming alcoholic beverages were classified as former drinkers, and those who reported no previous drinking were classified as lifetime abstainers. Current drinkers were asked how many glasses of wine (4 oz), servings of beer (12 oz), and drinks of hard liquor (1.5 oz) they usually consumed per week. As protective effects for alcohol consumption have been reported for persons who drink alcohol even occasionally,^{15–26,32} we classified persons who stated they drank alcoholic beverages as follows: drinkers who reported consuming at least one drink per week were classified as regular drinkers and drinkers who reported less were classified as occasional drinkers.

Statistical analysis

SAS version 8 was used for all analyses. Unadjusted means and distributions of baseline characteristics were determined for the study population and for the most restricted analytical samples (defined below). Logistic regression analyses were used to determine the odds ratios (OR) and 95% confidence intervals (CI). Analyses evaluated whether changes in drinking status or continued abstinence from drinking between examinations one and two were associated with health change between examinations two and three relative to persons who continued to drink. Health decline or improvement was evaluated respectively among persons in good health or poor health at examination two. For each set of analyses, two models were used: an unadjusted model, and a restricted model adjusting for covariates. Restriction was used to assure the exposure was not temporary and had

occurred before the outcome of interest. Participants selected for this analysis had to have health status stable between examinations one and two and the same drinking status at examinations two and three. In addition to restriction, the following covariates were included in the model: race, sex, age, highest ethnic/sex specific examination three income group, current smoking status, body mass index, and educational attainment.^{33–36} We included interaction terms to explore if some characteristics, such as smoking status, were effect modifiers of the association between change in alcohol consumption and change in health perception.

RESULTS

Baseline characteristics are shown in table 1 for the full study sample, for the analytical sample with the restrictions to assure the temporal association between the exposures and outcomes, and for persons excluded from the population sample. The study sample was middle aged (mean age 54), largely white (78%), contained 56% women, and about 80% with at least a high school education. While 84% of the study population reported good perceived health, 44% reported one or more chronic diseases. About one fourth of the study population reported never drinking; about 40% drank at least one drink per week; 18% drank less than one drink per week; and 17% reported drinking in the past. Many reported being current (23%) or former (33%) smokers and over one fourth were obese. The characteristics of the restricted sample were different in a number of ways that could affect health status relative to the overall study population. Comparisons of the analytical and excluded sample highlight the potential differences (table 1). In comparison with the excluded sample, the analytical sample contained significantly younger participants and a significantly larger proportion of persons with the following characteristics: good perceived health, highest income and educational levels, residence in Minnesota, and former smoker status. In contrast, the analytical sample contained a significantly smaller proportion of persons with the following characteristics: chronic disease prevalence, lowest income or educational level, residence in

Figure 1 Odds ratios (95% CI) for health decline or improvement between visits two and three among participants with good or poor perceived health at visit two for persons who stopped drinking, started drinking, or continued to abstain between examinations one and two relative to persons who continued to drink; analysis restricted to participants with stable health status between examinations one and two and stable drinking status between examinations two and three, adjusted for age, sex, race, income (3 levels), education (3 levels), current smoking status, and obesity. ARIC study, 1987–95.

Table 3 Among participants with poor perceived health, the odds ratios (95% CI) for perceived health improvement between examinations two and three for persons who stopped drinking, started drinking, or continued to abstain between examinations one and two relative to persons who continued to drink, ARIC study, 1987–95

Drinker status examinations 1 to 2 (n)	Study population Unadjusted	Analytical sample†	
	Health improved (n and %)	OR (95% CI)	Adjusted OR (95% CI)
Continued to drink (612)	287 (46.9)	1.0	1.0
Stopped drinking (129)	55 (42.6)	0.8 (0.6, 1.2)	1.1 (0.5, 2.1)
Started drinking (128)	49 (38.2)	0.7 (0.5, 1.04)	1.1 (0.5, 2.4)
Continued to abstain (993)	342 (34.4)	0.6 (0.5, 0.7)	0.9 (0.6, 1.3)

*Among persons with poor health at examination 2. †Analyses restricted to participants with poor health at examinations 1 and 2 and stable drinking status between examinations 2 and 3, adjusted for age, sex, race, income (3 levels), education (3 levels), current smoking status, and obesity.

Jackson, former drinkers, occasional drinkers, current smokers, and obese participants.

Logistic regression analyses showed that persons who stopped drinking, started drinking, or continued to abstain from drinking all had about twice the odds of health decline as persons who continued to drink, in unadjusted analyses (table 2). The effects were attenuated moderately in the restricted, analytical sample that was adjusted for socio-demographic factors and other characteristics that might predict health decline (that is, being obese and current smoking status). As smoking status was found to be a significant effect modifier (smoker-abstainer interaction $p = 0.05$; smoker-drink initiation interaction $p = 0.03$) analyses were stratified by current smoking status. Drinking cessation was associated with a detrimental effect on perceived health among both smokers and non-smokers, while starting to drink and continuing to abstain were found to have a significant, detrimental association only among non-smokers.

In logistic models with improvement in health as the outcome and persons who continued to drink as the reference population (table 3), drinking changes (stopping or starting) and continued abstinence were all inversely associated with health improvement in unadjusted analyses, but only continued abstinence from alcohol was statistically significant. In the restricted sample, adjusting for socio-demographic and health risk factors attenuated all associations and none remained significant. Smoking status was not found to be a significant effect modifier (p value ≥ 0.8 for each interaction). Figure 1 shows the odds of health improvement or worsening between visits two and three among participants with poor or good perceived health at visit two respectively by drinking status between visit one and two (analysis adjusted for confounding factors in the restricted subsample).

DISCUSSION

In this analysis of the ARIC cohort, it was possible to identify the dynamics between alcohol drinking status change and subsequent modifications in health perception. The direction of the associations suggests that the modification of alcohol consumption was not associated with physiological effects. Both starting and stopping drinking were associated with declining health perception, as was the state of permanent abstinence from alcohol. People who did not modify their pattern of consumption remained with good perceived health relative to persons who changed drinking status. These associations are in accordance with the interpretation that the beneficial health effects of alcohol consumption may be secondary to a healthier behaviour.²³ The low risk for stable drinkers could be secondary to their better health profile, for

What this paper adds

- The idea that the consumption of a moderate amount of alcoholic beverages protects against some diseases, in particular against coronary heart disease, is almost consensual among physicians and the public at large. The protection arising from alcoholic beverage consumption, however, may be a surrogate marker of healthy behaviour.
- This study shows the starting, stopping, and continuous abstinence from alcoholic beverages are associated with a subsequent decline in health perception (among persons with good health perception) in relation to continuous drinkers, and that starting, stopping, or continuous abstinence from alcoholic beverages did not improve health perception (among persons with poor health perception). These findings strengthen the interpretation that alcohol consumption is not biologically related to favourable health outcomes. The liberal view by physicians regarding alcohol consumption is questionable.

reasons other than the traditional confounders included in the multivariable analysis (for example, their psychological and behavioural characteristics). We could not find any plausible reason for the differential association between starting drinking and continuous abstinence with health perception in smokers and non-smokers. It is possible that the negative health effects of smoking overwhelm most relations between drinker status change and health change among smokers. As both starting drinking and continuous abstinence in smokers had risk ratios not different from the control group, it is unlikely that the reasons for this are among any biological effect of ethanol in smokers. Other reports have suggested the relation between health and alcohol is complex.^{12 23 37–39} Estimates of the actual causes of death in the USA continue to report alcohol consumption as one of the leading causes of death,⁴⁰ while numerous observational studies of the impact of alcohol consumption on all cause mortality have indicated a beneficial effect.^{1–7 9–12 14–16 41} Recent studies have shown the need to continue to evaluate alcohol's benefit.^{23 25 42 44} Studies have suggested publication biases,⁴² lack of protective effects from drinking in some populations,^{43 44} qualitatively different associations between drinking and coronary heart disease for black people compared with white people,²³ protective effects identified for diseases thought to be unrelated to alcohol's physiological effects,^{4 15} and protective effects at low levels or frequencies

(as few as 11 drinks per year)²⁶ of alcohol consumption that would not be anticipated to have a physiological effect.^{15 23 26 32} While underreporting alcohol consumption could explain the latter effect, so could residual confounding if drinking is an indicator of better socioeconomic status or other health promoting behaviours for which adjustment is incomplete. While non-differential confounding usually results in bias towards the null, studies in which there is a strong confounder and where the association between exposure and disease is weak can result in distorted relations.⁴⁵

In this study, continued drinkers had better health than continued abstainers suggesting a benefit of drinking alcohol. However, the significant interaction between the drinker status at examinations separated by three years suggest that the association may be more complex than a simple physiological effect of alcohol. Not only were persons who continued to abstain or who stopped drinking significantly more likely than persons who continued to drink to have a health decline, but so were persons who started to drink, and the magnitude of the effect was similar. If the reason for the beneficial associations between health and drinking were attributable to the physiological effects of alcohol alone, then it would be anticipated that those who continued to abstain would have had the greatest risk of health decline. Persons who stopped drinking should have experienced the putative beneficial effect of alcohol before drinking cessation while lifetime abstainers would not have. Therefore, unless the benefit is extremely short term, the similar risks of health decline among continued abstainers and new non-drinkers, even after adjustment and restriction of sample, suggest that at least some of the putative benefit of drinking is from confounding factors for which adjustment is incomplete. Also, it would have been anticipated that drinking would have had a beneficial effect on health improvement and none was found. Others have reported no improvement in longevity for new drinkers⁴⁶ supporting this aspect of our results. Of course those in poor health may have had health problems that are exacerbated by drinking such as hypertension or liver disease thus preventing any benefit.

While the association of drinking change with both health decline and health improvement may seem contradictory, it would be easy to speculate why such associations could occur. As persons become more unsteady from medical conditions or because of interaction of alcohol with medications, persons may stop drinking. However, much publicity has been presented about the benefit of drinking; so, persons who experience a decline in perceived health might start drinking.

The relation between health perception and underlying diseases is also complex and factors contributing to good perceived health are not uniform in all groups.⁴⁷ So, one limitation of this study is that perceived health was used as the outcome. However, as poor perceived health has been shown to be a strong predictor of mortality with a twofold²⁸ increase in mortality risk in several populations, independently of social class and other confounders,²⁹ the association identified in this study should be an adequate indicator of health.

There may have been misclassification. Subtle symptoms could have led to drinking cessation and yet have been insufficient to lead to self classification of poor health that could result in an underestimation of the proportion of persons who stop drinking after a health decline. One study showed that much of the previously reported cardioprotective effect of recent alcohol consumption was largely attributable to the confounding effect of prodromal symptoms on drinking.⁴⁸ So, we cannot exclude the possibility that subtle health differences led to misclassification of poor health that

may have biased the results toward the null. Alternatively, drinkers may continue to report their longstanding drinking status after they have stopped drinking. If this were to happen, non-drinkers not experiencing the effects of alcohol would be misclassified as drinkers resulting in an underestimation of the beneficial impact of alcohol.

Other limitations include self report of drinking, lack of information at baseline about the pattern of drinking,^{43 49 50} potential variation in associations across centres in the ARIC study, and missing data. Self reported consumption of specific alcoholic beverages has been found to provide useful estimates of alcohol intake^{51 52} and so the data in our study on drinking should be adequate. Our study includes centres where prevalence of drinking is low (Jackson) and high (Minnesota), so overall our study should provide an adequate sample of US drinking. However, given the low prevalence and level of alcohol consumption in ARIC, the results may not be applicable to populations with different drinking characteristics or attitudes. Over 2000 persons eligible for the study at baseline could not be included because of missing follow up data and persons missing data varied somewhat from those in the study population. Heavy drinkers, the persons at greatest risk of negative effects of drinking, were more likely to be missing from the follow up examination and so were excluded from this study. In this and other studies, the loss of heavy drinkers could result in overestimation of a beneficial effect for drinking. Few controlled clinical trials exist regarding the benefit of alcohol and the problem regarding the benefit of alcohol remains.⁵³ A recent trial failed in identifying any pharmacological effect of other components of wine,⁵⁴ strengthening the interpretation that the effects of alcoholic beverages are produced by alcohol itself, including the increasing of blood pressure.⁵⁵

In conclusion, we showed that starting or stopping drinking were associated with subsequent decline of health perception, and that starting or stopping drinking did not improve health of persons with poor perceived health. These findings suggest that change in health perception was not biologically related to alcohol consumption.

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REFERENCES

- 1 **Boffetta P**, Garfinkel L. Alcohol drinking and mortality among men enrolled in an American Cancer Society prospective study. *Epidemiology* 1990;1:342-8.

- 2 Cullen KJ, Knuiman MW, Ward NJ. Alcohol and mortality in Busselton, Western Australia. *Am J Epidemiol* 1993;137:242-8.
- 3 de Labry LO, Glynn RJ, Levenson MR, et al. Alcohol consumption and mortality in an American male population: recovering the U-shaped curve—findings from the normative Aging Study. *J Stud Alcohol* 1992;53:25-32.
- 4 Doll R, Peto R, Hall E, Wheatley K, et al. Mortality in relation to consumption of alcohol: 13 years' observations on male British doctors. *BMJ* 1994;309:911-18.
- 5 Duffy JC. Alcohol consumption and all-cause mortality. *Int J Epidemiol* 1995;24:100-5.
- 6 Camargo CA Jr, Hennekens CH, Gaziano JM, et al. Prospective study of moderate alcohol consumption and mortality in US male physicians. *Arch Intern Med* 1997;157:79-85.
- 7 Gronbaek M, Becker U, Johansen D, et al. Type of alcohol and mortality from all causes, coronary heart disease, and cancer. *Ann Intern Med* 2000;133:411-19.
- 8 Gaziano J, Buring J, Breslow J, et al. Moderate alcohol intake, increased levels of high-density lipoprotein and its subfractions, and decreased risk of myocardial infarction. *N Engl J Med* 1993;329:1829-34.
- 9 Klatsky AL, Friedman GD, Siegelau AB. Alcohol and mortality. A ten-year Kaiser-Permanente experience. *Ann Intern Med* 1981;95:139-45.
- 10 Marmot MG, Rose G, Shipley MJ, et al. Alcohol and mortality: a U-shaped curve. *Lancet* 1981;i:580-3.
- 11 Maskarinec G, Meng L, Kolone LN. Alcohol intake, body weight, and mortality in a multiethnic prospective cohort. *Epidemiology* 1998;9:654-61.
- 12 Rehm J, Semplos CT. Alcohol consumption and all-cause mortality. *Addiction* 1995;90:471-80.
- 13 Rimm E, Williams P, Fosher K, et al. Moderate alcohol intake and lower risk of coronary heart disease: meta-analysis of effects on lipids and haemostatic factors. *BMJ* 1999;319:1523-8.
- 14 Renaud S, Gueguen R, Schenker J, et al. Alcohol and mortality in middle-aged men from eastern France. *Epidemiology* 1998;9:184-8.
- 15 Thun MJ, Peto R, Lopez AD, et al. Alcohol consumption and mortality among middle-aged and elderly US adults. *N Engl J Med* 1997;337:1705-14.
- 16 Yano K, Rhoads GG, Kagan A. Coffee, alcohol and risk of coronary heart disease among Japanese men living in Hawaii. *N Engl J Med* 1977;297:405-9.
- 17 Lacoste I, Hung J, Lam JYT. Acute and delayed antithrombotic effects of alcohol in humans. *Am J Cardiol* 2001;87:82-5.
- 18 De Oliveira E, Silva ER, Foster D, et al. Alcohol consumption raises HDL cholesterol levels by increasing the transport rate of apolipoprotein A-I and A-II. *Circulation* 2000;102:2347-52.
- 19 Merriitt R, Guruge B, Miller D, et al. Moderate alcohol feeding attenuates postinjury vascular cell proliferation in rabbit angioplasty model. *J Cardiovascul Pharmacol* 1997;30:19-25.
- 20 Hayek T, Fuhrman B, Vaya J, et al. Reduced progression of atherosclerosis in apolipoprotein E-deficient mice following consumption of red wine, or its polyphenols quercetin or catechin, is associated with reduced susceptibility of LDL to oxidation and aggregation. *Arterioscler Thromb Vasc Biol* 1997;17:2744-52.
- 21 Hart C, Smith G, Hole D, 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.
- 22 Mortensen EL, Jensen HH, Sanders SA, et al. Better psychological functioning and higher social status may largely explain the apparent health benefits of wine: a study of wine and beer drinking in young Danish adults. *Arch Intern Med* 2001;161:1844-8.
- 23 Fuchs FD, Chambless LE, Folsom AR, et al. Association between alcoholic beverage consumption and incidence of coronary heart disease in whites and blacks. The atherosclerosis risk in communities (ARIC) Study. *Am J Epidemiol* 2004;160:466-74.
- 24 Eigenbrodt M, Fuchs F, Hutchinson R, et al. Health-associated changes in drinking: a period prevalence study of the atherosclerosis risk in communities (ARIC) cohort (1987). *Prev Med* 2000;31:81-9.
- 25 Shaper A, Wannamethee S. Alcohol intake and mortality in middle age men with diagnosed coronary heart disease. *Heart* 2000;83:394-9.
- 26 Sacco R, Elkind M, Boden-Albala B, et al. The protective effect of moderate alcohol consumption on ischemic stroke. *JAMA* 1999;281:53-60.
- 27 Emberson J, Shaper A, Wannamethee S, et al. Alcohol intake in middle age and risk of cardiovascular disease and mortality: accounting for intake variation over time. *Am J Epidemiol* 2005;161:856-63.
- 28 McGee DL, Liao Y, Cao G, et al. Self-reported health status and mortality in a multiethnic US cohort. *Am J Epidemiol* 1999;149:41-6.
- 29 Burstrom B, Fredlund P. Self rated health: Is it as good a predictor of subsequent mortality among adults in lower as well as in higher social classes? *J Epidemiol Community Health* 2001;55:836-40.
- 30 ARIC Investigators. The atherosclerosis risk in communities (ARIC) study: design and objectives. *Am J Epidemiol* 1989;129:687-702.
- 31 Wong TY, Klein R, Sharrett AR, et al. Retinal arteriolar narrowing and risk of diabetes mellitus in middle-aged persons. *JAMA* 2002;287:2528-33.
- 32 Klatsky AL, Armstrong MA, Friedman GD. Alcohol and mortality. *Ann Intern Med* 1992;117:646-54.
- 33 Adams WL, Garry PJ, Rhyne R, et al. Alcohol intake in the healthy elderly. Changes with age in a cross-sectional and longitudinal study. *J Am Geriatr Soc* 1990;38:211-16.
- 34 Dawson DA. Patterns of alcohol consumption: beverage effects on gender differences. *Addiction* 1993;88:133-8.
- 35 Herd D, Grube J. Black identity and drinking in the US: a national study. *Addiction* 1996;91:845-57.
- 36 Darrow SL, Russell M, Cooper ML, et al. Sociodemographic correlates of alcohol consumption among African-American and white women. *Women Health* 1992;18:35-51.
- 37 Wannamethee SG, Shaper AG. Alcohol, coronary heart disease and stroke: an examination of the J-shaped curve. *Neuroepidemiology* 1998;17:288-95.
- 38 Camacho TC, Kaplan GA, Cohen RD. Alcohol consumption and mortality in Alameda County. *J Chron Dis* 1987;40:229-36.
- 39 Anderson P. Alcohol consumption and all-cause mortality. *Addiction* 1995;90:481-4.
- 40 Mokdad AH, Marks JS, Stroup DF, et al. Actual causes of death in the United States, 2000. *JAMA* 2004;291:1238-45.
- 41 Liao Y, McGee DL, Cao G, et al. Alcohol intake and mortality: findings from the national health interview surveys (1988 and 1990). *Am J Epidemiol* 2000;151:651-9.
- 42 Corrao G, Rubbiati L, Bagnardi V, et al. Alcohol and coronary heart disease: a meta-analysis. *Addiction* 2000;95:1505-23.
- 43 Norstrom T. Per capita alcohol consumption and all-cause mortality in 14 European countries. *Addiction* 2001;96(suppl 1):S113-28.
- 44 Hemstrom O. Per capita alcohol consumption and ischemic heart disease mortality. *Addiction* 2001;96(suppl 1):S93-112.
- 45 Rothman KJ, Greenland S. Precision and validity in epidemiologic studies. In: Winters R, eds. *Modern epidemiology*. Philadelphia, PA: Lippincott-Raven, 1998:115-34.
- 46 Fillmore KM, Kerr WW, Bostrom A. Changes in drinking status, serious illness and mortality. *J Stud Alcohol* 2003;64:278-85.
- 47 Haveman-Nies A, de Groot LC, van Staveren WA. Relation of dietary quality, physical activity, and smoking habits to 10-year changes in health status in older Europeans in the SENECA Study. *Am J Public Health* 2003;93:318-23.
- 48 Wouters S, Marshall R, Yee RL, et al. Is the apparent cardioprotective effect of recent alcohol consumption due to confounding by prodromal symptoms. *Am J Epidemiol* 2000;151:1189-93.
- 49 Rehm J, Greenfield T, Rogers J. Average volume of alcohol consumption, patterns of drinking, and all-cause mortality: results from the US national alcohol survey. *Am J Epidemiol* 2001;153:64-71.
- 50 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.
- 51 Russell M, Welte JW, Barnes GM. Quantity-frequency measures of alcohol consumption: beverage-specific vs global questions. *Br J Addict* 1991;86:409-17.
- 52 Williams GD, Aitken SS, Malin H. Reliability of self-reported alcohol consumption in a general population survey. *J Stud Alcohol* 1985;46:223-7.
- 53 Davies MJ, Baer DJ, Judd JT, et al. Effects of moderate alcohol intake on fasting insulin and glucose concentrations and insulin sensitivity in postmenopausal women: a randomized controlled trial. *JAMA* 2002;287:2559-62.
- 54 Zilkens RR, Burke V, Hodgson JM, et al. Red wine and beer elevate blood pressure in normotensive men. *Hypertension* 2005;45:874-9.
- 55 Fuchs FD. Vascular effects of alcoholic beverages: is it only alcohol that matters? *Hypertension* 2005;45:851-2.