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Indonesia

The Indonesian House of Representatives is considering a bill that would ban the production, consumption or distribution of beverages that contain more than one percent alcohol throughout the country. In May, Indonesia passed a law blocking the sale of beer in convenience stores and other small neighborhood shops, but not supermarkets. Fears have been raised that if the current bill is passed, the local tourism industry could be adversely affected.

India

In India, the Cabinet approved amendments to the Motor Vehicle Bill on 3 August that propose hefty penalties for the violation of traffic norms. For drunk driving, the maximum penalty will rise from 2,000 to 10,000 Rupees and for a first offence, suspension of driving licence for three months. Repeat offenders could attract imprisonment and cancellation of their licence.

Ukraine

In the Ukraine, President Petro Poroshenko has signed a law that introduces more stringent penalties for drink driving, which increase with repeat offences. Offenders will be liable for a 10,200 hryvnia fine and a one-year driving license suspension, increasing to 20,400 hryvnia and a three-year suspension for a second offence, with a possibility of a 15-day custodial sentence. Offenders apprehended again up to four years after a second offense will be punished with a 40,800 hryvnia fine and a 10-year driving license suspension.

Cape Verde

The Government of Cape Verde has announced plans to reduce the legal blood alcohol concentration (BAC) limit from 0.8 mg/ml to 0.5 mg/ml. Ministry of Health National Public Health Director Maria da Luz Lima stated that the purpose of the measure is to decrease the serious health consequences attributed to harmful drinking. She added that alcohol dependence is deep-rooted in Cape Verden society, and asserted that the average age of first consumption for alcohol is “too early” and directly correlated to negative health impacts such as noncommunicable disease (NCDs) and mental illnesses.
Key findings on alcohol consumption and a variety of health outcomes from the Nurses’ Health Study

In a review published in American Journal of Public Health, Elizabeth Mostofsky, Kenneth J. Mukamal, Ed L. Giovannucci, Meir J. Stampfer, and Eric B. Rimm assess critical contributions from the Nurses’ Health Study (NHS) on alcohol consumption and health outcomes.

The research used data from phase one (1980-2012) and phase 2 (1989-2011) of the study with detailed information on self-reported alcohol drinking patterns obtained approximately every 4 years combined with extensive information on diet, lifestyle habits, and physician-diagnosed health conditions, NHS investigators have prospectively examined the risks and benefits associated with alcohol consumption.

Moderate intake, defined as up to 1 US drink (14g) a day, is associated with a lower risk of hypertension, myocardial infarction, stroke, sudden cardiac death, gallstones, cognitive decline, and all-cause mortality. However, even moderate intake places women at higher risk for breast cancer and bone fractures, and higher intake increases risk for colon polyps and colon cancer.

The research team concluded that regular alcohol intake has both risks and benefits. In analyses using repeated assessments of alcohol over time and deaths from all causes, women with low to moderate intake and regular frequency (> 3 days/week) had the lowest risk of mortality compared with abstainers and women who consumed substantially more than 1 drink per day.


The full text is available at ajph.aphapublications.org/doi/full/10.2105/AJPH.2016.303336

Alcohol intake and risk of thyroid cancer

A Korean study group undertook a meta-analysis of observational studies to assess whether alcohol intake is associated with the risk of thyroid cancer.

Researchers searched PubMed and EMBASE in June of 2015 to locate eligible studies. 33 observational studies with two cross-sectional studies, 20 case-control studies, and 11 cohort studies, which involved a total of 7,725 thyroid cancer patients and 3,113,679 participants without thyroid cancer in the final analysis.

In the fixed-effect model meta-analysis of all 33 studies, alcohol intake was consistently associated with a decreased risk of thyroid cancer (odds ratio 0.74, 95% confidence interval 0.67 to 0.83; I² = 38.6%). In the subgroup meta-analysis by type of study, alcohol intake also decreased the risk of thyroid cancer in both case-control studies (odds ratio 0.77, 95% confidence interval 0.65 to 0.92; I² = 29.5%; n =20) and cohort studies (relative risk 0.70, 95% confidence interval 0.60 to 0.82; I² = 0%; n = 11). Subgroup meta-analyses by type of thyroid cancer, gender, amount of alcohol consumed, and methodological quality of study showed that alcohol intake was significantly associated with a decreased risk of thyroid cancer.

The current meta-analysis of observational studies found that alcohol intake decreased the risk of thyroid cancer, the authors conclude.

Alcohol intake during middle age and later atherosclerosis


Authors’ Abstract

Background: Epidemiological evidence indicates a protective effect of light-moderate drinking on cardiovascular disease and an increased risk for heavier drinking. Nevertheless, the effect of alcohol on atherosclerotic changes in vessel walls is disputed. Most previous studies have only looked at the cross-sectional relationship between alcohol and carotid intima/media thickness (cIMT) – a surrogate marker of atherosclerosis. Single measurements of alcohol assume that alcohol exposure is stable and ignore the possible cumulative effects of harm, leading to possibly incorrect inferences.

Methods: Data were retrieved from two UK population based cohort studies: the Whitehall II cohort of civil servants and the MRC National Survey of Health and Development (combined sample size of 5403 men and women). Twenty-year drinking trajectories during midlife were linked to measures of cIMT when participants were in early old age, and adjusted for age, sex, socioeconomic position, ethnicity and smoking.

Results: Those who consistently drank heavily had an increased cIMT compared to stable moderate drinkers (pooled difference in cIMT 0.021 mm; 95 % CI 0.002 to 0.039), after adjustment for covariates. This was not detected in cross-sectional analyses. Former drinkers also had an increased cIMT compared to moderate drinkers (pooled difference in cIMT 0.021; 95 % CI 0.005 to 0.037). There were no appreciable differences in cIMT between non-drinkers and consistent moderate drinkers.

Conclusion: The drinking habits among adults during midlife affect the atherosclerotic process and sustained heavy drinking is associated with an increased cIMT compared to stable moderate drinkers. This finding was not seen when only using cross-sectional analyses, thus highlighting the importance of taking a life course approach. There was no evidence of a favourable atherosclerotic profile from stable moderate drinking compared to stable non-drinking.

Forum Comments

Carotid artery disease can be estimated from the thickness of the wall of the arteries measured with ultrasound (recorded as carotid artery intima/media thickness, cIMT), and by evidence of atherosclerotic plaques within the carotid arteries. The association between alcohol intake and ‘carotid lesions’ is unclear, as some studies show a positive association with cIMT and/or plaques while others show no association. Given that carotid disease relates to the subsequent risk of coronary artery disease, there is increasing use of carotid ultrasound measurements to help determine long-term risk of cardiovascular disease, especially among subjects with intermediate levels of risk as determined by usual risk factor assessment.

The present study provides valuable information by reporting the cross-sectional relation between alcohol consumption and cIMT as well as how the drinking pattern over 20 years in middle age may relate to cIMT later in life. Specifically, it combines data from two cohort studies (the Whitehall II cohort of civil servants and the MRC National Survey of Health and Development) each of which had repeated assessments of alcohol intake during middle age. The investigators were able to evaluate the effect on cIMT at the time of alcohol assessment (at age 50-74 in the Whitehall study and age 60-64 in the MRC study) as well as the effect on cIMT of the pattern of drinking over earlier periods in life. Thus, the investigators were able to report both cross-sectional associations between alcohol and cIMT and a trajectory of the pattern (always none, always moderate, always heavy, etc.) for estimating the effect of alcohol intake on cIMT. The analyses are well done and appropriate.

Specific comments on the study by Forum members: Reviewer Puddey carried out an extensive review of the methodology of this study: “This paper utilises a meta-analytic approach to 2 prospective British studies to link 20 year drinking pattern trajectories, characterised as either ‘stable heavy’ drinkers or ‘former drinkers’, to a finding of increased carotid artery IMT. In addition, a ‘mostly moderate’ 20 yr drinking trajectory was not linked to either anti-atherogenic or pro-atherogenic effects. These findings are of interest given the ongoing controversy with respect to potential anti-atherogenic and pro-atherogenic effects of alcohol consumption and the relative paucity of literature that has taken long term patterns of consumption into account.

“The meta-analysis utilises the difference from median IMT for each of the 2 cohorts as the dependent variable. Were median IMT and IMT distributions similar in the 2 cohorts? The reported values look significantly lower and with much wider
Further, consumption with meals provides additional disease apparent among regular moderate drinkers. It is clear that subjects who binge drink of drinking could not be assessed in the present analyses. It is possible that no significant differences would have been seen.

“The multivariate model has not included a measure of adiposity, and BMI is not listed in the table of characteristics of participants. BMI has been a predictor of carotid IMT in previous studies and could be a confounder of the reported 20 yr alcohol drinking pattern trajectory association with IMT. Further, increased BMI is associated with impaired glucose tolerance and it has been previously reported that there is an interaction between alcohol intake and glucose tolerance, with alcohol predictive of reduced carotid IMT in those with normal glucose tolerance but increased carotid IMT in those with impaired glucose tolerance (Cooper et al). A long term pattern of heavy drinking has been linked to not just increased prevalence of smoking and ex-smoking but also to poorer dietary choices and less physical activity. It has also been linked to type of alcoholic beverage (spirits vs beer or wine) which hasn’t been measured in the current study. The study has therefore not convincingly demonstrated that unmeasured confounding could have influenced the final result.”

Puddey concluded: “Several previous studies have indicated a potentially favourable association of increasing alcohol intake with increased carotid artery lumen diameter, despite simultaneously demonstrating a link to increased IMT. We do not know if carotid artery lumen diameter was measured in the current study and we are also not told whether carotid IMT was related to incidence of cerebrovascular disease events, an outcome which has been previously measured in the Whitehall study.”

Reviewer Stockley pointed out that the pattern of drinking could not be assessed in the present analyses. It is clear that subjects who binge drink do not get the protection against cardiovascular disease apparent among regular moderate drinkers. Further, consumption with meals provides additional protection, as shown in a clinical trial by Hendriks et al. That trial revealed that moderate alcohol consumption with dinner affects plasminogen activator inhibitor activity, plasminogen activator antigen level, and tissue type plasminogen activator activity temporarily, all of which lower cardiovascular risk through favorable effects on fibrinolysis.”

Forum member Finkel noted: “I am never sanguine about studies that depend on avatars; it would be more helpful to determine the frequency of cardiovascular events over time. There are many potential confounding variables (obesity, diet, physical activity, etc.) not taken into consideration in these analyses. How do we know that the group with thick carotids hadn’t spent most of their time away from the office at their bowling alley drinking ale and eating crisps?”

Does cIMT predict future cardiovascular disease? In 1997, Bots et al reported from the Rotterdam Study that “an increased common carotid IMT is associated with future cerebrovascular and cardiovascular events.” In 1999, O’Leary et al reported that “Increases in the thickness of the intima and media of the carotid artery, as measured noninvasively by ultrasonography, are directly associated with an increased risk of myocardial infarction and stroke in older adults without a history of cardiovascular disease.” Numerous studies since these have generally shown that cIMT is a risk factor for future disease. But there are still divided opinions as to whether carotid artery ultrasound studies should be offered to all patients in a clinical practice.

Measuring both cIMT and carotid atherosclerotic plaques: Previous studies have evaluated not only the carotid wall thickness (cIMT) but identified atherosclerotic plaques within the carotid arteries. The present study focuses only on the cIMT, and does not comment on the presence or absence of atherosclerotic plaques. Forum reviewer Ellison states: “Previous studies from our group have found no relation between alcohol intake and IMT or the presence or absence of atherosclerotic lesions [Djousse et al (a)]; other such studies have also shown a lack of effect on cIMT from albumin levels [Djousse et al (b)] or from measures of hostility or social support (Knox et al). However, both carotid atherosclerotic plaques and cIMT levels have been found to relate inversely to the intake of linolenic acid [Djousse et al (c)] and positively (at
least in men) to serum uric acid levels (Neogi et al). In any case, it would seem prudent that both cIMT and the presence or absence of atherosclerotic plaques within the carotid arteries be evaluated in estimating later cardiovascular risk.”

Alcohol intake, atherosclerosis and coagulopathy as risk factors for myocardial infarction (MI): Acute MI is known to relate especially to the rupture of an atherosclerotic plaque and subsequent clot formation. The degree of atherosclerosis of a coronary vessel is not a particular good predictor of whether or not a MI will occur from obstruction of that artery. Data indicate that the effects of wine and other alcoholic beverages on coagulation may be more relevant for the development of MI than from its effects on atherosclerosis. For example, Renaud proposed the hypothesis of a hemostatic mechanism, rather than an interaction with the atherosclerosis process, as a key factor for this paradox (Renaud & de Lorgeril).

Wine and alcohol intake have repeatedly been shown to relate to improvements in blood clotting, fibrinolysis, and other aspects of coagulation. Thus, any study evaluating alcohol intake for predicting the risk of MI must evaluate not only the development of atherosclerosis but also the effects on coagulopathy. While this study did not show that moderate drinking results in less thickness of the carotid arteries later in life, most studies show that such drinking is associated with a lower risk of MI, stroke, and total mortality. If indeed cIMT reflects coronary disease, the results of this study suggest that the protection against MI generally seen among moderate drinkers may be due more to improvements in coagulation than it is to the degree of atherosclerosis.

Forum member De Gaetano commented: “This is an interesting paper indeed. The apparent discrepancy between their results (lack of effect on cIMT later in life) and the well-known preventive effect of moderate alcohol consumption on fatal and non-fatal cardiovascular clinical events suggests that atherosclerosis is a benign disease unless thrombosis (activation of platelets and coagulation) occurs on an atherosclerotic vessel wall. Together with Ramon Estruch we showed, years ago, that wine polyphenols did not modify the lipid pattern of healthy volunteers, but inhibited several plasma and cell functions related to inflammation and ischemic disease (Badia et al, Estruch et al). The data on heavy or moderate drinkers on cIMT might suggest that both abstainers and moderate drinkers have a very slow progression of atherosclerosis while heavy drinking accelerates it. If moderate drinking is not preventive, heavy drinking is promoting the atherosclerotic process.”

Reviewer Waterhouse noted: “This study seems to confirm that the mechanism for the protective effect of moderate alcohol consumption is not so much related to preventing atherogenesis, but due primarily to its effects on coagulopathy. This is a very useful distinction for future investigations. If this is the case, it also suggests another line of investigation, and that is, how quickly is one’s risk of cardiovascular disease reduced after starting moderate alcohol consumption?”

Reviewer Skovenborg agreed with other reviewers that carotid artery wall thickness is a proxy variable with dubious value for the future risk of stroke. He adds: “Even so, recent data indicate that light to moderate alcohol consumption is associated with lower atherosclerotic burden in the proximal aortic arch (Kohsaka et al). Further, regarding the proposition that the largest effect of alcohol on cardiovascular disease is from its effects on coagulopathy, two conditions are of interest as they are associated with atherosclerosis and not with coagulopathy: stable angina pectoris and peripheral arterial disease (PAD). The Physicians’ Health Study demonstrated that moderate drinking decreases the risk of angina pectoris (RR 0.69, 95% CI 0.59-0.81) and myocardial infarction (RR 0.65, 95% CI 0.52-0.81) and also that, after control for confounding by smoking, moderate alcohol consumption decreases the risk of PAD (RR 0.74, 95% CI 0.57-0.97) [Camargo CA et al (a); Camargo et al (b)].”

Forum member Lanzmann-Petithory commented: “I agree that the role of coagulation rather than atherosclerosis is most important in the protection against cardiovascular disease for wine drinkers. The ‘heavy drinkers’ in this study, including binge drinkers, are probably not wine drinkers and are likely to have higher blood pressure. And blood pressure is directly related to cIMT, as shown by numerous studies (e.g., Ferreira et al). The positive relation of heavy drinking with cIMT may be reflecting partially the effect of higher blood pressure in binge drinkers, especially of beer and spirits.”

Overall, current scientific data provide strong evidence that moderate alcohol consumption
lowers the risk of essentially all manifestations of cardiovascular disease; it appears to work through a combination of effects on lipids, inflammation, coagulation, fibrinolysis, glucose metabolism, and other paths. In most studies it decreases the risk of the development and progression of atherosclerosis, but also helps prevent clot formation within arteries, often the precipitating event for an acute cardiovascular event.

Is carotid artery screening appropriate for most patients in clinical practice? Robertson et al stated that “It is increasingly recognised that in terms of assessing disease burden and risk of subsequent vascular events, both IMT and plaque should be considered separately. Carotid plaque itself has been shown to be associated with concomitant CVD, and indeed is considered to be an indicator of high vascular risk (as shown by Ebrahim et al).” The value of adding cIMT to already available conventional risk factors in predicting future disease remains unclear. As Robertson et al concluded: “Many large epidemiological studies have shown a strong relationship between IMT and incident CVD, but the evidence for the use of cIMT in clinical practice is incomplete. Current evidence suggests that for people at intermediate risk according to Framingham risk scoring, IMT may add useful information on vascular risk, but findings are inconsistent between studies and, in some cases, the improvement in classification is modest.”

References from Forum critique


Forum Summary

Carotid artery disease can be estimated by ultrasound from the thickness of the wall of the arteries (recorded as carotid artery intima/medial thickness, cIMT) and by evidence of atherosclerotic plaques within the carotid arteries. The association between alcohol intake and such lesions is unclear, as some studies show a positive association with cIMT and/or plaques while others show no association. Given that carotid disease relates to the subsequent risk of coronary artery disease, there is increasing use of carotid ultrasound measurements to help determine long-term risk.

The present large study from the UK provides valuable information by reporting the cross-sectional relation between alcohol consumption and cIMT as well as how the drinking pattern over 20 years in middle age may relate to cIMT later in life. While heavy alcohol intake was found to increase later cIMT measures, no clear differences were noted between subjects reporting abstinence and those reporting moderate drinking in middle age.

Forum reviewers considered this to be a well-done study. There were some concerns that only total average alcohol intake was considered, as there were no data on the type of beverage consumed, the pattern of drinking (binge versus regular moderate), whether the alcohol was consumed with food, etc., and there was an absence of data on diet, physical activity, adiposity, and other factors related to atherosclerosis. Further, only the thickness of the carotid artery was evaluated, and not the presence or absence of atherosclerotic plaques on the ultrasound readings.

Overall, current scientific data provide strong evidence that moderate alcohol consumption lowers the risk of most manifestations of cardiovascular disease; it appears to work through a combination of effects on lipids, inflammation, coagulation, fibrinolysis, glucose metabolism, and other paths. Further, heavy drinking is known to increase blood pressure, and is an important factor for developing hypertension (and a strong determinant of cIMT). The failure of the present study to find a significant association between moderate alcohol intake and later carotid thickness supports what has been shown in some, but not all, previous studies.

If indeed chronic alcohol use has little effect on atherosclerosis (using images from the carotids as an index of atherosclerosis in the coronary arteries and elsewhere in the body), it may indicate that the mechanisms for the protective effect of moderate alcohol consumption on cardiovascular disease are not so much related to preventing atherogenesis, but due primarily to the effects on coagulopathy. And it is known that clot formation within the arterial wall is often the precipitating event for an acute myocardial infarction or other cardiovascular event.

Comments for this critique by the International Scientific Forum on Alcohol Research were provided by the following members:

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Beer drinking associates with lower burden of amyloid beta aggregation in the brain

A study investigated the association between consumption of different alcoholic beverages and β-amyloid (Aβ) aggregation in the brain, one of the neuropathological lesions of Alzheimer’s disease.

The study included 125 males of the Helsinki Sudden Death autopsy series with an age at death ranging from 35 to 70 years. The consumption of alcohol, Aβ aggregation in the brain, and Apolipoprotein E (APOE) genotype were assessed. Relatives answered a questionnaire to gather alcohol consumption history, and Aβ was visualised by implementing immunohistochemical staining of brain sections. Aβ immunoreactivity (IR) was assessed in a dichotomised (yes/no) fashion and as a stained area fraction (%). APOE genotype was assessed in DNA extracted from paraffin-embedded cardiac muscle samples.

Increased age (p = 0.001; odds ratio [OR] = 1.09, confidence interval [CI] = 1.04 to 1.15) was associated with higher prevalence of Aβ-IR. Beer drinking decreased (p = 0.024; OR = 0.35, CI = 0.14 to 0.87) the prevalence of Aβ-IR and was associated with a significantly lower extent of Aβ-IR (p = 0.022). The amount of alcohol consumed was not linked with Aβ aggregation and neither was spirit nor wine consumption.

Beer consumption may protect against Aβ aggregation in brain, the authors suggest. Further studies are necessary to fully understand the effects of alcohol on Aβ pathology seen in brain tissue.


Influence of the concentrate of red wine polyphenols on glutamate neurotoxicity

An article originally published in Russian tells of research to assess the effect of a concentrate of red table wine Saperavi on the cultivated nerve cells exposed to glutamate. Saperavi was selected as the source of phenolic compounds because the type of grapes in the Krasnodar region from which it derives has the highest content of them - up to 4-5 g/dm3 or more.

Polyphenol concentrate was prepared. It was established that a large group of phenolic compounds with high antioxidant activity was present in the concentrate of polyphenols: procyanidins (total concentration up to 425 mg/dm3), quercetin (21.8-32.6 mg/dm3), gallic acid (124.2-164.7 mg/dm3), resveratrol (6.26-13.22 mg/dm3), catechins (1026 - 1480 mg/dm3).

The effect of Saperavi red wine concentrate on glutamate cytotoxicity was studied in the neuron culture of the cerebellum of 7-9-day-old rats. It was shown that the presence of antioxidants reduced the intensity of chemiluminescence in model systems that generate free radicals. It was established that quenching of chemiluminescence in the system <<citrate-phosphate-luminol>> composed 68.43%, and in the system of the yolk lipoproteins - 86.36%. The application of concentrate Saperavi significantly increased the survivability of neurons: at the doses 5, 10 and 30 mcg/ml the number of intact neurons was respectively 38.6; 41.5 and 37.1%. The dose of 20 mcg/ml was the most effective - the proportion of live neurons comprised 47.4%.

The authors state that the obtained results can be explained by the high antioxidant activity of concentrate flavonoids, including high content of biologically active compounds - catechins, quercetin, rutin, resveratrol. Thus, the consumption of red wine in quantities that exclude harmful effects, can have a positive impact on human health and the brain in particular.

A new study on the mechanisms of alcohol’s effects on health and disease


Authors’ Abstract

Background: High alcohol consumption is a major cause of morbidity, yet alcohol is associated with both favourable and adverse effects on cardiometabolic risk markers. We aimed to characterise the associations of usual alcohol consumption with a comprehensive systemic metabolite profile in young adults.

Methods: Cross-sectional associations of alcohol intake with 86 metabolic measures were assessed for 9778 individuals from three population-based cohorts from Finland (age 24–45 years, 52% women). Metabolic changes associated with change in alcohol intake during 6-year follow-up were further examined for 1466 individuals. Alcohol intake was assessed by questionnaires. Circulating lipids, fatty acids and metabolites were quantified by high-throughput nuclear magnetic resonance metabolomics and biochemical assays.

Results: Increased alcohol intake was associated with cardiometabolic risk markers across multiple metabolic pathways, including higher lipid concentrations in HDL subclasses and smaller LDL particle size, increased proportions of monounsaturated fatty acids and decreased proportion of omega-6 fatty acids, lower concentrations of glutamine and citrate (P<0.001 for 56 metabolic measures). Many metabolic biomarkers displayed U-shaped associations with alcohol consumption. Results were coherent for men and women, consistent across the three cohorts and similar if adjusting for body mass index, smoking and physical activity. The metabolic changes accompanying change in alcohol intake during follow-up resembled the cross-sectional association pattern (R²=0.83, slope=0.7260.04).

Conclusions: Alcohol consumption is associated with a complex metabolic signature, including aberrations in multiple biomarkers for elevated cardiometabolic risk. The metabolic signature tracks with long-term changes in alcohol consumption. These results elucidate the double-edged effects of alcohol on cardiovascular risk.

Forum Comments

Observational epidemiologic studies relating alcohol consumption to health and disease have been amazingly consistent over many decades: light to moderate alcohol intake is related to improved cardiovascular health and less diabetes, while heavy intake and binge drinking relate primarily to adverse cardiovascular and other disease outcomes. A multitude of animal experiments have identified many mechanisms by which moderate drinking affects cardiovascular disease, including beneficial effects on lipids, coagulation factors, fibrinolysis, glucose metabolism, inflammation, and endothelial function. The mechanisms for a direct effect of heavy drinking on upper airway cancers and liver disease have also been delineated, and suggested mechanisms given for the slight increase in breast cancer risk from even moderate drinking seen in most epidemiologic studies. However, the specific metabolic effects of alcohol have been little studied. The investigators for the present study have collected a large amount of metabolic data from population-based cohorts of relatively young and healthy men and women in Finland. Their analysis provides a wealth of information by relating such data to the reported alcohol intake of subjects.

Contributions of the present study: For the present study, data from a group of almost 10,000 young adults from population-based cohorts in Finland have been evaluated; the investigators have measured associations between self-reported alcohol consumption and a large number of lipid parameters, indices of fat intake, glutamine, citrate, and 56 metabolic measures. The investigators found, as expected, that total HDL-cholesterol increases linearly with reported alcohol intake. In addition, they found that alcohol intake was associated with higher lipid concentrations in HDL subclasses and smaller LDL particle size, increased proportions of monounsaturated fatty acids and decreased proportion of omega-6 fatty acids, lower concentrations of glutamine and citrate (P<0.001 for 56 metabolic measures). Many metabolic biomarkers displayed U-shaped associations with alcohol consumption. The findings of this study provide valuable clues to the biologic effects on health, both favorable and adverse, related to alcohol consumption. Further, the paper could also provide an interesting new approach for estimating the level of alcohol intake of individuals in epidemiologic studies, so that alcohol intake is judged not just on what the individual subject self-reports.

Improving our understanding of alcohol’s effects on health: The authors have summarised very well the potential importance of these analyses: “The detailed metabolic phenotyping further clarified
the association shapes for numerous established biomarkers related to alcohol and cardiometabolic risk. The metabolic signature of alcohol consumption included molecular perturbations linked with both higher and lower cardiovascular risk. Many metabolic measures displayed an optimum level at modest alcohol intake.” They conclude: “Comprehensive metabolic profiling in these large cohorts elucidated the metabolic influence of alcohol consumption and clarified the double-edged relation between alcohol and cardiometabolic biomarkers.”

Forum member Barrett-Connor commented: “I think this is an excellent paper, based on data from three different population-based cohorts for whom outcome data are available (the great advantage of having personal identifiers in a country). It is a meta-analysis and includes both cross-sectional and, for a subset, longitudinal data that further validates their reported alcohol intake. Associations are reported between alcohol intake and lipids, circulating fatty acid levels, and many other metabolites. The authors note that the dietary determinants of the circulating biomarkers (especially amino acids and other small molecules) remain poorly understood. The most prominent associations were for citrate and glutamine—both strongly inversely associated with alcohol but otherwise weakly correlated with established CVD risk factors.”

Reviewer Finkel was also pleased by this paper. “I particularly note the youth of the subjects, which brings to mind one of the old-time tenets of drinking’s oft-expressed health benefits: drinking moderately was usually said to be associated with cardiovascular and survival benefits only for middle-aged and older people, whereas this paper shows strong relations between alcohol and biomarkers among the young. Atherosclerosis is a chronic, slowly building disease: its clinical appearance in middle or old age surely means it started long before.”

Forum member Ursini was less impressed by these analyses. “As a basic scientist, interested on mechanisms and metabolism, I must admit I did not learn too much. Are we going to discover new biomarkers? Citrate and glutamate will become the new cholesterol? This type of well-organised fishing expedition without an educated working hypothesis will never solve relevant medical issues unless validated by a severe experimental approach.”

Reviewer Thelle noted: “I also am somewhat skeptical of the cross-sectional aspects of this study, while admitting that some such studies have provided the basis for new hypotheses. And that is the major contribution from this paper; it ignites the interest for experimental studies, and of course the inclusion of new variables in prospective studies.”

Reviewer Skovenborg tended to agree with the views of Ursini and Thelle, stating “Results from this cross-sectional study may best be used to generate new hypotheses regarding cardio-vascular risk factors and maybe candidates for laboratory confirmation of data on alcohol intake. I question whether the results from these Finnish populations – from a country with binge drinking traditions – are generalisable to populations with a Mediterranean drinking pattern. Also, I am not clear as to why participants with alcohol consumption above 500 g/week were excluded due to the likelihood that these intake volumes were spurious. Most literature regarding people with heavy alcohol consumption would not consider 6-7 drinks/day as improbable.”

Forum member Lanzmann-Petithory had some cogent comments on the lipid changes noted in this study. “Reading this paper I was struck by the fatty acid profiles in plasma in the cross-sectional association with alcohol. The pattern with alcohol shown in the present study is very similar to that from the intervention group given canola oil in the Lyon Diet Heart study (de Lorgeril et al; Renaud et al), i.e., increased MUFA and decreased linoleic acid. The Lyon study showed a large degree of cardiovascular protection from more mono-unsaturated, more omega 3 fatty acids and less linoleic acid. It is unclear why the essential fatty acid profile should be the same in Finnish drinkers: could there be a metabolic interaction of alcohol with diet or a confounding with diet?

“We know that the diet in Finland has turned towards a Mediterranean-type diet from the beginning of the North Karelia Project in 1972; rapeseed oil has become widely used, including in the margarine industry (Pietinen et al). During recent decades, there has also been a rapid decline in coronary heart disease in Finland that remains unexplained by classical risk factor correction (Laatikainen et al). The group led by Federico Leighton in Chile carried out clinical trials investigating the effect of wine on plasma fatty acids; they found that wine improved fatty
acid profile in subjects following a Mediterranean diet but not in subjects with more of an Occidental one (Urquiaga et al). Furthermore, we know from statistics of IREB that Finland turned more and more towards wine consumption: between 1961 and 2001, the consumption of pure alcohol soared from 2 liters to 7.4 liters /capita/year, and wine consumption between 1990 and 2001 from 6.4 liters to 20.1 liters/capita/year. Still, we do not know why the fatty acid profile associated with alcohol intake in Finland was so similar to that shown in France from increased canola oil. It deserves more investigation.”

Can the data in this study be used to help provide an accurate assessment of alcohol consumption of individuals? In addition to providing information on potential mechanisms by which alcoholic beverages affect health, reviewer Ellison has brought up another potential use of these data: can they be used to help scientists get a better estimate of the alcohol intake of individuals? “In almost all observational studies, estimates of the alcohol intake of individuals come from self-report, from food diaries or specific questions regarding beverages consumed within the preceding days, weeks, or years. Given that it is generally assumed that some people under-estimate their intake, the ability to obtain a more objective measure could be very helpful in seeking to relate alcohol intake to health and disease.

“Klatsky and colleagues (2006, 2014) have described a method for using multiple medical records from individuals to help identify those who are, or are not, under-reporting their alcohol intake (those deemed likely to be under-reporters showing more adverse effects of moderate drinking). Further, other investigators (Boniface et al, Livingston et al) have described common characteristics of subjects who tend to under-report their intake. Such approaches should improve epidemiologic studies by providing better assessments of actual alcohol intake. Another approach for estimating intake involves the relation of genetic factors that affect alcohol consumption to disease outcomes, using what is known as Mendelian randomisation technique to find a genetic surrogate measure of intake (Smith & Ebrahim). Unfortunately, there are currently not available simple genetic factors that are specific enough to serve as an accurate estimate of intake, and the limited Mendelian studies thus far reported provide incomplete information on alcohol effects on health and disease (e.g., Holmes et al, which was reviewed by our Forum; available at www.bu.edu/alcohol-forum/critique-143). Some Forum members wondered if a set of lipids, metabolites, etc., shown in this study to relate strongly to self-reported intake, could be used to help determine the accuracy of self-reported drinking.”

Forum member Klatsky reviewed the present paper and commented on this potential use of the results of this study: “Although my knowledge of it is limited, my impression from the considerable literature about the use of biomarkers to estimate alcohol intake in individuals is that estimation of recent intake is pretty good, but chronic intake is another matter. I’m also sure that the fact of under-estimation (“yes” or “no”) in an individual that claimed abstinence can quite accurately be determined by just a few tests, or even one. In terms of screening for epidemiological surveys, one would be limited to the available data. I know that clinicians in Kaiser Permanente that evaluated examinees undergoing multiphasic screening felt that, in the absence of other explanations, the triad of high HDL, elevated AST, and high MCV was a highly specific marker for alcoholism. Sensitivity was presumably much lower. But even these 3 tests were not consistently available over the years when screening was widely performed.”

Klatsky continued: “We used available AST and ALT values, available in a subgroup of examinees, to validate our suspected under-reporter concept in our study of that phenomenon, but didn’t use labs as part of the definition. So I think your concept is sound, but wonder whether there are available cohorts that have the relevant data. Also, how does one validate the fact of under-reporting?”

Reviewer Zhang commented that “While many such endeavors are for dichotomous outcomes (i.e., yes or no for risk of a specific disease), others have attempted to determine the predictive ability for continuous outcome variables (e.g., Ellison et al). Thus, it is certainly doable in theory to use these metabolites to predict alcohol consumption; however I am not sure what the purpose is of doing it. If the current ‘gold standard’ of alcohol consumption is self-reported intake (and this paper is using that to relate to metabolic outcomes), why not just obtain our estimates through questionnaire; why should we bother to get these biomarkers and use them to predict alcohol consumption? Among non-drinkers,
it is likely that distribution of these metabolites may vary. Sometimes, X may be a good predictor for Y, but that does not mean that Y will be a good predictor for X. In other words, while self-reported alcohol consumption (which is susceptible to misclassification bias) is associated with levels of these metabolites, it does not mean that these metabolites would predict self-reported alcohol consumption. Other factors, such as dietary pattern, may affect metabolites as well; in fact, there is a strong association between smoking and alcohol intake."

Reviewer Ellison argued: “Any additional data on potential biases for self-reported data, whether based on genetic, behavioural, laboratory, or other information (preferably on all), might allow epidemiologists to have more precise and accurate information on alcohol intake when relating such to disease outcomes. As noted, Klatsky et al previously showed that among ‘moderate drinkers,’ data from those deemed unlikely to be under-reporters showed no significant effect of alcohol on blood pressure or risk of cancer; those deemed to be under-reporters showed adverse effects of their supposedly ‘moderate drinking’ on these outcomes. Indeed, a more accurate assessment of exposure could benefit our analyses.” Added reviewer Thelle: “What comes to my mind is an extensive alcohol experiment taking into consideration genetic variation, and assessing the effects on all the variables mentioned in the paper (plus some others as well). Feasibility and ethics may be questionable, but still, the idea deserves to be mentioned.”

References in Forum critique


Forum Summary
While observational epidemiologic studies for many decades have consistently shown that moderate drinkers have a lower risk of cardiovascular diseases than do non-drinkers or heavy drinkers, the specific metabolic effects of alcohol have been little studied. In the present analysis, among almost 10,000 young adults from three population-based cohorts in Finland, associations of alcohol intake with 86 metabolic measures were assessed. Circulating lipids, fatty acids and metabolites were quantified by high-throughput nuclear magnetic resonance metabolomics and biochemical assays. The investigators found that alcohol consumption was associated with a complex metabolic signature, including aberrations in multiple biomarkers for reduced as well as elevated cardiometabolic risk; many factors showed different associations according to the estimated amount of alcohol consumed.

Among key associations found for greater alcohol intake were increases in HDL-cholesterol and its
subclasses, decreases in LDL size, an increase in monounsaturated fatty acids and a decrease in omega-6 fatty acids, and lower concentrations of glutamine and citrate. For unexplained reasons, the changes in fatty acids from alcohol in this study were similar to those occurring following the administration of canola oil in other research. Some Forum reviewers pointed out that the analyses were not theory driven, and should only be used to generate hypotheses that would need to be tested in future experiments. As stated by one reviewer, the major contribution from this paper could be that it ignites the interest for experimental studies and provides new variables to be evaluated in prospective studies. In any case, the findings of this study provide valuable clues to the biologic effects on health, both favorable and adverse, related to alcohol consumption.

While not discussed by the authors, Forum members considered that the results of this paper might also be useful in providing a new approach for judging the level of alcohol intake of individuals in epidemiologic studies. At present, alcohol intake is judged almost exclusively from self-reports by subjects. Previous attempts designed to identify subjects more likely to be under-reporting their intake have shown that they may sharpen relations between estimated intake and health outcomes. Forum members believe that approaches that identify sources of bias for self-reported data, whether based on genetic, behavioural, physiological, or other information (preferably on all), might allow epidemiologists to have more precise and accurate information on alcohol intake when relating such to disease outcomes. A new approach for providing more accurate and unbiased estimates of alcohol intake is suggested by this excellent analysis.

The effects of alcohol on other chronic liver diseases

Alcohol consumption is often a comorbid condition in other chronic liver diseases. It has been shown to act in synergy to increase liver injury in viral hepatitis, hereditary hemochromatosis, and nonalcoholic fatty liver disease (NAFLD), leading to an increased risk of cirrhosis, hepatocellular carcinoma, and liver-related mortality. Data suggest that modest alcohol consumption may be inversely related to the risk of developing NAFLD and lower rates of progression of NAFLD to nonalcoholic steatohepatitis (NASH). This article reviews data on the relationship between alcohol consumption and other chronic liver diseases.

C-reactive protein level partially mediates the relationship between moderate alcohol use and frailty

Frailty is an indicator of late-life decline marked by higher rates of disability and healthcare utilisation. Research has linked health benefits with moderate alcohol use, including frailty risk reduction. Past work suggests inflammation, measured by C-reactive protein (CRP), as one candidate mechanism for this effect.

This study examined a possible mechanism - CRP modulation - by which moderate alcohol consumption might protect against frailty.

The cross-sectional study used data from the 2008 wave of the Health and Retirement Study (HRS) conducted by the University of Michigan. The HRS is a cohort study on health, retirement and aging on adults aged 50 and older living in the USA. A final sample of 3,229 stroke-free participants, over the age of 65 years and with complete data, was identified from the 2008 wave. Alcohol use was measured via self-report. Frailty was measured using the Paulson–Lichtenberg Frailty Index. CRP was collected through the HRS protocol.

Results from structural equation modelling support the hypothesised model that moderate alcohol use is associated with less frailty and lower CRP levels. Furthermore, the indirect relationship from moderate alcohol use to frailty through CRP was statistically significant.

The authors say their findings suggest that inflammation measured by CRP is one mechanism by which moderate alcohol use may confer protective effects for frailty. These findings inform future research relating alcohol use and frailty, and suggest inflammation as a possible mechanism in the relationship between moderate alcohol use and other beneficial health outcomes.

Source: C-reactive protein level partially mediates the relationship between moderate alcohol use and frailty. Shah M; Paulson D. Age and Ageing. Published early online 4 July 2016.

Alcohol intake, drinking patterns, and prostate cancer risk and mortality

Epidemiologic evidence for an association between alcohol intake and prostate cancer has been inconsistent. Researchers prospectively investigated the association between midlife alcohol intake and drinking patterns with future prostate cancer risk and mortality in a population-based cohort of Finnish twins.

Data were drawn from the Older Finnish Twin Cohort in which 11,372 twins were followed from 1981 to 2012. Alcohol consumption was assessed by questionnaires administered at two time points over follow-up. Over the study period, 601 incident cases of prostate cancer and 110 deaths from prostate cancer occurred. Cox regression was used to evaluate associations between weekly alcohol intake and binge drinking patterns with prostate cancer risk and prostate cancer-specific mortality. Within-pair co-twin analyses were performed to control for potential confounding by shared genetic and early environmental factors.

Compared to light drinkers ( < / = 3 drinks/week; non-abstainers), heavy drinkers (> 14 drinks/week) were at a 1.46-fold higher risk (HR 1.46; 95 % CI 1.12, 1.91) of prostate cancer. Among current drinkers, binge drinkers were at a significantly increased risk of prostate cancer (HR 1.28; 95 % CI 1.06, 1.55) compared to non-binge drinkers. Abstainers were at a higher risk (HR 1.90; 95 % CI 1.04, 3.47) of prostate cancer-specific mortality compared to light drinkers, but no other significant associations for mortality were found. Co-twin analyses suggested that alcohol consumption may be associated with prostate cancer risk independent of early environmental and genetic factors.

Heavy regular alcohol consumption and binge drinking patterns may be associated with increased prostate cancer risk, while abstinence may be associated with increased risk of prostate cancer-specific mortality compared to light alcohol consumption, the authors conclude.

Comment On Alcohol and Cancer by Byron Sharp, Professor of Marketing Science at the University of South Australia

Does alcohol cause cancer? Can Good Intentions Corrupt Science?

Recently an article by a Kiwi Professor gained sensational newspaper coverage along the lines of “proof alcohol causes cancer”. I’m very interested in the corruption and abuse of science. Good intentions, moral vanity, together with a single source of funding are a strong cause of bias. That's what it looks like here*, most likely a case of either seeing what you want to see in the evidence, or perhaps deliberating misusing it to make a political point (‘the ends justifies the means’?).

The scientific evidence suggests alcohol might cause a few cancers, though these are rare and the elevated risk of death is tiny. It’s wrong to misrepresent this evidence, and unethical to scare the public.

I'm going to show how Professor Jennie Connor’s case is incomplete and a bit shoddy, in that she misses out important studies and misrepresents others. She even misses out evidence that helps part of her argument.

Her article isn't a new study, there is no new data, nor data analysis. It was published in the journal’s “For Debate” section. Its purpose is to examine whether it might be reasonable to consider the (weak) correlations seen in population studies as causal evidence that drinking alcohol increases your risk of cancer.

Connor says alcohol causes some cancers. Her conclusion is based almost entirely on epidemiology studies that show drinkers have slightly higher rates of some specific cancers (and slightly lower rates of some others). In doing so she places great faith in epidemiology, far more than many of her colleagues. A more sober analysis would adopt the epidemiologists’ rule of not considering risk assessments of less than a factor of 3 as indicating any causality (see Taubes, G. 1995. ‘Epidemiology faces its limits.’ Science, 269:5221, 164-69). Unlike smoking, alcohol and cancer studies seldom report risk estimates anywhere near this level.

The key problem with epidemiology studies is that they show correlations that are often not causal, they can be completely spurious due to confounding factors. For example, studies might show that elderly people who get regular exercise live longer, but elderly people who are afflicted with health issues are very likely to exercise less (many will be simply unable to), so it is really health that is determining both the exercise as well as the longevity. Exercise might be doing good here but the risk estimate (for not exercising) is going to be way over-estimated.

The problem with comparing alcohol drinkers, of various levels, to lifetime non-drinkers is that alcohol drinkers tend to smoke more, they also tend to be less rural and wealthier. Moderate drinkers might be people who tend to be adopt other healthy behaviours. Heavy drinkers may be self-medicating because of mental or physical ill-health. There are plenty of reasons for caution in treating a correlation seen in population data as causal. Here are some funny examples of real but non causal correlations.

Connor understands the possibility of confounds producing spurious correlations yet completely refrains from discussing any. Instead she goes off on a tangent discussing possible confounds for the well established link between drinking and much lower risk of heart disease. Here she suddenly loses her faith in epidemiology, i.e. when she doesn’t like the results. To even discuss heart disease is rather odd given that her article is about cancer – it’s truly bizarre given she doesn’t have a section covering confounds for alcohol and cancer.

In the absence of controlled experiments (which for cancer research are pretty much impossible for ethical and practical reasons) we need other evidence to support treating a epidemiology result as causal. Connor’s article makes 3 points:

1. First, Connor says, there is a dose-response relationship between alcohol consumption and some cancers, that is, any increase in drinking is associated with increased cancer risk. However, she then (rightly so) contradicts herself in pointing out that for most of these cancers meta-analysis shows no risk for light drinking. In fact some meta-analyses show risk only for the heaviest level of drinking (which is problematic as this group may contain alcoholics and people drinking as self-medication for health problems).
2. Second, Connor notes “evidence that, for some cancers, the risk associated with alcohol attenuates when drinking ceases”. Now this is what we would expect if there were a causal relationship, however it is odd that in this epidemiological analysis the risk drops so terribly slowly, taking decades before drinking cessation results in the same risk as a non-drinker. This is far longer than the effects of cigarette cessation, suggesting the apparent alcohol risks have more to do with confounding factors than a direct causal relationship (i.e. lifestyle factors that slowly regress to the mean). Besides, this evidence is of poor quality; to quote from one of the meta-analyses that Connor cites “Too few studies have addressed this question and of the studies that have, all have significant limitations” and “the only statistically significant relationship that we observe is that drinkers who recently quit drinking have a higher risk of liver cancer than current drinkers”! It would clearly be wrong to make much of this evidence, the models have huge error margins, and are hardly supportive of a causal link between alcohol and cancer.

3. Third, Connor discusses briefly potential biological mechanisms for alcohol perhaps causing cancer. Alcohol in itself is not carcinogenic to human cells, and the epidemiology shows associations with only some cancers not others (indeed drinking seems to be associated with less of some cancers). As Connor writes: “The mechanisms by which alcohol causes cancer are not well understood, but are thought to depend upon the target organ. Pure ethanol does not act as a carcinogen in animal studies, and evidence that it causes mutations directly in humans is weak”. A possible mechanism is that bacteria in the mouth and digestive tract convert alcohol to acetaldehyde, which is a carcinogen. As Connor notes “Stronger associations and more susceptibility at low doses is seen for the cancers where alcohol and [hence] acetaldehyde come into direct contact with the tissues”. She doesn’t mention, but it is important, that this potential bio-mechanism is supported by the evidence that people with mutated ALDH enzymes (somewhat common in Asia, very rare for West Europeans) and so are slow to break down acetaldehyde, have substantially higher rates of these cancers even though they drink less.

So for oral/throat cancers we have the highest correlations in epidemiology data (though nowhere like the magnitude for smoking), and we have a plausible mechanism (the culprit being acetaldehyde). Plus we can add the evidence that Connor missed, there are much higher rates of oral/throat (aerodigestive) cancer amongst people who do not have the genes to quickly process acetaldehyde. This is by far the best case for making a causal connection between drinking alcohol and a cancer. Though it must be noted that aerodigestive cancers are rare (eg a tiny 0.3% of US deaths are from oral cancers, and most are caused by smoking, age and genes) even though most of the population drinks alcohol.

For the other cancers Connor mentions, the evidence of causality is extremely poor. In comparison, the evidence is vastly more convincing that drinking alcohol reduces heart disease, diabetes, dementia, and overall mortality. In addition to meta-analyses of many many population studies, we have plausible biological mechanisms, supported by lab studies, animal experiments, and hospital trials. Meta-analysis of dozens of experiments shows administering alcohol to subjects leads to rapid changes in biomarkers for heart disease (Brien et al 2011). We also see changes in drinking linked to changes in rates of heart disease, diabetes and overall mortality, e.g. people who increase their drinking lower their risk of heart disease. In sum, this is what a plausible case for causality looks like.

Apart from oral/throat and perhaps liver cancer Connor is wrong, there isn’t evidence to support drinking causing cancer. And even for oral/throat cancer the risk may be confined to smokers, and people with particular ALDH enzyme mutations.

But that’s if we stop with Connor’s superficial analysis. There is more evidence to consider if we are to form a proper judgement about causality. Read on…

Connor concluded that drinking probably was causal for 7 cancers. This has a nice ring about it…. like the ‘7 Deadly Sins’ (Oil of Olay understand this with their ‘fights the 7 Signs of Aging’ slogan). Now, Connor used the old medical convention of labeling cancer according to where the tumour occurs. It’s possible to talk of hundreds if not thousands of different cancers, but more reasonably for this discussion we have (1) oral/throat/oesophagus (aerodigestive cancers) which we have already discussed, (2) liver, (3) colorectal, and (4) breast cancer. It’s this last one that drives most public health forecasts that reductions in
drinking could reduce cancer, this is because breast cancer is the most common potentially deadly cancer for women. That said, less than 3% of female deaths in the USA are from breast cancer. 90% of breast cancer is not fatal, due to curative treatment but also because many breast cancer diagnoses are for non-fatal cancer. Over-diagnosis is a very real problem.

Breast cancer is a prime example of how deficient Connor was not to discuss evidence on confounding factors. Meta-analysis shows no link between drinking and breast cancer mortality, nor with recurrence (Gou et al 2013). While a very new study from the Women’s Health Initiative (Lowry 2016) again shows no link between drinking (before or after breast cancer diagnosis) and mortality. Similarly a large study with long follow-up of women with breast cancer (Newcomb et al 2013) showed they had better chances of survival if they were regular drinkers before diagnosis. If they altered their drinking after diagnosis this had no link to their chance of dying from breast cancer, but an increase in drinking was associated with an overall increase in life expectancy (largely due to substantially fewer heart disease deaths among those who increased their alcohol consumption). This is strong causal evidence that alcohol prevents heart disease, and it seriously conflicts with the idea of alcohol causing breast cancer.

Similarly while animal experiments show drinking results in less heart disease, and longer life overall they do not show a link between alcohol consumption and breast cancer (see Hackney et al 1992, Singletary 1997). While alcohol actually reduced the risk of breast cancer metastasis in mice (Vorderstrasse et al 2012).

The (weak) correlation seen in population studies between drinking and breast cancer diagnosis is then probably due to a confounding factor, as so often can happen in population studies. In this case probably simply that drinkers are more likely to screen for breast cancer (shown in Mu and Mukamal 2016) – screening absolutely increases diagnosis (and quite a few of these diagnoses will be false, or non harmful). Land et al 2014, which screened all their subjects (i.e. controlling for screening incidence), showed no link between drinking and breast cancer diagnosis – indeed drinking was associated with slightly less risk of diagnosis (of both breast and colon cancer).

The modest degree of increased risk of colorectal cancer for alcohol drinkers may also be spurious or exaggerated for the same reason – drinkers screen more, which results in more diagnoses. As far as a potential biological mechanism some fecal bacteria have been shown to convert alcohol to acetaldehyde in vitro (Jokelainen et al 1994). While other bacteria have been shown to break down acetaldehyde (Nosova et al 2000) – the flora of the human gut are complex. Acetaldehyde has been shown to exist in the colons of rats but the level was not affected by feeding the rats alcohol (Seitz et al 1990). Research is needed to see if drinking really can increase acetaldehyde levels in the colon. In humans alcohol is absorbed in the stomach and small intestine which makes it harder to explain how alcohol might reach the colon where it could be converted to acetaldehyde by bacteria. Whereas risk of oral cancer is much higher among people without the genes to produce some adetaldehyde processing enzymes this does not appear to be the case for colorectal cancer (Tiemersma et al 2003). So a causal link between drinking alcohol and colorectal cancer remains speculative, and if there is a link it is not strong – non and light drinkers still get colorectal cancer at much the same rate as heavy drinkers.

That leaves us only with liver cancer to discuss. This is a rare but deadly cancer, causing 1% of deaths in the USA, though most cases are due to viral (hepatitis) infection, obesity, diabetes, other disease, and genetics. We perhaps again have the plausible biological mechanism of acetaldehyde but only at high levels of drinking as the liver is very efficient and fast at breaking down acetaldehyde. It’s also very plausible that alcohol, through its effect on liver disease, leads to higher risk of liver cancer. However very few drinkers develop liver disease, so the degree of absolute increase in risk is absolutely tiny and probably only for long-term alcoholics or those unlucky enough to have liver damage from hepatitis, other disease, or obesity.

So it’s plausible that alcohol contributes to some cancer, but only a few rather rare cancers, and its influence can’t be great. So we would not expect drinkers to die much more often from cancer, and this is exactly what is observed. Meta analyses report cancer mortality is barely higher among drinkers than occasional/zero drinkers and confined to the cohort who admit to consumption of more than 50g of
alcohol per day (see Jin et al 2012, table 2). In Thun et al 1997 (a study of death among middle class, middle aged and elderly americans) the heaviest drinkers showed higher rates of death from alcoholism and injury, as expected. They were more likely to die from aero-digestive and liver cancer, again as expected, though none of these deaths were common. As expected, they were less likely to die from heart disease, stroke and other circulatory disease – each of which were major causes of death. There was no consistent relationship with rates of drinking and rates of death from colorectal or breast cancer, as expected.

Finally it is worth noting, there is clear way that drinking alcohol (at least to non-alcoholic levels) increases your chance of any cancer, and that is through its effect on living longer. For most cancers age is the dominant risk factor (e.g. half of all colorectal cancers occur in people aged over 70 years old, half of breast cancer is for women aged over 62). Drinkers live longer, largely due to reduced levels of heart disease (also diabetes, and dementia), perhaps it’s also due to drinkers being more social or some other beneficial behaviours – but whatever the cause it means that drinkers will live longer and will therefore have to get more cancer (as everyone has to die from something). This fact alone is enough to produce an association, but not a direct causal one, between drinking and cancer.

*Otago University website describes Connor as having “a long-standing research interest in alcohol-related harm”.

Thanks to these people for commenting on earlier drafts:

Ian Olver, Professor of Translational Cancer Research, Director Sansom Institute for Health Research, University of South Australia.


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Reducing the harms of alcohol through weaker beer

A review in Lancet Gastroenterology & Hepatology suggests that a small drop in the alcohol content of beer or other drinks could reduce the harmful effects of alcohol in society at large.

Dr Jürgen Rehm, lead author and Director of the Institute for Mental Health Policy Research at the Centre for Addiction and Mental Health (CAMH) in Toronto, Canada explains "The idea is that a small reduction in alcohol - such as beer with 4% ethanol content versus 6% - would reduce alcohol intake per drinker if the same overall amount of beverage is consumed."

A decrease in ethanol would lead to lower blood alcohol levels in drinkers which, in turn, could reduce immediate harms such as injuries or accidents, as well as alcohol-related chronic diseases that develop over time, such as liver cirrhosis or cancer.

The researchers note that there is more incentive for the alcohol industry to adopt this proposal, compared to other policy measures such as higher taxation, limited access and marketing restrictions. And in addition, the industry holds some responsibility for their product. A key concern is that consumers would notice the difference in alcohol content, and consume more to compensate or switch to other beverages with more alcohol.

The researchers searched for studies and reviews on all of these points. Overall, there was not much research that directly examined the effects of lower alcohol content in relation to reducing harms on a large scale. Some research however suggests that concerns around drinkers' behaviours are not warranted.

"We know from experiments that consumers can’t distinguish between beers of different strengths," said Dr Rehm. In one study set at three fraternity parties, the amount party-goers drank didn’t differ with weaker versus stronger drinks. In another study, participants were given lower- and higher-strength beer on two different occasions, and most did not report differences in how they felt after these sessions. In both studies, participants had a significantly lower blood alcohol concentration with lower-alcohol drinks.

On a broader scale, The Northern Territories of Australia levied a tax on alcohol with more than 3% ethanol, which led to greater availability of lower-strength beer. This policy change resulted in fewer alcohol-related deaths, although it ran in parallel with educational efforts, greater controls on availability and new treatment services.

Another approach to reducing alcohol harms identified would be offering alcohol-free drinks as a cheaper alternative in bars or restaurants. The drawback is that not only does it require drinkers to choose this option. Currently, there is limited evidence that it affects drinking levels or alcohol-related harms.

Ultimately, the question of whether lower-strength alcohol can help reduce the burden of alcohol harms will depend on how any measure is implemented and evaluated, the researchers note. But the evidence suggests it is worth considering as a win-win for public health efforts and alcohol producers. "The proposal presents a unique situation, where public health interests in reducing alcohol consumption is not in conflict with the alcohol industry," Dr Rehm commented.


Alcohol Licensing: Responding to Reform

London | Thursday 24th November | 11:00-15.30

The Policing and Crime Bill 2016 contains reforms to alcohol licensing. The Government has also outlined changes to alcohol licensing in the Modern Crime Prevention Strategy. This new approach seeks to strengthen Local Authorities’ powers to license premises and tackle alcohol-related crime in their area.

Attend this Westminster Briefing to hear about the upcoming changes and how to put new licensing policy into effective practice in your area

www.westminster-briefing.com/AlcoholLicensingBriefing
Young adults and Drunkorexia in the UK and US

According to the 2016 National Health Report by Benenden, young British men and women are skipping meals in favour of binge-drinking. The report found that the problem of ‘Drunkorexia’ is now prolific in the UK. 43% of men and 35% of women aged between 18-24 admitted to skipping meals in favour of binge drinking. The Pressure to be slim, an awareness of exercising calorie control, and peer pressure to drink large amounts of alcohol are all factors in this phenomenon. When asked, 41% of 18-24 year olds said they ate healthily purely with a view to looking good, without any concern for their overall health.

The report found that a reckless attitude towards personal health, and relative lack of concern as to the long-term health ramifications of Drunkorexia were not just confined to young people. A lack of knowledge was displayed across all ages in the 2016 National Health Report.

Dr John Giles, Medical Director at Benenden, commented: “It seems that basic information about diet and wellbeing is not getting through to the public, and despite drinking less, many young people are seemingly favouring alcohol consumption over a healthy, balanced diet.”

A similar pattern has been observed in a US study involving 1,184 college students between the ages of 18 and 26 years. Most of the students attended the University of Houston in Houston, Texas.

Researchers found that 80% of those studied had demonstrated some kind of drunkorexia in the past three months. They had performed heavy physical exercise, eaten low calorie meals or even missed meals for up to a full day before drinking alcohol.

Dipali Rinker organised the study and presented its findings to the Research Society on Alcoholism in June. Rinker teaches psychology at the University of Houston. She says students see drunkorexia as a way to keep their body weight down while drinking alcohol. And it causes them to feel the effects of alcohol quickly and with more intensity.


Adolescent alcohol use: Protective and predictive parent, peer, and self-related factors

A recent study highlights variables that mitigate or predict alcohol use and heavy episodic drinking. Adolescent alcohol use has been linked with a multitude of problems and a trajectory predictive of problematic use in adulthood. Thus, targeting factors that enhance early prevention efforts is vital.

Using Monitoring the Future (MTF) data, multiple path analytic models revealed links between parental involvement and alcohol abstinence and initiation. Parental involvement predicted enhanced self-esteem and less self-derogation and was negatively associated with peer alcohol norms for each MTF grade sampled, with stronger associations for 8th and 10th graders than 12th graders. For younger groups, self-esteem predicted increased perceptions of alcohol risk and reduced drinking. Self-derogation was associated with peers’ pro-alcohol norms, which was linked to lower risk perceptions, lower personal disapproval of use, and increased drinking. Peer influence had a stronger association with consumption for 8th and 10th graders, whereas 12th graders’ drinking was related to personal factors of alcohol risk perception and disapproval. In all grades, general alcohol use had a strong connection to heavy episodic drinking within the past 2 weeks.

The authors suggest that across-grade variations in association of parent, peer, and personal factors suggest the desirability of tailored interventions focused on specific factors for each grade level, with the overall goal of attenuating adolescent alcohol use.

Progression from first drink, first intoxication, and regular drinking to alcohol use disorder

Authors of a recent study state that differences between African Americans (AAs) and European Americans (EAs) in the prevalence and age at onset of alcohol use and alcohol use disorder (AUD) have been documented, but distinctions in the timing of early stage transitions and contribution of various psychiatric and psychosocial risk factors to the progression from initiation to AUD have yet to be investigated. Their study characterised progression from alcohol use initiation-defined alternatively as first drink, first intoxication, and regular drinking onset to AUD in AA and EA youth.

The study was based on telephone psychiatric interviews with 1,461 participants (56% AA, 44% EA) in a high-risk family study. 50.3% of participants were female and the mean age was 17.6. Cox proportional hazards regression analyses were conducted separately for the AA and EA subsamples to predict DSM-5 AUD as a function of age at alcohol use initiation, with age at first drink, age at first intoxication, and age at regular drinking onset as the point of origin in separate models.

Across race/ethnicity, regardless of how it was measured, early alcohol use initiation predicted AUD, but hazard ratios (HRs) were lowest for first drink. Regular smoking and social anxiety disorder were significant predictors in both racial/ethnic groups, but associations with conduct disorder (all 3 models: HR range = 2.07 to 4.15) and major depressive disorder (regular drinking: HR = 4.51, confidence interval [CI]: 1.60 to 12.69 for AUD onset ≥ age 20) were specific to AAs. Posttraumatic stress disorder (HR = 5.38, CI: 1.44 to 20.08) and generalised anxiety disorder (HR = 7.35, CI: 2.31 to 23.34 for AUD onset ≤ age 17) were strongly associated with progression from regular drinking to AUD exclusively in EAs.

Early alcohol use initiation is a marker of risk for AUD in both AA and EA youth, but the contributions of various psychiatric risk factors to the development of AUD are not universal across racial/ethnic groups, the authors conclude.


Alcohol consumption by youth: peers, parents, or prices?

Using data from the National Longitudinal Survey of Adolescent to Adult Health, researchers estimated the effect of peers' alcohol consumption and alcohol prices on the drinking habits of high-school-age youth.

They found that peer effects are statistically and economically significant regarding the choice to participate in drinking but are not significant for the frequency of drinking, including binge drinking. Alcohol prices were not found to be significant.

The authors conclude that no significant impact on underage drinking will result from low-tax states' increasing excise taxes on alcohol in line with those of high-tax states. The results may be important for policymakers who are considering policies to reduce underage drinking, the authors suggest that policymakers may choose to focus instead on the influence of peers and changing the social norms behaviour instead.

Source: Alcohol consumption by youth: peers, parents, or prices? Ajilore O; Amialchuk A; Egan K Economics and Human Biology Vol 23, 2016, pp76-83.
AIM SOCIAL AND POLICY NEWS

Could physical activity be good for alcohol and substance use disorders?

A team of researchers from Plymouth University Peninsula Schools of Medicine and Dentistry and Plymouth Hospitals NHS Trust, has received funding of £154,000 from the NIHR Research for Patient Benefit (RfPB) programme, to carry out a systematic review of research to see if and how physical activity and exercise could help those with alcohol and substance use disorders.

The study is led by Dr Tom Thompson, Research Fellow at Plymouth University Peninsula Schools of Medicine and Dentistry, who commented: "We wanted to investigate the evidence about physical activity in relation to these disorders because it could help in prevention, reduction, and treatment. It may be that physical activity diverts attention away from the addiction, helps to build confidence or improves mood, reduces withdrawal symptoms, and supports a shift towards a healthy identity, which is incompatible with excessive alcohol or substance use. It is also possible those who engage in physical activity in their younger years are less likely to develop problems with alcohol and substances later in life. Compared with drug treatments and other therapies, physical activity has little or no bad side effects and is potentially cheap and easily accessible."

The research team will analyse the evidence from existing research, which in turn will lead to a better understanding of if and how physical activity could be used as a tool for prevention, reduction, and treatment.

Once the research team has findings, it will share them with health and social service providers, funders, service users and others to gain their views on what the best methods are and to whom they should be offered. The team will also estimate costs for using physical activity to address alcohol and substance use disorders.

The study will complete with a final report which will provide important practical information on what has worked so far, and what may be most effective in the future as an alternative approach to preventing and treating alcohol and substance use disorders.

www.plymouth.ac.uk/news/could-physical-activity-be-good-for-alcohol-and-substance-use-disorders

The association between parental attitudes and alcohol consumption and adolescent alcohol consumption in Southern Ireland

A study aimed to investigate the association between adolescent alcohol consumption and their parent’s consumption pattern and attitude toward alcohol use in Southern Ireland.

A cross-sectional survey was undertaken in November 2014 of 982 adolescents in their final two years of second level education and at least one of their parents from a local electorate area in Southern Ireland. The survey gathered data on alcohol use, self-reported height and weight, smoking status, mental health and well-being along with attitudinal questions.

A 37% response rate was achieved. 34.2% of adolescents and 47% of parents surveyed reported hazardous drinking. Over 90% of parents disagreed with allowing their adolescent to get drunk and rejected the idea that getting drunk is part of having fun as an adolescent. 79.5% of parents surveyed believed that their alcohol consumption pattern set a good example for their adolescent. Multivariate logistic regression highlights the association between adolescent hazardous alcohol consumption and hazardous drinking by the father. Furthermore either parent permitting their adolescent to drink alcohol on special occasions was associated with hazardous alcohol consumption in the adolescent.

The research finding suggest that a liberal attitude to alcohol and increased levels of consumption by the parent are linked to hazardous adolescent drinking behaviour. Future action plans aimed at combatting adolescent hazardous alcohol consumption should also be aimed at tackling parents’ attitudes towards and consumption of alcohol, the authors conclude.

### Desire to drink alcohol is enhanced with high caffeine energy drink mixers

Authors of a study published in the journal Alcoholism, Clinical and Experimental Research state that consumption of alcohol mixed with energy drinks (AmED) has been associated with a variety of risks beyond that observed with alcohol alone. Consumers of AmED beverages are also more likely to engage in heavy episodic (binge) drinking. Their study was to investigate whether the consumption of high caffeine energy drink mixers with alcohol would increase the desire to drink alcohol compared to the same amount of alcohol alone using a double-blind, within-subjects, placebo-controlled study design.

26 participants who were social drinkers attended 6 double-blind dose administration sessions that involved consumption of alcohol and energy drinks, alone and in combination. On each test day, participants received 1 of 6 possible doses: (i) 1.21 ml/kg vodka + 3.63 ml/kg decaffeinated soft drink, (ii) 1.21 ml/kg vodka + 3.63 ml/kg energy drink, (iii) 1.21 ml/kg vodka + 6.05 ml/kg energy drink, (iv) 3.63 ml/kg decaffeinated soft drink, (v) 3.63 ml/kg energy drink, and (vi) 6.05 ml/kg energy drink. Following dose administration, participants repeatedly completed self-reported ratings on the Desire-for-Drug questionnaire and provided breath alcohol readings.

Alcohol alone increased the subjective ratings of ‘desire for more alcohol’ compared to placebo doses. Energy drink mixers with the alcohol increased desire for more alcohol ratings beyond that observed with alcohol alone.

This study provides laboratory evidence that AmED beverages lead to greater desire to drink alcohol versus the same amount of alcohol consumed alone. The findings are consistent with results from animal studies indicating that caffeine increases the rewarding and reinforcing properties of alcohol.


### Prevalence and correlates of drink driving within patrons of Australian night-time entertainment precincts

A study examined the prevalence and correlates of drink driving behaviour in a sample of night-time entertainment precinct attendees in Australia. Interviews were conducted with 4,214 night-time entertainment precinct attendees in two metropolitan and three regional cities in Australia. Seven correlates of self-reported drink driving were examined: gender, age, occupation, blood alcohol concentration (BAC), alcohol consumed prior to attending a licensed venue, energy drink consumption, and other drug consumption.

14% of respondents reported drink driving in the past three months. Bivariate logistic regression models indicated that males were significantly more likely than females to report drink driving in the past three months. Blue-collar workers and sales/clerical/administrative workers were significantly more likely to report drink driving behaviour in the past three months than white-collar workers. The likelihood of reporting drink driving during the three months prior to interview significantly increased as BAC on the current night out increased, and when patrons reported engaging in pre-drinking or other drug use. The multivariate model presented a similar pattern of results, however BAC and pre-drinking on the night of the interview were no longer independent significant predictors.

Males, blue collar/ sales/ clerical/ administrative workers, and illicit drug consumers were more likely to report engaging in drink driving behaviour than their counterparts. The authors state that interventions should focus on addressing the considerable proportion of night-time entertainment precinct attendees who report engaging in drink driving behaviour.

A new Public Health England report examines the available data on alcohol use and harm among young people under 18 years of age to investigate trends and highlight any areas for public health action.

Key findings are:

- There is an ongoing downward trend in alcohol consumption among those aged under 16. However, by the age of 17, half of all girls and almost two-thirds of boys report drinking alcohol every week.
- Young White populations are much more likely to drink than those from a Black and Minority Ethnic group background.
- Young people in the least deprived areas are more likely to drink and more likely to drink regularly at the age of 15.
- Hospital admissions for alcohol-specific conditions, particularly intoxication, are declining among the under 18s.
- Girls are more likely to be admitted to hospital for alcohol-specific reasons than boys, and are admitted at younger ages.
- The number of young people accessing specialist substance misuse services for alcohol problems is at its lowest level, following a peak in 2008-09. These young people have a range of related risk factors and vulnerabilities that should be addressed in tandem with their substance misuse.
- There is some evidence that the alcohol-harm paradox seen among adults is also present for young people living in the most deprived areas.
- There is a strong relationship between smoking and drinking, with current smokers much more likely to drink alcohol frequently than non-smokers.

The report concludes that fewer young people are drinking alcohol than they did in the past and fewer are suffering serious health implications needing attendance at hospital. However, the proportion of children in the UK drinking alcohol remains well above the European average and British children are more likely to binge drink or get drunk compared to children in most other European countries. Other consequences of alcohol consumption such as regretted sexual activity, arguments, involvement in crime and violence are more prevalent than hospital attendance.

According to the report, alcohol use can be linked to other risk taking behaviour and therefore young people are likely to benefit from integrated wellbeing services. The strong interaction with smoking also suggests that joint action tackling both behaviours would be beneficial. This may also help to reduce health inequalities as alcohol harms and smoking prevalence are more likely to occur in more deprived areas.

Professionals from health, education, social care and youth justice agencies need to be able to identify, assess and, where necessary, appropriately refer young people experiencing alcohol-related problems.

UK government advice on low risk drinking is published

On 25 August, the UK government published ‘Alcohol consumption: advice on low risk drinking’. Produced by the 4 UK chief medical officers, the advice issued in January by the chief medical officer Sally Davies remains unchanged.

The guidelines for both men and women are that:

- keep health risks from alcohol to a low level it is safest not to drink more than 14 units a week on a regular basis.
- If you regularly drink as much as 14 units per week, it is best to spread your drinking evenly over 3 or more days. If you have one or two heavy drinking episodes a week, you increase your risks of death from long term illness and from accidents and injuries.
- The risk of developing a range of health problems (including cancers of the mouth, throat and breast) increases the more you drink on a regular basis.
- If you wish to cut down the amount you drink, a good way to help achieve this is to have several drink-free days each week.

The document states that the UK CMOs’ guidelines and the Guidelines Development Group report that underpins them, have been developed on the principles that:

- ‘People have a right to accurate information and clear advice about alcohol and its health risks. Consequently the guidelines have been developed so that the known health risks of different levels and patterns of drinking, particularly for people who want to know how to keep long term health risks from regular drinking of alcohol low, are both accurate and expressed in an understandable way.
- Government has a responsibility to ensure this information is provided for the public in a clear and open way, so they can make informed choices. It is for individuals to make their own judgements as to the risks they are willing to accept when they drink alcohol, also whether to drink alcohol, and how much and how often to drink. These guidelines should help people to make those choices’.

There is, however, an attempt to qualify Davies’ statement that “drinking any level of alcohol regularly carries a health risk”. The report’s advice on regular drinking is based on the evidence that if people drink at or above the low risk level advised, overall any protective effect from alcohol on deaths is cancelled out and the risk of dying from an alcohol-related condition would then be expected to be at least 1% over a lifetime. This level of risk is comparable to those posed by other everyday activities that people understand are not completely safe yet still undertake.

‘The latest research also indicates that when drinking within the low risk guidelines, overall levels of risk are broadly similar for men and women; although the risks of immediate harms such as deaths from accidents are greater for men; longer term harms from illness are greater for women’.

‘The expert group was also clear that there are a number of serious diseases, including certain cancers, which can occur even when drinking within the low risk guidelines. Whilst they judge the risks to be low, this means there is no level of regular drinking that can be considered as completely safe in relation to some cancers. People can reduce these risks by drinking less than the guidelines or by not drinking at all’.

Reported road casualties in Great Britain

Final estimates for 2014 show that 240 people were killed in accidents in Great Britain where at least one driver was over the drink drive limit. This is unchanged from 2013.

The fatalities figure is an estimate based on coroners’ and procurators’ fiscal reports for 62% of the drivers or riders who were killed in road traffic accidents in 2014. As the figure is based on an incomplete sample, the true figure could be between 220 and 260 fatalities at a 95% confidence level. Following a sharp drop in deaths between 2009 and 2010 (a fall of around 40%) drink drive deaths have been stable since 2010, at a very low level of between 230 and 240 deaths each year.

• the number of seriously injured casualties in drink drive accidents decreased by 3% from 1,100 in 2013 to 1,070
• the total number of casualties of all types in drink drive accidents is 8,210
• the total number of drink drive accidents of all severities fell by 1% to 5,620

Males account for 70% of killed or seriously injured (KSIs) in all road accidents in 2014. This is reflected in drink drive accidents with 77% of KSIs are sustained by males. The proportion of killed drivers and riders over the limit is highest amongst 25 to 39 years old. In 2014, around 25% of those killed from this age group were found to be over the limit, compared with less than 15% for older age groups. The proportion of the killed youngest driver age group (16 to 24 years old) that were found to be over the limit was 21%.


Northern Ireland - Strategy to prevent and address the harm related to the misuse of alcohol and drug misuse – annual report on progress

The Northern Ireland Department of Health (DoH) has published its fourth annual report on progress against the cross-departmental strategy to prevent and address the harm related to the misuse of alcohol and drug misuse. Launching the report, Northern Ireland Minister of Health Michelle O’Neill applauded the progress made with regard to alcohol. Following a review of alcohol and drug services, new services are in place and revised care pathways have been put into operation and new alcohol guidelines have been consulted on by the Chief Medical Officer.

O’Neill noted that while the DoH has made progress in various areas of harm prevention, harmful drinking still costs Northern Ireland approximately GBP £900 million annually, and that there are approximately 400 deaths attributed to harmful drinking and drug abuse every year.

Summer drink-driving crackdown in the UK

The National Police Chiefs’ Council (NPCC) launched a campaign to target those who drive under the influence in England, Wales and Northern Ireland. As a result, almost 50,000 vehicles were stopped between June 10 and July 10, with 45,000 breath tests carried out. Of those, roughly 10%, 4,539, were found to be positive, refused - meaning the driver would not give a specimen of breath - or failed - meaning a specimen is given, but it is not sufficient to determine a result. Fewer cars were stopped and fewer tests were carried out compared to the initiative last summer, but a larger proportion of alcohol tests were positive, refused or failed.

Drug screening devices were deployed on 2,588 occasions, with 1,028 positive tests. Police can also make those they stop carry out a ‘field impairment assessment’ if they suspect drivers to have taken drugs. Some 279 of these tests were carried out over the month, with 80 resulting in an arrest. Under new legislation legal driving limits were laid down for 17 prescription and illegal drugs in March last year. There is virtually zero tolerance for drivers apprehended with substances such as heroin, cocaine and cannabis in their system.

Chief Constable Suzette Davenport, national lead for roads policing, said: "It is encouraging to see that our intelligence-led approach continues to work - fewer tests administered but increased criminal justice outcomes, with forces actively targeting hotspots and using their local knowledge to get drink- and drug-drivers off our roads."

"Even though this has been a successful summer campaign, it is still disappointing to see during the campaign over 4,500 people drink driving and over 1,000 people driving whilst under the influence of drugs”.

Airport alcohol sales to be 'examined' in the UK

The way alcohol is sold in airports is to be examined after a number of recent incidents involving drunk passengers. Lord Ahmad, the new aviation minister will look at the times alcohol is on sale, and passenger screening.

While airlines are able to police the amount of alcohol served in flight, and refuse to serve passengers who are drunk, they cannot control what happens before boarding. Some passengers, especially groups of young people, “pre-load” in airport bars. In addition, some travellers open bottles of spirits bought in airport shops once on board the plane, even though this contravenes regulations. Latest figures show that 442 people were held on suspicion of being drunk at an airport or on a plane in the last two years.

In August, Leisure airline, Jet2.com, banned the sale of alcohol on-board before 8am on all its morning flights ‘to ensure everyone has an enjoyable and comfortable journey’.

Drinkaware Crew launched

Drinkaware has launched a new scheme in Bolton to help keep young people safe and enjoy their night out. Run in partnership with Greater Manchester Police (GMP), the scheme provides specially trained staff known as Drinkaware Crew at a nightclub in Bolton. The objective is to help promote a positive social atmosphere, reduce negative experiences related to drunkenness – including anti-social behaviour and sexual harassment – and help minimise the associated costs that drunken behaviour can have on the local night-time economy.

Drinkaware Crew will engage with clubbers and support young people who might be vulnerable as a result of drinking too much, the staff are easily recognised and present throughout the night to make sure that young people are having fun and get home safely.

The scheme’s launch in Bolton follows a six month pilot in November 2015 that included five areas in South West England: Cheltenham, Exeter, Plymouth, Truro and Torbay – in addition to Nottingham It forms part of Drinkaware’s Drunken Nights Out campaign, developed to reduce alcohol related harm in the night time economy. Following the pilot, the initiative is expected to be expanded across the UK.
UK Independents warned over teenage drinking following GCSE results

Independent retailers were warned to be extra vigilant after research revealed an “alarming number” of 15- and 16-year-olds intended to celebrate with alcohol when they find out their GCSE results on August 25.

The survey of 1,000 15- and 16-year-olds – which was commissioned by Under Age Sales found that 45% of the 700,000 GCSE students said that they could well end up celebrating results day with alcohol, with one-third planning to purchase the alcohol themselves from independent retailers. 30% said that they would ask a family member or friend to buy alcohol for them, while 10% intended to use a fake ID.

Tony Allen, managing director of Under Age Sales, said “The law around age-restricted products is there to protect young people and our communities. Retailers are the gatekeepers to preventing teenagers accessing items such alcohol and effective training can help empower them to carry out their work legally, fairly and confidently.

“Lack of knowledge or resources isn’t an excuse – retailers have a responsibility to ensure all staff receive ongoing training and that they have the correct procedures in place to stop young people obtaining products they shouldn’t.”

www.underagesales.co.uk/learning.html

Austria to introduce alcohol interlock programme in 2017

In Austria, Transport Minister Jörg Leichtfried has announced that the country’s convicted drink drivers will be given the option of installing an alcohol interlock in their vehicles from 2017, rather than face a driving ban. The optional scheme is likely to be attractive to people who rely on their cars for their job, and the government expects demand for the interlock option to be high. Every year about 26,000 people in Austria have their driver’s license revoked for drink driving.

The cost of the system, paid for by the driver, will be around EUR 2,500 a year plus administration costs including issuing a new driving license which includes the standardised EU code for drivers who are subject to alcohol interlocks (code 69).

In 2012 and 2013, the Austrian government commissioned two successful trials of the programme. Austria will be the eighth EU country to introduce an alcohol interlock programme following Poland’s adoption of the technology last year.

Statistics on Wales’ alcohol and drugs treatment programme

Services for people with drug and alcohol misuse problems in Wales have been criticised after new figures showed just a fraction of people have successfully completing their treatment. The data was collated by NHS Wales from the Welsh National Database for Substance Misuse (WNDSM) which contains details of all referrals to drug and alcohol agencies in Wales.

The latest statistics from the Welsh Government show that just 13% of patients were deemed substance-free by the end of their treatment between January and March. That equates to 805 out of 6,084 people who were treated across the country during that period. It was a similar picture for October to December 2015 (12.9%), July to September 2015 (12.2%) and April to June 2015 (10.4%). In addition, of the 4,019 people who started treatment between January and March 2016, 12% waited between four to 12 weeks before they were seen and 2.4% waited between six to nine months.

The Welsh Government commented that the fact someone is not drug free at the end of treatment does not mean the treatment has failed. A Government spokeswoman said the criticism was naive and demonstrated a lack of understanding about the complexities of dealing with substance misuse “Let’s be clear, we do not have drop out rates as high as suggested – the fact that someone is not drug free at the end of treatment does not mean the treatment has failed… The completion of a structured, formal treatment is not the end of treatment. For many it will be a lifelong battle and many will seek and receive other forms of support”.
Europe – the cost of fake alcohol

The European Union Intellectual Property Office (EUIPO) has published a report presenting the results of the eighth sectorial study, covering the production of spirits and wine.

It is estimated that the legitimate industries loses approximately €1.3 billion of revenue annually due to the presence of counterfeit spirits and wine in the EU marketplace, corresponding to 3.3% of the sectors' sales. This also means that 4.4% of legitimate sales of spirits and 2.3% of legitimate sales of wine are lost each year due to counterfeiting of alcoholic drinks. Those lost sales translate into 4,800 jobs directly lost across the spirits and wine sectors in the EU, as legitimate manufacturers employ fewer people than they would have done in the absence of counterfeiting.

When the knock-on effects of counterfeit wines and spirits in the marketplace are taken into account, 18,500 additional jobs are lost in the EU economy, of which an estimated 8,600 jobs are in agriculture and 1,300 jobs in the food industry.

The total yearly loss of government revenue as a result of counterfeit products in these sectors across the EU-28 is estimated at €1.2 billion.

New York tightens regulations for alcohol, drug use while boating

New York state is taking a tougher stand on boaters operating their vessels under the influence of alcohol or drugs by linking them to prior drinking while driving offenses involving a vehicle.

Tiffany Heitkamp's Law is legislation that strengthens penalties for boating while intoxicated offenses. The bill requires courts to consider prior Driving While Intoxicated or Driving While Ability Impaired convictions when sentencing a person for Boating While Intoxicated or Boating While Ability Impaired. Under current law, there is no linkage between alcohol or drugs related driving and boating offences, so it is impossible to convict an individual as a repeat offender, despite prior violations of similar laws. The bill addresses this gap and provides harsher penalties for repeat offenders.

This change would require a sentencing judge to impose a higher sentence on those who repeatedly get behind the wheel in an intoxicated state, regardless of whether it is a car or a boat. When an individual is cited for a BWI, carrying a 30-day sentence, the court must now consider any prior DWIs or DWAIs by the same individual within a five-year period.

Malta: Drink-driving limit to fall

In Malta, the Prime Minister's Office has announced that the government will reduce the legal blood alcohol concentration (BAC) limit for motorists from 0.8 mg/ml to 0.5 mg/ml, as part of an effort to reduce drink driving.

An government spokesperson stated that a government consultative committee is also drafting recommendations for new drink driving penalties, and that, while these have yet to be finalised, the government intends to impose “harsh” punishments in order for drivers to take road safety more seriously. - A 2010 Eurobarometer study found that 99% of drivers did not know the BAC limit in Malta.
Trends in perception of risk and availability of substance use among full-time college students in the US

A CBHSQ Report issued by SAMSHA in August provides 2014 estimates of past month substance use among young adults aged 18 to 22 overall and by college enrollment status (full-time college students vs. young adults who were not full-time college students). In addition the report provides estimates of perceptions of risk and availability for 2014 and examines trends in perceptions of risk and availability between 2002 and 2014 among full-time college students aged 18 to 22.

According to the report, full-time college students aged 18 to 22 may differ from young adults who are not full-time college students in their perceptions of whether there is great risk of harm from using substances. Differences in risk perception between these two groups may arise from many factors, including familiarity with the substances, the dangers associated with the frequency or amount of substance used, and knowledge of negative outcomes experienced by people who have used specific substances. The study found that full-time college students were more likely than same-aged young adults who were not full-time college students to perceive great risk of harm from daily binge drinking (63.9% vs. 55.3%), but there was no significant difference between the two with regard to their perception of great risk of harm from weekly binge drinking.

Some differences were seen in trends in perceptions of great risk of harm from weekly binge drinking. The percentage of full-time college students aged 18 to 22 who perceived great risk of harm from weekly binge drinking was higher in 2014 than the percentages from 2003 to 2006, but the percentage in 2014 was similar to the percentages in 2007 to 2013.

15th International Alcohol Ignition Interlock Symposium

September 13th–15th, 2016, at the Radisson Blu Royal Hotel in Brussels, Belgium.

Join international policy makers, criminal justice and health professionals, licensing authorities, researchers, government officials, industry representatives and innovators for three days of engaging interaction, exploration of new ideas and technological developments in the expanding field of alcohol interlocks.
MADD offer parents short topical guides

As part of its Power of Parents® programme, MADD is to produce customised, bite-sized topical guides, each focused on a specific topic related to preventing underage drinking. Sponsored by the National Alcohol Beverage Control Association, the guides will serve as quick reads that deliver focused information and strategies.

The first topical guide, Your Teen’s World, was published this summer. It will include data gathered by Dr Robert Turrisi, whose groundbreaking research on teens and alcohol consumption. Parenting Styles, an in-depth examination of various parenting styles and the impact it has on whether a child drinks underage or not will be published in the Autumn and Talking About Alcohol a will provide tools to talk with teens in a manner that gets through to them, will be published in the winter break.

These shorter guides, more focused guides will make it possible for parents to receive just the information needed for specific topic at hands and will be offered in addition to MADD’s Power of Parents handbook.

Mobile messages focus on health and safety issues facing students

In Indiana, a back-to-school programme is to be implemented that uses mobile and social media messaging to specifically target cell phones on college campuses and high school student populations. The mobile messages focus on health and safety issues facing students, including alcohol awareness and signs of alcohol poisoning and illustrate how to get help if faced with a medical emergency as well as tips on how to protect themselves and their friends under Indiana’s Lifeline Law while calling or Texting 911.

The programme is the first major public awareness initiative targeting college campuses to increase awareness about ‘Text-to-911’. The year-long public education campaign is initially funded with nearly $100,000 through a partnership which includes Indiana’s Statewide 911 Board; Indiana Youth Services Association and its Make Good Decisions Initiative.

The mobile messaging will target students across Indiana using “digital domes” - like a blanket over college campuses and large high school events - throughout the next school year. The ads and video messages are delivered through social media apps, and other popular mobile phone apps, when students access them on campus. Many of the ‘text messages’ featured in the ads are based upon actual emergency text conversations received by IN911.

Targeted prevention programmes in Victoria, Australia

While overall alcohol consumption is declining in Australia, recently published analysis shows that this is masking heavy drinking among certain subgroups. The Alcohol Culture Change Grants Initiative, launched on 10 August by VicHealth, will see councils apply for grants to offer more targeted harm reduction projects.

VicHealth chief Jerril Rechter stated “There is not one public health message to get people exercising — we need a different strategy for younger women than we do for older men, for example — and we need to do the same for drinking.” The programme is part VicHealth’s 10-year goal to have 200,000 more Victorians drink less alcohol by 2023.

New Zealand Government announces fetal alcohol plan

The New Zealand Government has a plan to reduce the number of babies born with fetal alcohol spectrum disorder (FASD). Associate Health Minister Peter Dunne says it’s considered internationally to be a leading preventable cause of intellectual and developmental problems, occurring in up to one in 100 children. “This is the first national step towards understanding and addressing FASD and its effects in New Zealand,” Mr Dunne said. The plan involves increased support for women with alcohol and drug issues, and research into the incidence of FASD in New Zealand. $12 million has been allocated over four years for intensive alcohol and drug support for pregnant women.
Alcohol industry to fund randomised control trial on the effects of moderate drinking

In response to some governments lowering low risk guidelines on alcohol consumption, and a lack of randomised control trials looking at the effect of moderate alcohol consumption on long term health in older populations, a group of beverage alcohol producers AB InBev, Diageo, and Heineken have announced that they will provide a joint contribution of USD $55.4 million to fund the first randomised study assessing alcohol’s effects on health, which will be led by the United States National Institute of Alcohol Abuse and Alcoholism (NIAAA). Although alcohol and its health effects are studied in detail across the world, there are a lack of randomised control trials due to the cost and complexity of commissioning such trials.

Researchers plan to enrol 8,000 subjects who are older than 50 and at risk for heart disease. Some will be randomly assigned to abstain from alcohol and the others to have one drink a day. The study will compare the incidence of heart attack, stroke and Type 2 diabetes in the groups after about six years. It isn’t designed to measure cancer risk.

The companies know “findings could go either way,” said a Pernod Ricard spokesman, Jack Shea, but they believe “it’s important to get a conclusive and definitive answer for public-health reasons for consumers and our business.”

New, improved guidelines for diagnosing fetal alcohol spectrum disorder in the US

On August 10, a group of experts on fetal alcohol spectrum disorders (FASD), organised by the National Institute on Alcohol Abuse and Alcoholism (NIAAA), released proposed clinical guidelines for diagnosing FASD. The new guidelines clarify and expand upon widely used guidelines issued in 2005, which were the first to help clinicians distinguish among the four distinct subtypes of FASD described by the Institute of Medicine. The updated guidelines, developed over one year by experts in the field, are based on analysis of 10,000 individuals involved in studies of prenatal alcohol exposure funded by NIAAA, part of the National Institutes of Health.

The proposed guidelines include a new definition of documented prenatal alcohol exposure, guides to evaluating facial and physical deformities characteristic of FASD, and updated information about the cognitive and/or behavioural impairments seen in different FASD subtypes.

“These new guidelines will be a valuable resource for clinicians to accurately diagnose infants and children who were affected by alcohol exposure before birth,” said NIAAA Director George F. Koob, PhD. “They represent the most data-driven diagnostic criteria for fetal alcohol syndrome and fetal alcohol spectrum disorder produced to date.”

The updated guidelines were developed and accepted by the Collaboration on FASD Prevalence and NIAAA’s Collaborative Initiative on Fetal Alcohol Spectrum Disorders.


Australian schools programme challenges social norms

The Australian Capital Territory government is to launch a pilot programme in four Canberra schools to equip students with the skills to critically analyse alcohol marketing in the hope of reducing underage drinking rates.

Game Changer+ is an initiative of the Foundation for Alcohol Research and Education who received $15,000 from the government to develop the programme.

The eight-lesson programme, which complements schools’ existing health education curriculum, will be taught to Year 9 and 10 students. Developed by a team of researchers from the Australian Catholic University’s Centre for Health and Social Research based in Melbourne, Game Changer + aims to provide a positive influence and challenge social norms around alcohol and young people. Lead researcher, Professor Sandra Jones, hopes the school programme will empower students to make wiser decisions over alcohol.

“We’re not telling them what to do, or what they’re doing is wrong... We’re just empowering students by giving them the information to make decisions,” she said.

According to professor Jones, research suggests children are exposed to a stream of alcohol advertising and their drinking attitudes and behaviours are strongly influenced by exposure to these messages.
Alcohol-related injuries in Queensland most likely to happen at home

A study from the University of New England in New South Wales looked at more than 12,000 alcohol-related injuries treated in Queensland hospitals over 10 years to ascertain where injuries most commonly happen.

There were 12,296 alcohol-related injuries over the 10 years, which represented 3.02% of all injuries treated in Queensland hospitals. 36% of alcohol related injuries took place in the home compared with 10% in licensed venues and 13% on the street.

Of the 3,971 alcohol-related injuries from assaults presenting to Queensland emergency departments, 29.2% (1,161) were at home, compared with 16.5% (656) in licensed venues and 13.1% (520) on the street. Just over 500 injuries were identified as the result of domestic violence by a spouse or partner, almost 60% of these taking place at home.

Mandatory alcohol interlock for some drink drive offenders in New Zealand

The New Zealand Government is making alcohol interlock devices mandatory for serious and repeat drink-drivers.

Associate Transport Minister Craig Foss announced the devices will become mandatory for anyone convicted of two or more drink-driving offences within five years, as well as first-time offenders caught driving more than 3.2 times over the legal limit. Once up and running, up to 5,000 people a year would have a sentence involving a mandatory interlock, Foss said - up from about a hundred at the moment.

The interlock would typically be in place for a year, with the sentence starting again if an offender blew a positive test with the device. Offenders would have to report to a service centre once a month for the data from their interlocks to be uploaded.

While it was always possible for people to abuse the system, Foss said there would be “significant penalties” - including potential jail time - for those who tampered or abused the interlocks.

Cabinet had agreed to provide $4 million of funding to help pay for the devices, as the $2,500 cost to install and run them was one of the main barriers to their use. However, offenders would have still have to pay for the interlocks, with the government funding going towards a subsidy scheme.

Pregnant Pause: Be a hero, take zero

Pregnant Pause, the Australian Capital Territory health promotion campaign that asks participants to take a break from alcohol during their pregnancy or the pregnancy of a loved one, is relaunching with a new radio and television advertising campaign, featuring local media personalities.

The campaign promotes Australia’s national alcohol guidelines developed by health professionals, which state that for women who are pregnant or planning a pregnancy, no alcohol is the safest option.

Pregnant Pause takes a novel approach to making giving up alcohol easier for mums-to-be, by raising awareness of this important health message and building a strong support system that will help women achieve an alcohol free pregnancy.

Pregnant Pause Project Officer at FARE, Ms Kamara Buchanan commented “Nine months, or 270 days, can be a long time to go without alcohol. But expectant mothers don’t have to do it alone. That’s why Pregnant Pause is a campaign everyone can get involved in.

We know around a third of Australian women say they’d be less likely to drink during pregnancy if their partner or spouse also stopped drinking. This is about encouraging Canberrans to support each other and give newborn babies the best possible start in life by pledging to go alcohol free.”

Pregnant Pause is an initiative of the Foundation for Alcohol Research and Education (FARE), and is supported by the ACT Government under the ACT Health Promotion Grants Programme.
AIM – Alcohol in Moderation was founded in 1991 as an independent not for profit organisation whose role is to communicate “The Responsible Drinking Message” and to summarise and log relevant research, legislation, policy and campaigns regarding alcohol, health, social and policy issues.

AIM Mission Statement

• To work internationally to disseminate accurate social, scientific and medical research concerning responsible and moderate drinking
• To strive to ensure that alcohol is consumed responsibly and in moderation
• To encourage informed and balanced debate on alcohol, health and social issues
• To communicate and publicise relevant medical and scientific research in a clear and concise format, contributed to by AIM's Council of 20 Professors and Specialists
• To publish information via www.alcoholinmoderation.com on moderate drinking and health, social and policy issues – comprehensively indexed and fully searchable without charge
• To educate consumers on responsible drinking and related health issues via www.drinkingandyou.com and publications, based on national government guidelines enabling consumers to make informed choices regarding drinking
• To inform and educate those working in the beverage alcohol industry regarding the responsible production, marketing, sale and promotion of alcohol
• To distribute AIM Digest Online without charge to policy makers, legislators and researchers involved in alcohol issues
• To direct enquiries towards full, peer reviewed or referenced sources of information and statistics where possible
• To work with organisations, charities, companies and associations to create programmes, materials and policies built around the responsible consumption of alcohol.

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