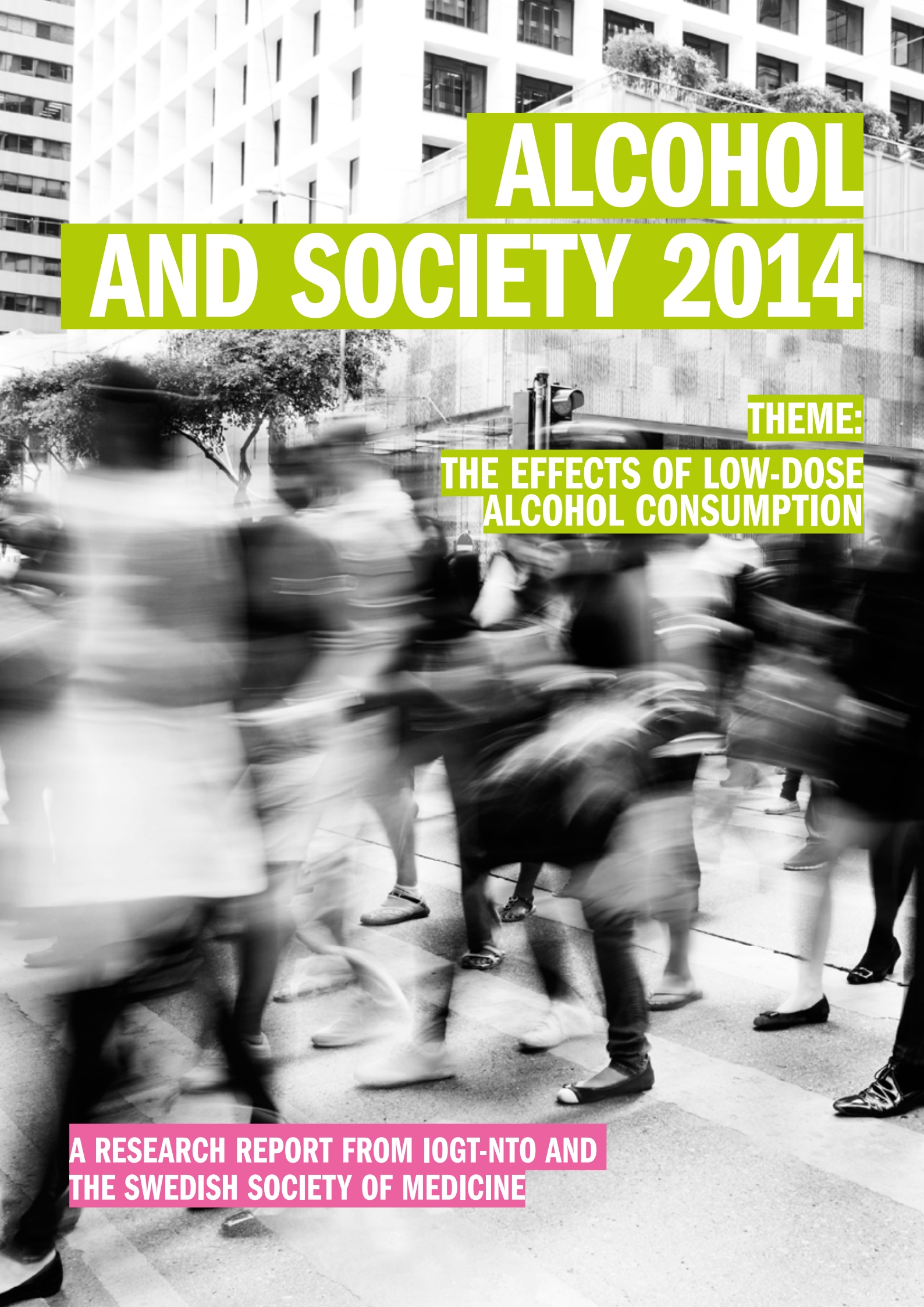


ALCOHOL AND SOCIETY 2014

THEME:
THE EFFECTS OF LOW-DOSE ALCOHOL CONSUMPTION

A RESEARCH REPORT FROM IOGT-NTO AND THE SWEDISH SOCIETY OF MEDICINE



A SWEDISH LANGUAGE VERSION OF THIS REPORT IS ALSO AVAILABLE FROM IOGT-NTO (WWW.IOGT.SE) OR THE SWEDISH SOCIETY OF MEDICINE (WWW.SLS.SE).

© IOGT-NTO & SVENSKA LÄKARESÄLLSKAPET, 2014

GRAPHIC DESIGN: PERNILLA FÖRNES FORM

URN: urn:nbn:se:iogt-2014-aos-en

A RESEARCH REPORT FROM IOGT-NTO AND THE SWEDISH SOCIETY OF MEDICINE

ALCOHOL AND SOCIETY 2014

THEME: THE EFFECTS OF LOW-DOSE ALCOHOL CONSUMPTION

Foreword
By Anna Carlstedt and Kerstin Nilsson 5

Main article
Evidence about health effects of “moderate” alcohol consumption 6

Interviews
Beer is good for the heart! Wine can cause cancer! – Media attitudes towards alcohol research 24
The ultimate curiosity job 28

Latest research
Socioeconomic differences and deaths attributable to alcohol 31
Alcohol and alcohol related harm in China 32
Does the alcohol policy environment have any effect on those who drink excessively? 34
Are alcohol policies effective in low- and middle-income countries? 35
Under the influence – an investigation into the debate on minimum pricing for alcohol in England 37
Does reduction in drinking reduce risk of death for individuals with alcohol use disorders? 40
Does alcohol production and consumption increase economic growth? 43

The report’s authors
Presentation of the research group authors 44

FOREWORD

Our organisations – IOGT-NTO and the Swedish Society of Medicine – are proud to present the second in our series of reports entitled "Alcohol and Society". The concept behind the report is, in many respects, unique. We brought together an international group of leading alcohol researchers and gave them a task. We asked them, on the basis of the theme that we had identified as being both important and topical, namely low-dose alcohol consumption, to review the latest global research in the field, to describe the scientific status, and then to draw conclusions on measures tailored for Sweden and the rest of the Nordic region.

Focusing on low-dose alcohol consumption was, for us, as obvious as it was important. We live in a world where the media switch rapidly, and on a daily basis, between reporting on the supposed health benefits of a glass of wine and issuing warnings about the self-same thing. No other area in the field of alcohol research and alcohol-related media reporting sends out as many mixed signals as this one, and we accordingly asked the researchers to take a comprehensive view of the situation, in order both to analyse the facts and also explain why the conclusions diverge so widely. The results are both unique and interesting. It is our hope that they will provide greater clarity in the Swedish debate and in the media reporting on low-dose alcohol consumption.

We have also, in addition to our main article, interviewed representatives of several of the leading media organisations in Sweden, asking them how they see their role, how they evaluate research findings, and what their position is on contradictory information about the health-related effects of alcohol. And finally, at the end of the report, we touch, as we did last year, on some of the latest alcohol research findings in other areas.

The Swedish version of the report has been written with accessibility in mind. The research it contains is presented in a way that makes it easily accessible by anyone with an interest in public health issues. For a more in-depth view, there is an English language version that also presents the references for the main article. Both language versions of the report are available on our respective websites at the URLs shown on the back cover.

We hope that you will find this report absorbing and that it provides you with valuable information on the latest findings in the field of alcohol research. This is the second year that we have produced an "Alcohol and Society" report and we look forward to presenting it for many more years to come.

Anna Carlstedt

Chair, IOGT-NTO

Kerstin Nilsson

Chair, The Swedish Society of Medicine

EVIDENCE ABOUT HEALTH EFFECTS OF "MODERATE" ALCOHOL CONSUMPTION:

REASONS FOR SCEPTICISM AND PUBLIC HEALTH IMPLICATIONS

By Sven Andréasson, Tanya Chikritzhs, Frida Dangardt, Harold Holder, Timothy Naimi and Tim Stockwell



EXECUTIVE SUMMARY

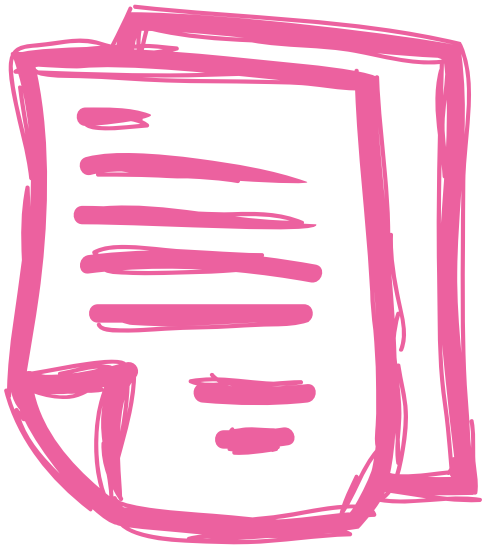
This report summarizes and examines the scientific evidence regarding the health effects of "moderate" (i.e., low-dose) alcohol consumption, and discusses the implications of this research for clinical practice, low-risk drinking guidelines, and alcohol policy development.

The existing evidence finding cardiovascular benefits from low-dose alcohol consumption is weak, and emerging evidence suggests that these protective effects are spurious (i.e., do not exist, or are harmful). The view that alcohol confers health benefits is therefore even less of a valid counter-argument against the adoption of effective alcohol control policies (e.g., those which reduce alcohol's availability and affordability).

- Although alcohol consumption is a leading cause of preventable death and social problems worldwide, previous studies often find an association between low-dose consumption and a reduced risk of cardiovascular (CVD) disease. Despite shortcomings in the science, this information has been promoted extensively, used to argue against the adoption of policies to reduce excessive drinking and led some doctors to advise patients to drink for better health.
- However, there have been no "randomised" studies of low-dose alcohol consumption and any disease or death outcomes to confirm findings from non-randomised studies. Randomised studies are the gold standard used to determine the safety and effectiveness of medical drugs. There are more than 10 recent examples in which conclusions from observational studies were contradicted later by randomised studies (e.g. hormone replacement therapy for the reduction of heart disease in women).
- Laboratory studies have indicated that low-dose alcohol consumption reduces some biological markers of heart disease. However, more recent and sophisticated studies have refuted some markers as causal factors of CVD mortality (e.g., HDL cholesterol). Further, low-dose alcohol consumption is associated with physiological effects that should increase CVD mortality, such as increased blood pressure.
- There are many methodological problems with non-randomised (i.e., observational) studies. Most important among these are confounding and misclassification. Non-drinkers and moderate drinkers differ in many ways besides alcohol consumption. The majority of observational studies classify people as abstainers who have cut down or quit drinking, many of whom have health problems. This makes moderate drinkers appear to be healthier than they really are.
- The observation of apparent health benefits from moderate drinking has also been made for a number of health conditions for which there is no plausible physiological basis (e.g., liver cirrhosis, improved childhood development, cancers, hip fractures, deafness and the common cold), suggesting that protective associations with other conditions may not exist.
- A large international genetic (Mendelian) randomisation study found that having a genetic disposition that causes less drinking is associated with a significantly reduced risk of coronary disease, even among those who consume modest amounts of alcohol.
- Studies of populations that have experienced reductions in total alcohol consumption do not find any evidence of increased rates of cardiovascular disease.
- Even assuming cardiovascular benefits from moderate drinking are real, the WHO estimates are that alcohol causes far more death and disability than it prevents. Further, if real, the optimal mortality benefits apply at very low levels (maximally half a drink per day for women, and less than one drink per day for men) and increase thereafter.
- Physician advice to patients and low-risk drinking guidelines should focus on reducing consumption to safer levels among current drinkers, and should discourage drinking initiation or increased consumption on the basis of health-related considerations
- From the public health perspective, governments should adopt and strengthen effective alcohol control policies to reduce alcohol-related deaths, social problems and economic costs. The growing scientific scepticism regarding evidence about the health effects of low-dose alcohol consumption should further enhance their rationale for doing so.

SECTION 1. INTRODUCTION: "MODERATE" (LOW-DOSE) ALCOHOL CONSUMPTION

Is alcohol good for health? Over the past 40 years a growing list of epidemiological studies suggest that when drunk in "moderation" alcohol is associated with a reduced risk of death from all causes and, in particular, a significantly reduced risk of cardiovascular disease (CVD) [1] and diabetes.[2] On the other hand, a longstanding and much larger literature has made it clear that heavy drinking causes a multitude of medical harms.[3, 4] The list of alcohol related medical conditions has grown over the years and now includes more than 60 major types of health condition, reflecting that the toxicity of alcohol affects all tissues and organs of the human body. Globally, about 3.3 million deaths or 5.9% of all deaths were estimated to be caused by alcohol in 2012. This figure is a net figure estimated after the assumed beneficial effects of low-dose alcohol consumption have been taken into account.



Although alcohol has toxic and carcinogenic properties, this does not necessarily preclude the possibility of health benefits in low doses, as is the case with a large number of pharmaceuticals. A number of mechanisms whereby alcohol could exert a beneficial effect have been proposed, including its effect on blood lipids and blood clotting. An important observation, however, is that the literature on beneficial effects primarily addresses chronic disease while the literature on alcohol's detrimental effects to a large extent involve acute effects such as accidents and violence. Even low doses of alcohol consumption increases the risk for acute harm, e.g. from traffic injuries. Of the traffic deaths in Sweden, 21% are caused by drivers under the influence of alcohol.[5] Low-dose alcohol also increases the risk for several chronic conditions like cancer and hypertension.

Nevertheless, the notion of beneficial effects from low-dose or "moderate" drinking has had a huge impact in the alcohol field, with implications for medical practitioners as well as for policy makers. Almost every time a new study suggesting health benefits has been published, these results have been given good coverage in the media and this appears to have shaped attitudes in the general population regarding the potential risks versus benefits from drinking alcohol. For many practitioners, the message from researchers about positive health effects has caused uncertainty about what advice is appropriate to provide to patients. Even if most practitioners recognize the hazards related to alcohol, they may be hesitant to convey a message that is too restrictive as this might deny their patients a positive health effect. In some cases moderate drinking may be recommended by physicians, even for abstainers.

For policy makers, the question arises as to how to regulate a dangerous commodity where research also suggests positive health effects when this commodity is used in moderation. The message from the alcohol industry is clear: alcohol policies should focus on the minority in the population with problem drinking, offering these individuals treatment, and leave the rest, the majority who are moderate drinkers, alone. This conflicts with the conclusions from alcohol policy research, where policies that reduce total consumption through restrictions on the economic and physical availability have been shown to be more effective in reducing alcohol problems. A challenge to researchers and policy makers alike is the fact that in reality low-dose alcohol consumption does not exist in isolation. There is a strong link between the prevalence of moderate drinking and excessive drinking, where an increase in the former is followed by the latter. Low-dose consumption is not something that we can "choose" as a preferred drinking option for populations, and among developed countries a substantial fraction of drinkers consume alcohol in ways that clearly increase the risk of health and social consequences for themselves and others.

The notion of beneficial effects on CVD mortality from moderate drinking therefore is crucial. One important question is whether the conclusions about these effects from the published literature are in fact correct. Over the past decade a number of doubts have been raised regarding the methodology underlying the studies informing this evidence base, which is comprised entirely of non-randomised studies. It is increasingly being understood that a large part of the beneficial effect of alcohol found in many studies is likely due to a number of methodological limitations, which are discussed below. It is also likely that moderate alcohol use is an indicator of positive health and social well-being. The studies that find beneficial effects all involve asking people questions about their drinking patterns which are then matched with their personal health outcomes. Even if large in number, such studies (often called observational studies) all share the methodological weaknesses that are inherent in this type of research, chief among these is a lack of randomisation of exposure. Critically, there are no experimental studies in which participants are randomly assigned to groups where alcohol is consumed or not consumed

(the control group). Such experimental studies, i.e., randomised controlled studies, are normally required in medicine as a basis for testing an intervention such as a pharmaceutical drug.

SECTION 2. HARMS FROM ALCOHOL CONSUMPTION

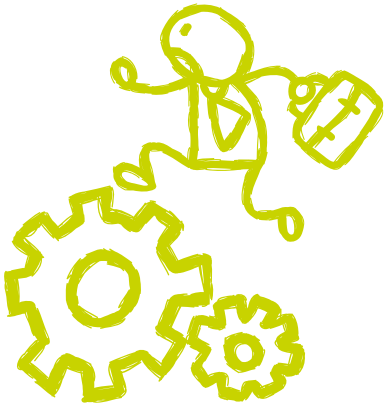
Alcohol is a toxic substance with psychoactive properties and the capability to cause dependence among users along with a variety of other health conditions. As a result, globally, about 3.3 million net deaths were estimated to be caused by alcohol in 2012 (this estimate took into account the assumed beneficial effect of low-dose alcohol consumption).[6] The estimated burden of alcohol-related death, disease and disability has increased in the last decades in WHO sponsored international studies. In 2010, out of more than 60 risk factors, alcohol was ranked as the fifth leading cause of death and disability globally, up from eighth place in 1990.[7]

For people aged 15–49 years, alcohol is the leading health-related risk factor worldwide, followed by tobacco smoking, high blood pressure and high body-mass index.[8] This is greater than, for example, the proportion of deaths from HIV/AIDS (2.8%), violence (0.9%) or tuberculosis (1.7%). Not all of the conditions linked to alcohol are included in these estimates.[9] The estimated negative effect on the global burden of disease from alcohol is more than 30 times as large as the beneficial effect.[10]

The proportion of alcohol-attributable burden of disease is highest in the WHO European Region (12.8 %). In high-income countries within Europe, such as Sweden, there is a much higher alcohol-attributable disease burden compared to alcohol-attributable deaths because of the disabling impact of alcohol use disorders.[11]

2.1. Harms from Chronic Health Conditions

Alcohol produces a large chronic disease burden as a necessary cause of a large number of specific conditions such as alcoholic liver cirrhosis and fetal alcohol syndrome. In addition it is a contributing causal factor in a large number of other disease conditions, such as cancers, cardiovascular disease, and infectious disease.[12]



Alcohol has been classified as carcinogenic to humans since 1988 by the WHO International Agency for Research on Cancer, IARC.[13] In 2007 two new reviews on alcohol and cancer were published, one by IARC and one by the World Cancer Research Fund / American Institute for Cancer Research. Both reviews concluded that alcohol not only caused malignant tumours of the oral cavity, pharynx, larynx, oesophagus and liver, conditions that were linked to alcohol already in the 1988 report, but that alcohol also was a cause for colorectal and female breast cancer.[14, 15] As breast cancer and colorectal cancer are two of the most common cancers worldwide, the proportion of cancers attributable to alcohol consumption became higher than previously estimated.

The cancer risk from alcohol increases with the amount of ethanol drunk, in the absence of any threshold below which no effect is evident.[16, 17] For example, the relative risk of breast cancer is estimated to increase with increasing alcohol intake by about 10% per 10 g per day.[18] Among other disease categories, alcohol is directly responsible for between 4% and 25% of the disease burden related to specific cancers worldwide. Alcohol consumption also contributes to about 10% of the disease burden due to tuberculosis, epilepsy, haemorrhagic stroke and hypertensive heart disease in the world.

For the majority of diseases linked to alcohol the risk increases with increasing consumption without a threshold under which there is no increased risk.[19] A meta-analysis from 2004 [20] concluded e.g. that the risk of hypertension increased by 43 per cent for a consumption of two standard drinks per day. For the same level of consumption the risk for haemorrhagic stroke increased 19 per cent, and the risk for liver cirrhosis was almost three times greater, compared to non-drinkers.

2.2 Harms from Acute Health Conditions

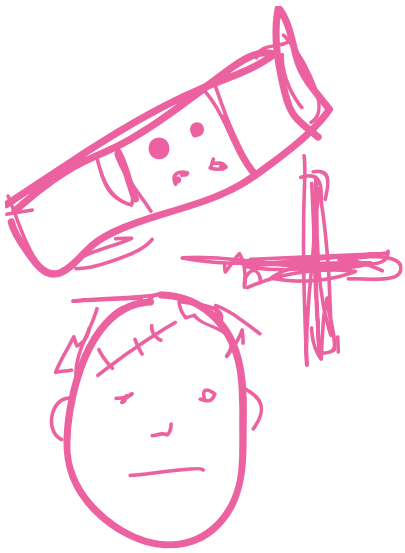
The effects of alcohol consumption are not only confined to chronic diseases arising from long-term exposure but also increase the risk for acute conditions which typically occur from acute intoxication with alcohol. Alcohol is a psychoactive substance which produces specific in-the-moment impairment for hand-eye coordination, depth perception, general judgment, and reflex response. As a result, alcohol is involved in a number of acute harms even at low dosage which require specific skills and responses including operating automobiles, boats, machinery, and other complex tasks. A recent German experiment found that low-dose alcohol had a greater impairment on attention performance for adolescents compared to adults on the same test.[21] These results suggest that low-dose drinking by youth can result in more impairment in complex tasks like driving or operating machinery than for adults. A significant number of personal injuries and violent events are associated with

alcohol impairment. The impairment of the drinker also has serious social and economic consequences for individuals other than the drinker, e.g. assaults, traffic crashes, property damage, domestic violence, and child neglect or abuse.

That drinking to intoxication increases risks for injuries is well-known, however, low-dose consumption also increases the risk for several types of injuries. In Sweden, like most developed countries in which driving is a major mode of travel, alcohol-impaired drivers cause a substantial number of traffic deaths.[22] Experiments with alcohol consumption in connection with driving have shown that a BAC of 0.03% significantly increases stop distance and the ability to avoid obstacles.[23] A review in 2004 of studies from the past fifty years came to the conclusion that there is no evidence of a threshold below which impairment do not occur and that significant impairment occurs at very low BAC, below 0.02 %.[24]

Low-dose alcohol consumption also increases the risk for injuries other than those that are traffic-related. A study from a Swiss hospital emergency department of all types of injuries found that of alcohol related injuries, acute low-dose consumption (one unit or fewer of alcohol for women and two units or fewer for men) was related to 50 per cent of transport injuries, 44 per cent of falls, 50 per cent of exposure to forces and other events and 24 per cent of injuries from interpersonal violence. As percentage of all injuries, low-dose alcohol consumption was related to 21.5 per cent of transport injuries, 22 per cent of falls, 21 per cent of exposure to forces and other events and 16 per cent of injuries from interpersonal violence.[25] A recent Norwegian study, found that, the risk of an alcohol-related injury increases linearly with frequency of binge drinking.[26]

While alcohol has been often linked to interpersonal violence in naturalistic observational studies, there are recent laboratory experiments which suggest a linear dose-response relationship up to a high dose of 1.0g/kg relationship between alcohol and aggression.[27]



SECTION 3. REASONS FOR SCEPTICISM ABOUT THE EFFECTS OF LOW-DOSE ALCOHOL

3.1. Limitations of existing observational studies, lack of randomised trials

Most of the studies that provide evidence for health benefits associated with moderate drinking involve the observation of a group of individuals followed up over a number of years. There are no control groups as in an experimental study rather, people are compared according to various behaviours such as their diet, substance use and exercise habits and/or on the basis of characteristics such as gender, socio-economic status and ethnicity. Such "observational studies" on their own can identify associations between potential risk factors and disease outcomes over time but are not generally sufficient to prove causation. Thus, in studies of alcohol consumption and disease, observed associations can be caused by a variety of other lifestyle, psychosocial, genetic and physiological factors each of which may be independently associated with alcohol consumption in the population being studied. The strongest scientific evidence for causal relationships is generally accepted to be from randomised controlled trials (RCT), where potential confounding factors can be reduced by randomising participant exposure to a potential factor like drinking alcohol at a particular level and then comparing them to a control group who are not exposed to that factor.

In alcohol research, however, there has not been any RCT involving alcohol that assesses a morbidity or mortality outcome/endpoint. One reason for this lack of RCTs is that there are substantial practical and possibly even ethical problems with randomising individuals to drink or not drink over a period of many years. Regardless, this represents a major limitation in the evidence base about the effects of low-dose alcohol consumption. Other sources of evidence for the existence of causal relationships will be discussed here including: (i) the use of laboratory experiments to identify the impacts of low-doses of alcohol on biological markers known to be risk factors for disease; (ii) the study of the impact of random genetic variations in the population; and, (iii) the impact on disease outcomes of population level changes in exposure to alcohol consumption.

Although many observational studies have found a J-shaped curve in which people with low average alcohol consumption have lower mortality from all causes than people who do not drink at all, it is important to remember that even a large number of consis-

tent observational studies showing similar outcomes can be consistently wrong. Indeed, findings from even well done observational studies with plausible biologic hypotheses may differ from those of randomised controlled trials, and these differences are believed to be partly due to residual or unmeasured confounding.[28]

OBSERVED HEALTH BENEFITS CONTRADICTED BY CONTROLLED STUDIES

Hypothesis	Observational	Randomised controlled trials
Beta carotene protective for cancer and CVD	Yes	No
Vitamin E protective for dementia and CVD	Yes	No
Hormone replacement therapy protective for coronary heart disease	Yes	No
Bisphosphonates protective for post menopausal breast cancer	Yes	No
Omega 3 fatty acids protective for diabetes	Yes	No

Considering the much wider literature on the interpretation of observational studies examining the psychosocial and behavioural risk factors for diseases, it is most often found that frequently observed associations are confirmed in randomised controlled trials.[29] However, this is not always the case and some notable exceptions have been recorded in which multiple observational studies appear to have been biased to produce misleading conclusions. For example, many observational studies suggested that increased beta carotene intake might be associated with reductions in CVD and cancer, that hormone replacement therapy and vitamin E supplementation were associated with reductions in CVD and dementia, and that Chlamydia infection was associated with atherosclerotic heart disease. However, beta-carotene, vitamin E, hormone replacement therapy, and antimicrobial treatment for Chlamydia were found to be ineffective when subjected to randomised trials.[30, 31, 32, 33, 34] Hormone replacement therapy offers a particularly striking example, since multiple well-done observational studies by eminent epidemiologists suggested 40% reductions in coronary heart disease, and no effect was found when RCTs were conducted.

Recently statins, which have been used in medicines to lower cholesterol levels used to prevent cardiovascular disease, have been added to this list. Observational data have shown statins to have a beneficial effect on acute respiratory distress syndrome and chronic obstructive pulmonary disease but this has now been disproved in randomised controlled trials.[35] Another recent example is the use of bisphosphonates associated with a substantially decreased risk of breast cancer found in several observational studies. In the RCTs, contrary results were found, showing 3 to 4 years of bisphosphonate treatment did not decrease the risk of invasive postmenopausal breast cancer.[36]

While it may be challenging to conduct population level trials in which individuals are randomised into drinking alcohol or abstaining over long periods, two other main approaches have been employed to investigate whether the observed health benefits are causally related to moderate alcohol consumption. One has been to conduct laboratory experiments over relatively short time periods in which individuals are randomised to receive measured doses of alcohol or to abstain under controlled conditions. Biological measures believed to be indicative of cardiac health and functioning have been used as the main outcome measures. Another more recent approach is known as Mendelian randomisation in which the observed effects of genetic variations between individuals can be considered equivalent to a randomised controlled trial. In the field of alcohol and health studies, this has become possible through the identification of a genetic variation thought to be uniquely associated with abstinence or greatly reduced alcohol consumption. The results of these studies will be summarised below along with a number of other methodological concerns which need to be considered when interpreting the large and apparently compelling literature of studies connecting improved health outcomes with moderate or low volume alcohol consumption.

Confounding

Confounding (i.e., when a factor that is associated with both the exposure and the outcome, but when the factor is not in the causal pathway between them) is an important threat to validity for observational studies that can lead to erroneous associations between an exposure (i.e., low-dose alcohol intake) and disease outcomes (e.g., cardiovascular disease). If the health of moderate drinkers is to be compared to that of non-drinkers in order to determine the effect of alcohol, a meaningful comparison would require that the two groups be generally similar in most respects other than alcohol consumption. However, evidence demonstrates that confounding is a serious problem in studies of alcohol consumption and cardiovascular disease conducted among Western populations. Specifically, studies from Europe and North America find that among non-drinkers most traditional cardiovascular risk factors are more prevalent and intense among non-drinkers compared to those who drink moderately, particularly those who drink small amounts frequently.[37, 38, 39, 40, 41, 42] Assuming that the differential distribution of many of these factors is not the result of alcohol consumption (or lack thereof), these risk factors represent poten

tial confounders that could make low-dose alcohol consumption appear to be protective for cardiovascular disease.

While most analyses try to adjust for these differences, studies do not always collect information about relevant potential confounders, including "traditional" cardiac risk factors.[43] However, even in well-controlled studies, the disproportionate number and intensity of risk factors associated with non-drinking status means that the threat of residual confounding is high (i.e. confounding that persists even after attempts to control for it in analyses) and likely to bias studies in favour of moderate drinkers such that they appear in better health. Furthermore, those with more risk factors have more possible combinations of risk factors that could be synergistic in terms of risk. To the extent that synergistic risk (i.e., independent additional risk beyond the sum of independent risks) is not captured in observational studies, this would again bias studies in favour of moderate drinkers. Finally, because coronary heart disease risk factors tend to cluster in certain individuals and populations, it is plausible that unknown or unmeasured confounders may be more prevalent among non-drinkers than those with low average alcohol consumption, which could again favour apparent reduced risk among moderate drinkers.



In addition to the distribution of traditional cardiac risk factors, low-dose alcohol consumption appears to be a marker of "non-traditional" socio-economic factors such as affluence, leisure, education, mental health, and dentition.[44, 45] These non-traditional risk factors are major determinants of mortality, and few of these factors are plausibly caused by alcohol consumption itself.[46] Since there is no likely causal relationship between, for example, drinking alcohol and having previously achieved higher educational attainment, it seems likely that moderate drinking is merely a reflection or result of prosperity and wellness, rather than its genesis. This makes non-traditional risk factors a rich source of confounding that could distort the apparent relationship between alcohol consumption and health outcomes. Unfortunately, few surveys that include alcohol consumption also include questions about many of these non-traditional risk factors, which make it difficult or impossible to account for them statistically.

Findings from Sweden are consistent with the notion that non-drinkers have more risk factors and worse health at baseline. A study of Swedish women found that abstainers and occasional drinkers had lower levels of education, more use of psychotropic drugs, and were more likely to receive a disability pension.[47] In terms of mortality, non-drinkers had a significantly increased risk for death compared to moderate drinkers, but after accounting for household composition, level of education, employment, social network, smoking, regular medical control for a physical or mental disease, hypertension and diabetes, nondrinking was no longer a risk factor for death. The authors concluded that this underlines the importance of including health status at base-line when prospectively studying the association between alcohol use and mortality, otherwise moderate alcohol consumption may appear more beneficial than is the case.[48]

Among Swedish abstainers, two groups can be distinguished: those who abstain for reasons of principle (e.g., religion, healthy lifestyle, social solidarity, etc.) and those whose abstinence is related to economic hardship, social isolation or health.[49] A Swedish conscripts study found that nondrinking conscripts deviated from moderate drinkers on a number of psychosocial measures. The study population consisted of young men, mostly aged 18 to 19 years. Abstainers compared with moderate drinkers had lower emotional control, felt more insecure in the company of others, reported being less popular in school, had fewer friends, and were more anxious. U-shaped curves were produced when indicators of poor sociability were depicted in relation to level of alcohol consumption. Abstainers also had more psychopathology than moderate consumers.[50]

Because of the heterogeneity of non-drinkers, it may be most relevant to examine the risk of death among those who abstain for religious or family reasons rather than for reasons related to poor health or economic deprivation. A U.S. national survey of more than 40 000 adults aged 21 years or greater obtained reasons for not drinking among abstainers. People stating that the main reason for not drinking was "have responsibility to my family", "religious or moral reasons" or "don't socialize very much" had an equal risk of death as current drinkers with a consumption of less than one drink per day. People stating reasons as "do not like alcohol", "am an alcoholic", "thought I might become an alcoholic", "medical or health reasons" and "costs too much" had a higher risk of death.[51, 52]

Corroborating concerns about confounding, there are several diseases for which low-dose consumption has an implausible protective association for which convincing biological mechanisms have not been proposed. These include protective effects of low-dose alcohol for deafness, hip fracture, asthma, the common cold, and overweight.[53] Moderate consumers of alcohol have even been shown to have lower risks for conditions like alcoholic liver cirrhosis[54] and cancer[55] than non-drinkers, despite the fact that alcohol is a leading cause of cirrhosis and that alcohol is recognized as a human carcinogen.

Another study on self-reported health of adults and children living in the same family in a national representative US sample from 2008 to 2010 has highlighted the importance of residual confounding as a major source of misleading results. The study found that family members including children who co-habited with light to moderate drinkers but who were not necessarily themselves drinkers had better health than abstainers.[56] This 'shared' protection is unlikely to be due to physiologic effects from alcohol, particularly in relation to those under 18 years (most of whom would have been non-drinkers). Rather, the finding is more likely to be explained by shared socioeconomic and lifestyle characteristics.

Furthermore, several studies have found that offspring of mothers who consumed small amounts of ethanol during pregnancy have better developmental outcomes compared with offspring of mothers who abstained from drinking during pregnancy. This is likely a result of residual confounding remaining after attempts to control for the markedly privileged socio-economic status of low-volume drinking mothers, particularly since ethanol is the world's leading fetal neurotoxin.[57]

That people in southern France seem to have lower rates of heart disease in spite of eating food rich in fat and drinking alcohol, mainly wine, has been called 'the French paradox' - a well-publicized phenomenon. However, in a recent study randomised with respect to the Mediterranean diet but not in relation to alcohol consumption found that those consuming the Mediterranean diet had a lower risk of cardiovascular mortality. This shows that the apparent cardiovascular benefit asserted by the 'French paradox' can be explained by diet, irrespective of alcohol consumption.[58] Another explanation of a large part of this seeming paradox may be the coding practices of French doctors, who have been shown to overuse non-specific codes for cardiovascular disease referred to as 'garbage codes', which has the effect of artificially lowering the reported prevalence of ischaemic heart disease per se.[59]

Misclassification of drinkers and abstainers

In the classic studies, the relationship between drinking level and risk of disease or death is described as a J shaped curve, where "moderate" drinkers are observed to have a lower risk than people classified as

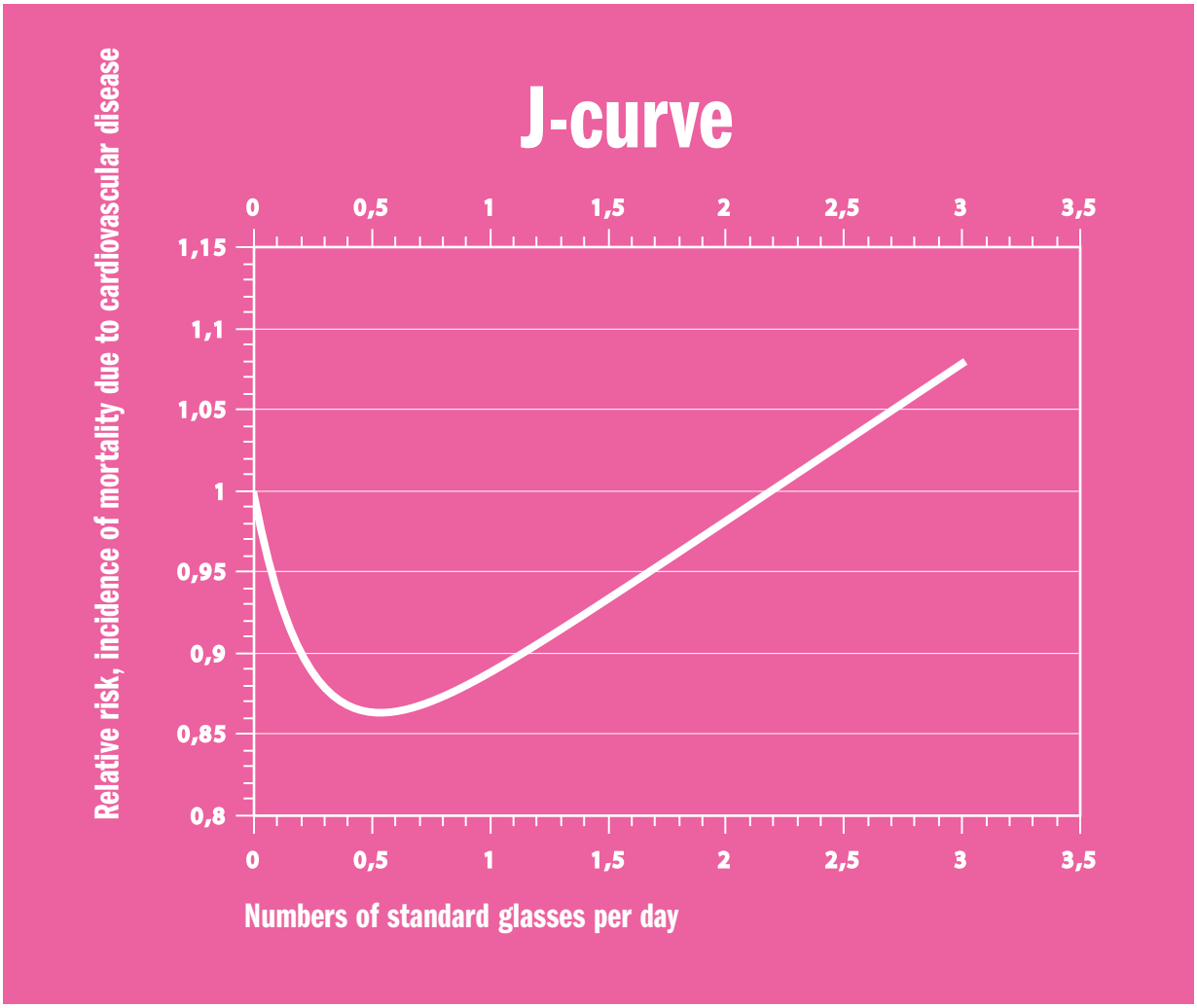


Figure 1. A hypothetical example of a J-curve. The risk for abstainers is set at 1, and falls for low-dose consumers, rising in conjunction with increased consumption.

"abstainers" but those drinking at heavier levels have higher risk than abstainers.

Numerous problems can arise in these studies, however, in relation to how accurately different studies classify who is an abstainer and who is a moderate drinker. Failure to make this classification accurately can lead to biased comparisons between these two groups. Mostly, such biases cause the people identified as moderate drinkers to appear healthy in comparison with those classified as abstainers. The best-known example of this is sometimes referred to as the "sick quitter effect" whereby former drinkers are mixed in with lifetime abstainers. Because people who give up alcohol have significantly worse health profiles, this procedure contaminates the abstainer reference group and makes the moderate drinkers "look good" by comparison.

There are several other examples of drinker misclassification errors which can bias this literature. In many developed countries it has been observed that as people age and become increasingly frail they also tend to either completely abstain from drinking or cut right down and become occasional drinkers. The common practice of combining these near abstainers into the abstainer reference group could therefore also create bias by making this reference group less healthy and hence moderate drinkers appear more healthy by comparison. A well-known review of this literature by Fillmore et al (2006) [60] attempted to identify all the studies which contained either former or occasional drink bias i.e., the abstainer reference group included former and/or occasional drinkers. They reported that among the relatively few (seven) studies which did not contain such bias there was no longer evidence of reduced mortality risk among moderate drinkers.

It is important to stress that misclassification errors are the rule rather than the exception among studies of the relationship between alcohol consumption and health. Specifically in relation to the literature on cardio-protection and moderate drinking, Stockwell and colleagues (2012) [61] examined the 84 studies used by Ronksley et al (2011) [62] in their influential meta-analysis with a view to identifying how many contained serious methodological problems including misclassification evidence. As illustrated in Figure 2 below, after eliminating studies that were duplicates, that did not control for basic lifestyle confounding factors such as smoking and that did not adequately measure both quantity and frequency of alcohol consumption, only 49 studies remained. Among these 49, 32 contained former drinker bias and a further seven contained occasional drinker bias i.e. former and/or occasional drinkers were included in the reference group of "abstainers". A further eight studies contained "reverse occasional drinker bias" whereby occasional drinkers were grouped with moderate drinkers which is also capable of biasing comparisons with abstainers. The two remaining relatively error-free studies produced

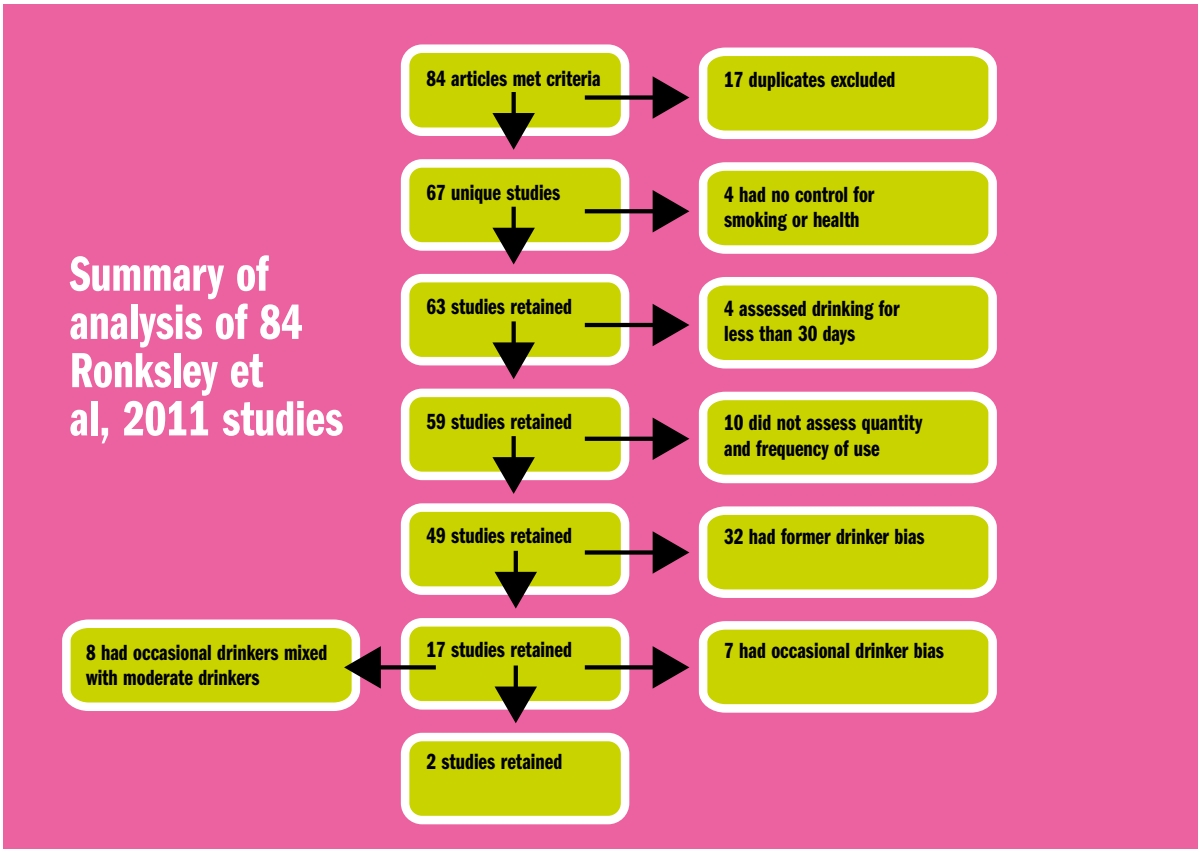


Figure 2. From Stockwell et.al. (2012), How good is the science. BMJ 2012, 344:e2276, reference 61.

inconsistent findings in relation to the presence of cardio protection.

Another perspective on the "sick quitter effect" or former drinker bias, is that it is insufficient to separate out these former drinkers into their own group and report mortality outcomes for them separately from people who continue to drink. Over the course of these long-term observational studies, arguably this just result in weeding out sick people with poor outcomes from different groups of drinkers including those classified as "moderate". Liang and Chikritzhs (2013) [63] pursued this idea and investigated what happens to the J shaped curve when former drinkers are classified into different groups of current drinkers. Since past drinking status of former drinkers is rarely known or recorded in cohort studies, Liang and Chikritzhs obtained an estimate of this by using multiple imputations, a common strategy used for dealing with missing values. They demonstrated that when former drinkers were placed back into drinker groups, evidence of health protection at light and moderate levels was substantially diminished.



Liang and Chikritzhs (2013) [64] argue that this reallocation of former drinkers is necessary and is akin to the problem of dealing with missing cases in randomised clinical trials. If either the treated or untreated individuals in such studies are more likely missing at follow-up this will create bias. An "intention-to-treat" approach is recommended to deal with this problem so that missing cases are included in the final analysis, both of the treated and untreated groups. It is suggested that to be analogous with a clinical trial approach and to avoid bias, data on former drinkers should be replaced back into a drinking category which best describes their past level and pattern of alcohol exposure. This methodological problem has also been recognised in tobacco research where it has been emphasized that ex-smokers and current smokers should be combined in analyses rather than being treated as two distinct groups.[65]

Epidemiological studies rely heavily on accurate participant recall, that is, an individual's ability to faithfully report the quantity and frequency of their own drinking. Unfortunately, recall bias, whereby people (invariably) underestimate their drinking, is well documented in relation to self-reported alcohol consumption.[66] This is a particular problem for studies of alcohol and chronic disease as it undermines the ability of researchers to correctly classify drinkers and non-drinkers. A study based on the 1958 British Birth Cohort provides a striking example of the extent of recall bias in relation to alcohol exposure. Caldwell et al. reviewed respondent's own reports of their current alcohol use (e.g. non-drinker, occasional drinker, drinks on most days) at ages 16, 23, 33, 42 and 45 years. Remarkably, some 60% of 45 year old respondents who self-identified as 'never' drinkers had actually reported drinking alcohol at any one of the previous follow-up surveys -- almost a quarter had previously reported drinking at least once a week. Moreover, almost 60% of 45 year olds who self-reported as lifetime 'occasional only' drinkers had previously reported drinking at least once a week.[67]

Although misclassification is an important type of selection bias, there are other types of selection bias that confront studies of alcohol consumption. After one begins drinking one might become a moderate drinker, quit drinking, become a heavy drinker, or die prior to the study's inception. In observational studies, these last three possibilities are not taken into account. Quitters have been discussed previously, but even if former drinkers are correctly classified as such, removing them from the analysis biases results in favour of moderate drinkers because former drinkers are generally unhealthy, regardless of whether they stopped due to the effects of alcohol or for other reasons. However, from an intention-to-treat perspective, their poor outcomes should rightly accrue to drinkers. In addition, heavy drinkers are ignored since they do not meet inclusion criteria as moderate drinkers. Not including those who already died also biases results in favour of drinking, assuming that deaths at young ages among drinkers are more likely to be from alcohol than they are to be from not drinking among non-drinkers. This is likely the case, since alcohol consumption is a leading cause of death among young and middle aged persons, and because protective effects of alcohol are not observed in young age groups.

Publication bias

Another concern with this literature is that bias may be present in terms of which types of studies are more likely to get published. It is known to be harder to publish studies with no significant results and studies finding that alcohol in moderation is good for you may be more likely to be published. It has also been suggested that wealthy commercial groups with an interest in the sale of alcoholic beverages are more likely to fund researchers with a track record of using methods which detect health benefits from drinking. Evidence to support this concern was reported in one of the major early reviews of this literature by Corrao and colleagues.[68] They concluded that among smaller studies, estimates of mortality risk among moderate drinkers were skewed significantly downwards away from mean values.

3.2. Contradictory lines of evidence from epidemiological studies

Increases in aggregate per capita alcohol consumption are not associated with reduced CVD

Aggregate-level studies can eliminate bias in individual-level studies, and it is important to weigh together evidence from various sources of data: clinical, observational as well as aggregate data, rather than to rely on one kind of data only. For example, if there is a substantial cardioprotective effect of low-dose drinking, mortality rates would be expected to respond to changes in aggregate consumption. If there is no aggregate-level effect on cardiovascular disease of changes in drinking, the explanation could very well be that the cardioprotective effect is the result of confounding in individual-level studies or that it is too small to be of much interest from the point of view of public health.[69]

It is well established from international studies that changes in per capita alcohol consumption are significantly and positively correlated with corresponding changes in rates of alcohol-related diseases. One study of 14 European countries spanning over 45 years of data found that this held for a number of specific outcomes such as liver cirrhosis and injury rates as well as for total alcohol-related mortality.[70, 71, 72, 73] However, no relationship was found between per capita alcohol consumption and rates of cardiovascular mortality.[74] A similar study on Canadian data from 1950 to 1998 reported an increase of IHD mortality of one per cent for a 1-litre increase in per capita consumption, but the estimates did not reach statistical significance. The study concludes that an increase in overall alcohol consumption is more likely to cause an increase in IHD mortality than to lower the number of IHD deaths.[75] A study on data from US states from 1950 to 2002 found a similar effect on total consumption of alcohol, i.e. an increase of one per cent IHD mortality per litre of alcohol.[76] A study of Norwegian time series data from 1955 to 1977 reported a protective effect of alcohol bordering on statistical significance between per capita alcohol consumption and ischaemic heart disease mortality only in the age group 60-74 years.[77] This effect was only apparent the same year as per capita consumption changed. A study from Hong Kong in connection with a decrease of excise taxes on beer and wine by fifty per cent in March 2007, reported that ischaemic heart disease mortality increased by 18% for elderly men and 15% for elderly women. Alcohol duty on beer and wine was eliminated one year later, in March 2008, but this was not found to have impacted the CVD death rates.[78]

Several aggregate population level studies have reported an increase in total mortality related to an increase in alcohol consumption. In a study of 25 European countries between 1982 and 1990, an increase in consumption of one litre of pure alcohol increased total mortality by 1.3 per cent.[79] A study of European countries demonstrated that mortality significantly increased with increasing consumption in eight out of 14 countries. The effect on mortality tended to be stronger in low-consumption countries (3% per litre) than in medium- and high-consumption countries (1%). In no country were increases in consumption significantly associated with decreased mortality.[80] A similar effect was found for Canada where for every one litre increase in per capita consumption there was a 1.7 per cent increase in total mortality.[81]

While measures of per capita alcohol consumption of the population do not discriminate between light, moderate or heavy drinking, some authors have suggested that there may be cardiac benefits for heavy as well as moderate drinking.[82, 83] If this was the case, arguably increases in per capita alcohol consumption should result in reductions in cardiovascular disease but this has been shown not to occur. These findings weaken the argument that alcohol exposure is causally related to reduced risk of mortality.

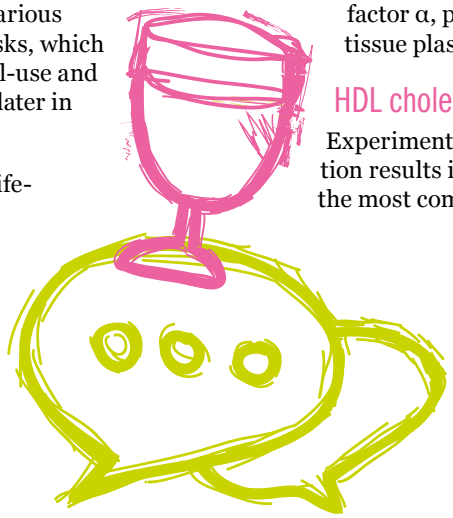
No CVD benefit from low-dose consumption among non-white and non-Western Cultures

Several studies report no reduced risk for heart disease or mortality for non-white or non-western cultures. In a study by Flavio Fuchs the risk for coronary heart disease increased linearly for black American men but decreased for white male consumers of low doses.[84] A study on different ethnic American groups found a reduced risk for mortality only in whites but not in blacks or Hispanic.[85] Similarly in studies on Chinese or Indian alcohol consumption, cardiovascular disease is reported to increase already at low-dose consumption of 1-6 drinks per week.[86, 87, 88] This raises the question whether not alcohol consumption, and especially moderate consumption, is a sign of healthy living, rather than the cause of reduced risk, a connection that is present in some cultures but not in others. This is in agreement with the positive effects of fish oil consumption and other dietary elements on mortality and cardiovascular disease seen in large observational studies, an effect not easily repeated in randomised controlled trials.[89]

Importance of assessing cohorts over the life course, ideally soon after drinking initiation

Another line of concern has emerged from a recent and thorough investigation of a large cohort (n=400,000+) from the European Prospective Investigation into Cancer (EPIC) reported by Bergmann et al.[90] This study recognized the fact that over the life course individuals have competing risks of death from different causes. Earlier in life there is a greater risk of death from injuries while death from cardiovascular disease tends to occur much later in life. By only examining risk of death from one particular outcome at a time and ignoring these competing risks, biased results may emerge. If drinkers are more likely to die from alcohol-related causes earlier in life it may give the appearance that risk of death from a different cause such as coronary heart disease later in life is produced for surviving

drinkers. This problem is exacerbated based on the length of time that elapses between the age of drinking initiation and the time in which a study group or cohort is identified, or by having study populations that are relatively older in age. After conducting their competing risk analysis the authors of the study concluded: "The apparent health benefit of low to moderate alcohol-use found in observational studies could therefore in large part be due to various selection biases and competing risks, which are related to both lifetime alcohol-use and risk of disease, usually occurring later in life."



In a US study the relationship of life-course drinking patterns to diabetes, heart problems, and hypertension among those 40 and older in the 2005 was assessed. Normally, studies only take into account current drinking as reported by those who participate. The study did not find evidence of a protective effect of life-time moderate drinking on heart problems or hypertension, nor did it find evidence of increased risk for heart problems among lifetime heavy drinkers. The results did confirm previous findings of a protective effect of lifetime moderate drinking on diabetes risk.[91]

Contradicting examples exist also for diabetes. A study on a national representative sample of adolescents in the USA reported that adolescents with a frequent heavy alcohol use (consuming an average of 5+ drinks on 3 or more days/week) was 12 times more likely to develop diabetes than abstainers.[92] In a review of seven cohort studies on Japanese, alcohol consumption, even at low doses were linked to an increased risk of diabetes. The effect was larger for men with a relative low BMI (BMI <= 22). For higher BMI the results varied. Some of the studies reported a lower risk for diabetes and some a higher risk.[93]

3.3. Some biological mechanisms supporting the plausibility of cardiovascular protection are now in question, or are inconsistent with epidemiologic studies

Biological mechanisms that can be the cause of a correlation found in observational studies strengthen the plausibility that an observed correlation is causal, e.g. the correlation between low-dose alcohol consumption and coronary heart disease. Such mechanisms have been found and a review and meta-analysis of 44 RCT laboratory studies found that moderate alcohol consumption had favourable effects on levels of the good cholesterol high density lipoprotein (HDL) cholesterol, apolipoprotein A1, adiponectin, and fibrinogen. The study concluded that the results strengthened the case for a causal link between alcohol intake and reduced risk of coronary heart disease. The analysis found that alcohol consumption did not affect a number of other factors also associated with risk of coronary heart disease, as total cholesterol, low density lipoprotein (LDL) cholesterol, triglycerides, Lp(a) lipoprotein, C reactive protein, interleukin 6, tumour necrosis factor α, plasminogen activator inhibitor 1 and tissue plasminogen activator antigens.[94]

HDL cholesterol

Experimental studies show that alcohol consumption results in increases in HDL, and this has been the most compelling source of biologic plausibility for how alcohol might reduce CVD. However, the cardioprotective effect of HDL has recently come into question. First, a meta-analysis of studies on the use of statins and other lipid-lowering drugs, has shown that these medicines have no independent effect on CVD mortality after controlling for their effects on the "bad cholesterol" (i.e., LDL cholesterol).[95] Second, pharmaceutical drugs that raise HDL levels have not resulted in decreases in CVD mortality.[96] And third, Mendelian randomisation, in this case focusing on a genetic effect that raises HDL in the blood, study results have also shown no effect of higher HDL levels for reducing the risk for myocardial infarction.[97]

Coronary calcification, carotid intima-media thickness

Serum biomarkers such as HDL are thought to work through improvements in vascular health. However, histologic markers of vascular health are a more proximate indication of vascular health than serum biomarkers, and alcohol consumption is associated with worse vascular health by these measures. For example, coronary calcification was measured over 15 years in a sample of 3,037 U.S. participants aged 33–45 years. It was found that coronary calcification was associated with increased rates of atherosclerosis at all levels of consumption. Among those consuming less than 7 drinks per week the risk was increased by 10 % compared with abstainers, was 50% higher among those drinking on average 7 to 14 drinks per week and 100% higher for those drinking more than 14 drinks per week. Among binge drinkers the risk was also doubled. The lowest proportion of participants with coronary calcification was found among lifetime abstainers.[98] Similarly, a study on Finnish data on carotid intima–media thickness (IMT), a marker of subclinical atherosclerosis, in young, healthy adults found a direct positive relationship between alcohol consumption and carotid intima–media thickness, with a significant increase starting from consumption of less than two drinks per day compared to non-drinkers.[99]

Brain atrophy, cognition

A number of epidemiological studies find that low-dose alcohol consumption is associated with better cognition. However, a study on middle-aged US men found that each additional drink per week was associated with increased brain atrophy as measured by MRI imaging.[100] In addition, two recent Mendelian randomisation studies found no association between alcohol use and improved cognitive ability.[101, 102] Collectively, these findings further strengthen the notion that the observed apparently beneficial effects on cardiovascular health among low and moderate drinkers may actually reflect good cognitive health, rather than cause good cognitive health.

Blood pressure, hypertension

Although a number of epidemiologic studies find a linear relationship between alcohol consumption and blood pressure and hypertension,[103] others find a J-shaped curve in which low-dose alcohol consumption is associated with lower blood pressure.[104, 105, 106] However, in a meta-analysis of Mendelian randomisation studies of a gene related to reduced alcohol consumption, alcohol consumption increased blood pressure and the risk of hypertension among men, even at moderate levels of consumption. The researchers were also able to estimate that for men, the lifetime effect of drinking 1 g of alcohol a day (1/12 standard drink in Sweden) increased systolic blood pressure by 0.24 mmHg. No association was found in females, for whom drinking levels were low in the studies.[107]

3.4. New genetic evidence: Mendelian randomisation study suggests CVD harm, not benefit, from alcohol consumption, even at low doses

In the absence of clinical RCTs, genetic (Mendelian) randomisation studies are perhaps the strongest available study design to assess the effects of alcohol consumption, particularly for chronic disease-related outcomes. The distribution of genetic variants is usually random in a population, and can therefore minimize the leading sources of bias encountered in observational studies. Furthermore, because genes are present from birth, they can better capture the effects of lifetime exposures to a particular factor.

Although this type of study has been used to assess the relationship of alcohol consumption with health-related conditions and risk factors (e.g., blood pressure, HDL, cognition), it is only very recently (July, 2014) that this study design has been applied to cardiovascular mortality.[108] This is important since CVD is the driver behind the possible mortality benefit among those who consume modest amounts of alcohol.

REASONS FOR SCEPTICISM ABOUT EFFECTS OF LOW-DOSE ALCOHOL CONSUMPTION

- Benefits have not been confirmed in controlled studies
- Other observed health benefits have not been confirmed by RCTs
- Biological mechanisms for health benefits recently disconfirmed
- Evidence for adverse physiological effects of low-dose alcohol
- “Moderate” drinkers have generally healthier lifestyles than abstainers
- Many studies systematically exclude unhealthy drinkers
- Most studies misclassify unhealthy ex-drinkers as abstainers
- Unlikely health benefits observed e.g. liver cirrhosis, cancer, deafness
- Benefits usually observed only in Caucasian populations
- Genetic disposition to drink less provides reduced coronary risk
- Studies showing benefits are more likely to be published
- Reduced population drinking is not associated with increased CVD

Individuals with genetic variants of alcohol metabolism genes that are associated with less alcohol consumption were compared to individuals without this variant. If the protective effect of alcohol consumption on CVD mortality were real, one would expect the group carrying the genetic variant to have a higher risk of cardiovascular mortality because of their lower alcohol consumption. However, this study of more than 260 000 individuals showed that individuals with a genetic predisposition to consume less alcohol had lower, not higher, mortality rates from coronary heart disease. This effect was observed among those within low and moderate drinking categories. Furthermore, the fact that the genetic variant was not significantly associated with CVD mortality among non-drinkers is evidence that this genetic variant does not affect CVD except through its effects on alcohol consumption. In addition, there was no association of the gene with type 2 diabetes or coagulation markers.

SECTION 4. SUMMARY, IMPLICATIONS FOR PUBLIC HEALTH

This report is primarily a summary and critique of the methodology and evidence in scientific research on the effects of low-dose alcohol consumption. To date, all studies of the relationship between alcohol consumption and morbidity and mortality outcomes have been observational by nature (i.e. mainly non-randomised cohort and case-control studies). While there are many epidemiological studies of this nature, they all suffer from the same fundamental weaknesses that are inherent in observational research illustrating that a substantial body of observational studies can be consistently wrong.

In general, the chief threats to validity for observational studies are confounding and selection bias, and recent evidence demonstrates that observational studies about effects of low-dose alcohol from developed countries are plagued by both. These methodological problems are clearly apparent in a number of studies where protective effects from low-dose alcohol are found despite the absence of any biologically plausible mechanism, including deafness, hip fracture, the common cold, and alcoholic liver cirrhosis.

Fundamentally, observed associations can be caused by a variety of other lifestyle factors as well as socio-economic and psychosocial factors which may be independently associated with alcohol consumption in the population being studied. These background factors can to some extent be controlled through statistical methods, but the extent to which this has been done varies considerably. Furthermore, there are many factors that are only partially understood, and, most likely, many factors that are unknown.

The reported beneficial effects of alcohol are largely the result of comparisons with abstainers. It has become increasingly clear however that many abstainers are at higher risk for ill health in ways unrelated to their non-consumption of alcohol.

Another major source of methodological uncertainty in observational research is the misclassification of research subjects. Numerous problems can arise in these studies in relation to how accurately different studies classify who is an abstainer and who is a moderate drinker. Failure to make this classification accurately can lead to biased comparisons between these two groups. Mostly, such biases cause the people identified as moderate drinkers to appear healthy in comparison with those classified as abstainers. The best-known example of this is sometimes referred to as the "sick quitter effect" whereby former drinkers are mixed in with lifetime abstainers. Because people who give up alcohol tend to have significantly worse health profiles, this procedure contaminates the abstainer reference group and makes the moderate drinkers "look good" by comparison. Furthermore, it is insufficient to separate out former drinkers into their own group and report mortality outcomes for them separately from people who continue to drink. Over the course of long-term observational studies, this arguably results in weeding out sick people with poor outcomes from different groups of drinkers including those classified as "moderate". To avoid spurious conclusions, reallocation of former drinkers may be necessary and is akin to the problem of dealing with missing cases in randomised clinical trials.

Correlations between alcohol and health outcomes found in observational studies also require plausible biological mechanisms to be considered causal. Such mechanisms have been identified and include favourable effects of moderate alcohol consumption on some blood biomarkers such as high density lipoprotein (HDL) cholesterol. A number of RCTs, summarized in a review and meta-analysis have supported this effect. However, the cardioprotective effect of HDL has recently come into question so that the alcohol-induced increase of HDL should also be questioned. Recent research on atherosclerosis demonstrates that alcohol consumption is only positively associated with coronary calcification and increased carotid artery thickness. The lowest risk of coronary calcification was found among lifetime abstainers.

In the medical literature, many observed associations are confirmed in subsequent randomised controlled trials. However, this is not always the case, and some notable exceptions have been recorded in which multiple observational studies were subsequently refuted by RCTs. Notable examples described in this report include beta carotene intake for the reductions in CVD and cancer, and hormone replacement therapy for the reduction of CVD, among other examples. However, both beta-carotene and hormone replacement therapy, were found to be ineffective when subjected to

performing randomised controlled studies, where both known and unknown background variables can be controlled through randomisation, but so far no RCTs have been performed in this area. This scientific standard is warranted particularly for an agent that is a leading cause of death, disability and social problems.

The protective effect of moderate drinking is not commonly found in different ethnic groups, eg black American men, Chinese and Indian populations. This adds to doubts about the cardioprotective role of alcohol, as opposed to other lifestyle factors and cultural differences.

The initiation of alcohol consumption should not be recommended for reasons of health.

Aggregate level research from many countries involving whole populations finds that changes in per capita alcohol consumption are significantly and positively correlated with corresponding changes in rates of alcohol-related diseases. However, no relationships have been found between per capita alcohol consumption and rates of cardiovascular mortality. This is yet another indication that the cardioprotective effect may be confounded in individual-level studies.

In the absence of randomised controlled trials on alcohol and mortality, Mendelian randomisation studies which utilize genetic variants that affect alcohol consumption in individuals have emerged as a good alternative to randomised clinical trials. A recent meta-analysis found that those with a genetic predisposition to consume less alcohol had lower, not higher, odds of dying from coronary heart disease, including among those with moderate alcohol consumption. This is a powerful piece of evidence suggesting that concerns with observational epidemiologic studies of the health effects of low-dose alcohol on cardiovascular health are justified.

4.1 Implications of weak evidence for health benefits: Why it matters

Our conclusion is that the evidence of protective effects for low-dose alcohol consumption is surprisingly weak, and does not warrant the far reaching conclusions that have been drawn from it. This report should be considered in the context of the fact that alcohol consumption in general is a major negative health determinant in terms of mortality, morbidity and social problems. Furthermore, alcohol results in far more adverse health impacts than it prevents, even assuming some cardiovascular benefit for low-dose consumption. With new research being reported over the last decades, estimates of the total disease burden caused by alcohol has increased considerably. New disease categories have been added, where the role of alcohol had not been recognized earlier. This particularly applies to cancer, where alcohol now is recognized as a major carcinogenic agent.

For clinicians: drinking guidelines, whether drinking should be recommended

While it is possible that low-dose alcohol consumption may be beneficial for some health outcomes including cardiovascular disease, the current evidence in support of this is weak. However, the appeal of purported health benefits, and of alcohol in particular, clearly has resulted in a lower than usual scientific standard when evaluating evidence for clinical intervention. No randomised controlled studies have been undertaken in contrast to what is required for most medical procedures or pharmaceutical products. Furthermore, the many side effects of alcohol, if viewed as a pharmaceutical, would prohibit its use, even in very moderate doses.

Strong alcohol control policies targeting price and availability should not be undermined by claims of beneficial effects of low-dose alcohol consumption.

Given the lack of evidence from randomised trials and considering the many negative consequences of alcohol consumption, public health recommendations should remain focused on: 1) reducing excessive drinking among those who already drink, and 2) discouraging initiation of alcohol consumption or more frequent drinking on the basis of health and safety considerations.

Implications for policy

The purported beneficial effects of low-dose alcohol consumption have been used as an argument against the implementation of effective population-level policies. Given the strong possibility that there are actually no cardio-protective effects and given the negative effects of alcohol, there are no reasons to oppose effective policies to reduce alcohol-related harm, e.g. raising alcohol prices and restricting the physical availability of alcohol.

However, even if there were cardio-protective effects, there are still compelling reason to adopt public popu-

lation-based polices to reduce alcohol consumption. First, alcohol consumption results in far more adverse health impacts than it is thought to prevent. Second, the evidence of negative effects of excessive alcohol consumption is more robust than for effects of low-dose consumption for the following reasons: 1) alcohol is a predominant risk factor or has 100% attribution in many conditions; 2) These associations have large effect sizes; and 3) many conditions have short latency periods between the exposure to alcohol and the adverse outcomes. Third, current meta-analyses of all-cause mortality suggest that the lowest risk for death is associated with very low levels of consumption (approximately half a drink a day for women and less than one per day for men). Therefore, population-wide reductions in consumption through the implementing effective alcohol policies would not only reduce the death and disability from excessive drinking, but could increase the number of persons to whom any benefits of moderate consumption might accrue.

The disease burden of alcohol is enormous. Over the centuries nations have struggled with the challenge to control and reduce the costs from alcohol to individuals and society. There are clearly opposing forces in this struggle. On the one hand are commercial forces that gain profit from increased consumption of alcohol and on the other hand are health and safety interests that seek to reduce the harm from alcohol through reduced drinking.

A message that moderate drinking is good for health has been used to undermine efforts to achieve effective alcohol policies on the national level. This message also sometimes confuses medical practitioners as to appropriate advice regarding alcohol consumption. The grounds for challenging the protective effects of moderate drinking have increased. This report attempts to summarise the scientific evidence concerning this issue. It concludes that the evidence for the beneficial health effects of moderate drinking in many respects is quite weak and should not compromise society's response to the problems caused by alcohol.

REFERENCES

1 Ronksley PE, Brien SE, Turner BJ, Mukamal KJ, Ghali WA.(2011). Association of alcohol consumption with selected cardiovascular disease outcomes: a systematic review and meta-analysis. BMJ 2011;342:d671.

2 Baliunas DO, Taylor BJ, Irving H, Roerecke M, Patra J, Mohapatra S, Rehm J. (2009). Alcohol as a risk factor for type 2 diabetes: A systematic review and meta-analysis. Diabetes Care. 2009 Nov;32(11):2123-32.

3 Babor T, Caetano R, Casswell S, Edwards G, Giesbrecht N, Graham K. et al. (2010) Alcohol: No Ordinary Commodity—Research and Public Policy. Oxford, UK: Oxford University Press; 2010.

4 Rehm J, Baliunas D, Borges GL, Graham K, Irving H, Kehoe T, Parry CD, Patra J, Popova S, Poznyak V, Roerecke M, Room R, Samokhvalov AV, Taylor B. (2010). The relation between different dimensions of alcohol consumption and burden of disease: an overview. Addiction. 2010 May;105(5):817-43.

5 Alkohol, droger och trafik (Alcohol, drugs and traffic), Trafikverket (Swedish Transport Administration) 2012

6 Global status report on alcohol and health – 2014, WHO: Geneva

7 Lim SS et al. (2012). A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010. Lancet. 2012 Dec 15;380(9859):2224-60.

8 Ibid.

9 Global status report on alcohol and health – 2014, WHO: Geneva

10 Rehm J, Mathers C, Popova S, Thavorncharoensap M, Teerawattananon Y, Patra J. (2009). Global burden of disease and injury and economic cost attributable to alcohol use and alcohol-use disorders. Lancet. 2009 Jun 27;373(9682):2223-33.

11 Global status report on alcohol and health – 2014 ed. (2014) WHO: Geneva

12 Rehm J, Baliunas D, Borges GL, Graham K, Irving H, Kehoe T, Parry CD, Patra J, Popova S, Poznyak V, Roerecke M, Room R, Samokhvalov AV, Taylor B. (2010). The relation between different dimensions of alcohol consumption and burden of disease: an overview. Addiction. 2010 May;105(5):817-43.

13 IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Alcohol drinking. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, volume 44. 1988: Lyon, France

14 Robert Baan, Kurt Straif, Yann Grosse, Béatrice Secretan, Fatiha El Ghissassi, Véronique Bouvard, Andrea Altieri, Vincent Cogliano; WHO International Agency for Research on Cancer Monograph Working Group. (2007). Carcinogenicity of alcoholic beverages. Lancet Oncol. 2007 Apr;8(4):292-3.

15 World Cancer Research Fund / American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective. Washington DC: AICR, 2007

16 Ibid.

17 P. Boyle et.al. (2003) European Code Against Cancer and scientific justification: third version (2003). Annals of Oncology 14: 973–1005, 2003

18 Secretan B, Straif K, Baan R, Grosse Y, El Ghissassi F, Bouvard V, Benbrahim-Tallaa L, Guha N, Freeman C, Galichet L, Cogliano V; WHO International Agency for Research on Cancer Monograph Working Group. (2009). A review of human carcinogens--Part E: tobacco, areca nut, alcohol, coal smoke, and salted fish. Lancet Oncol. 2009 Nov;10(11):1033–4.

19 Rehm J, Baliunas D, Borges GL, Graham K, Irving H, Kehoe T, Parry CD, Patra J, Popova S, Poznyak V, Roerecke M, Room R, Samokhvalov AV, Taylor B.. (2010). The relation between different dimensions of alcohol consumption and burden of disease – an overview. Addiction, 105:817–843.

20 Corrao G, Bagnardi V, Zambon A, La Vecchia C. (2004). A meta-analysis of alcohol consumption and the risk of 15 diseases. Prev Med. 2004 May;38(5):613–9.

21 Scheel J, Schelke K, Lautenbacher S, Aust S, Kremer S, Wolstein J. (2013). Low-Dose Alcohol Effects on Attention in Adolescents. Zeitschrift fur Neuropsychologie. 24 (2), 103–111.

22 Alkohol, droger och trafik (Alcohol, drugs and traffic), Trafikverket (Swedish Transport Administration) 2012

23 Hans Laurell. (1977). Effects of small doses of alcohol on driver performance in emergency traffic situations Accident Analysis & Prevention, Volume 9, Issue 3, September 1977, Pages 191–201

24 Ogden EJD, Moskowitz H. (2004). Effects of Alcohol and Other Drugs on Driver Performance. Traffic Injury Prevention, Volume 5, Issue 3, 2004, pages 185–198

25 Kuendig H, Hasselberg M, Laflamme L, Daeppen JB, Gmel G. (2008). Acute alcohol consumption and injury: risk associations and attributable fractions for different injury mechanisms. J Stud Alcohol Drugs. 2008 Mar;69(2):218–26.

26 Rossow I, Bogstrand ST, Ekeberg Ø, Normann PT. (2013). Associations between heavy episodic drinking and alcohol related injuries: a case control study. BMC Public Health. 2013 Nov 14;13:1076.

27 Duke, A, Giacola, P. Morris, D., Holt, J.; and Gunn, R. (2011). Alcohol Dose and Aggression: Another Reason by Drinking More is a Bad Idea. Journal of Studies of Alcohol and Drugs. 72(1), 34-43.

28 Hartz A, He T, Wallace R, Powers J. (2013). Comparing hormone therapy effects in two RCTs and two large observational studies that used similar methods for comprehensive data collection and outcome assessment. BMJ Open. 2013 Jul 15;3(7).

29 Anglemyer A, Horvath HT, Bero L. (2014). Healthcare outcomes assessed with observational study designs compared with those assessed in randomized trials. Cochrane Database Syst Rev. 2014 Apr 29;4:MR000034.

30 Greenberg ER. (1996). Antioxidant vitamins, cancer, and cardiovascular disease. N Eng J Med. 1996;334:1189–1190.

31 Blacker D. (2005). Mild cognitive impairment -- no benefit from vitamin E, little from donepezil. N Eng J Med. 2005;352:2439–2441.

32 Women's Health Initiative Investigators. (2002). Risks and benefits of estrogen plus progestin in healthy postmenopausal women – principal results from the Women's Health Initiative randomized controlled trial. JAMA. 2002;288:321–333.

33 Yaffe K. (2003). Hormone therapy and the brain --- deja vu all over again? JAMA. 2003;289:2717–2719.

34 Anderson JL. (2005). Infection, antibiotics, and atherothrombosis -- end of the road or new beginnings? N Eng J Med. 2005;352:1706–1709.

35 Drazen JM, Gelijns AC. (2014). Statin strikeout. N Engl J Med. 2014 Jun 5;370(23):2240–1.

36 Hue TF, Cummings SR, Cauley JA, Bauer DC, Ensrud KE, Barrett-Connor E, Black DM. (2014). Effect of bisphosphonate use on risk of postmenopausal breast cancer: results from the randomized clinical trials of alendronate and zoledronic Acid. JAMA Intern Med. 2014 Oct 1;174(10):1550–7.

37 Naimi TS, Brown DW, Brewer RD, et al. (2005). Cardiovascular risk factors and confounders among nondrinking and moderate-drinking U.S. adults. Am J Prev Med. 2005;28:369–73.

38 Wannamethee G, Shaper AG. (1988). Men who do not drink: a report from the British Regional Heart Study. Intl J Epidemiol. 1988;17:307–316.

39 Ng Fat L, Shelton N. (2012). Associations between self-reported illness and non-drinking in young adults. Addiction 2012, 107(9):1612–1620.

40 Camacho TC, Kaplan GA, Cohen RD. (1987) Alcohol consumption and mortality in Alameda County. J Chronic Dis. 1987;40(3):229–36.

41 Naimi TS, Xuan Z, Brown DW, Saitz R. (2013). Confounding and studies of 'moderate' alcohol consumption: the case of drinking frequency and implications for low-risk drinking guidelines. Addiction. 2013 Sep;108(9):1534–43.

42 Hansel B, Thomas F, Pannier B, Bean K, Kontush A, Chapman MJ, Guize L, Bruckert E. (2010). Relationship between alcohol intake, health and social status and cardiovascular risk factors in the urban Paris-Ile-De-France Cohort: is the cardioprotective action of alcohol a myth? Eur J Clin Nutr. 2010 Jun;64(6):561–8

43 Stockwell T, Greer A, Fillmore K, Chikritzhs T, Zeisser C. (2012). How good is the science. BMJ 2012, 344:e2276.

44 Naimi TS, Brown DW, Brewer RD, et al. (2005). Cardiovascular risk factors and confounders among nondrinking and moderate-drinking U.S. adults. Am J Prev Med. 2005;28:369–73.

45 Hansel B, Thomas F, Pannier B, et al. (2010). Relationship between alcohol intake, health and social status and cardiovascular risk factors in the urban Paris-Ile-De-France Cohort: is the cardioprotective action of alcohol a myth? European J Clin Nutr. 2010;64:561–8.

46 Marmot M. (2005). Social determinants of health inequalities. Lancet. 2005;365:1005–6.

47 Rundberg J, Lidfeldt J, Nerbrand C, Samsioe G, Romelsjö A, Ojehagen A. (2008). Abstinence, occasional drinking and binge drinking in middle-aged women. The Women's Health in Lund Area (WHILA) Study. Nord J Psychiatry. 2008;62(3):186–91.

48 Rundberg J, Nilsson PM, Samsioe G, Ojehagen A. (2014). Alcohol use and early mortality in Swedish middle-aged women: Nine-year follow-up of the Women's Health in Lund Area study. Scand J Public Health. June 2014 42: 344–348

49 Kühlhorn E, Björ J. (1991). De nyktra och alkoholkonsumenterna (The abstainers and the alcohol consumers), in Gustavsson A (ed): Alkoholister och nykterister, vol 10. Uppsala, Etnolore 1991, pp 271–293

50 Leifman H, Kühlhorn E, Allebeck P, Andréasson S, Romelsjö A. (1995). Abstinence in late adolescence-Antecedents and covariates to a sober lifestyle and its consequences. Soc Sci Med 41:113–121, 1995

51 Rogers RG, Krueger PM, Miech R, Lawrence EM, Kemp R. (2013) Nondrinker Mortality Risk in the United States. Population Research and Policy Review June 2013, Volume 32, Issue 3, pp 325–352

52 Rogers RG, Krueger PM, Miech R, Lawrence EM. (2013) Lifetime abstainers and mortality risk in the United States. Working paper, Institute of Behavioral Science, University of Colorado Boulder, http://www.colorado.edu/ibs/pubs/pop/pop2012–0006.pdf

53 Fekjaer HO. (2013) Alcohol-a universal preventive agent? A critical analysis. Addiction. 2013 Dec;108(12):2051–7.

54 Rehm J, Taylor B, Mohapatra S, Irving H, Baliunas D, Patra J, Roerecke M. (2010) Alcohol as a risk factor for liver cirrhosis: a systematic review and meta-analysis. Drug Alcohol Review, 29:437–45.

55 Jin M, Cai S, Guo J, Zhu Y, Li M, Yu Y, Zhang S, Chen K. (2013). Alcohol drinking and all cancer mortality: a meta-analysis. Annals of Oncology 2013; 24:807–816.]

56 Liang W, Chikritzhs T. (2013). Alcohol consumption and health status of family members: health impacts without ingestion. Intern Med J. 2013 Sep;43(9):1012–6.

57 Robinson M, Oddy W, McLean N, Jacoby P, Pennell C, de Klerk N, Zubrick S., Stanley F, Newnham J. (2010) Low-moderate prenatal alcohol exposure and risk to child behavioural development: a prospective cohort study. BJOG 2010 Aug;117(9):1139–50

58 Estruch R, Ros E, Salas-Salvadó J, Covas MI, Corella D, Arós F, Gómez-Gracia E, Ruiz-Gutiérrez V, Fiol M, Lapetra J, Lamuela-Raventos RM, Serra-Majem L, Pintó X, Basora J, Muñoz MA, Sorlí JV, Martínez JA, Martínez-González MA; PREDIMED Study Investigators. (2013). Primary prevention of cardiovascular disease with a Mediterranean diet. N Engl J Med. 2013 Apr 4;368(14):1279–90

59 Kerr WC, Greenfield TK, Ye Y, Bond J, Rehm J. (2013). On French and American paradoxes. Addiction. 2013 Nov;108(11):2029–30.

60 Fillmore, KM, Kerr WC, Stockwell T, Chikritzhs T, Bostrom A. (2006). Moderate alcohol use and reduced mortality risk: systematic error in prospective studies. Addiction Research and Theory, 14, 101–132

61 Stockwell T, Greer A, Fillmore K, Chikritzhs T, Zeisser C. (2012). How good is the science. BMJ 2012, 344:e2276.

62 Ronksley PE, Brien SE, Turner BJ, Mukamal KJ, Ghali WA.(2011). Association of alcohol consumption with selected cardiovascular disease outcomes: a systematic review and meta-analysis. BMJ 2011;342:d671.

63 Liang W, Chikritzhs T. (2013). The association between alcohol exposure and self-reported health status: the effect of separating former and current drinkers. PLoS One. 2013;8(2):e55881.

64 Ibid.

65 Doll R, Peto R, Boreham J, Sutherland I. (2004) Mortality in relation to smoking: 50 years' observations on male British doctors, BMJ, 328, 1519.

66 Dawson D. (2000). Alcohol consumption, alcohol dependence and all-cause mortality. Alcohol Clin Exp Res 2000;24:72–81.

67 Caldwell T, Rodgers B, Power C, et al. (2006). Drinking histories of self-identified lifetime abstainers and occasional drinkers: findings from the 1958 British Birth Cohort study. Alcohol Alcohol 2006;41:650–4.

68 Corrao G, Rubbiati L, Bagnardi V, Zambon A, Poikolainen K. (2000). Alcohol and coronary heart disease: a meta-analysis. Addiction. 2000 Oct;95(10):1505–23.

69 Norström T, Skog OJ. (2001) Alcohol and mortality: methodological and analytical issues in aggregate analyses. Addiction. 2001 Feb;96 Suppl 1:S5–17.

70 Ramstedt M. (2001). Per capita alcohol consumption and liver cirrhosis mortality in 14 European countries. Addiction. 2001 Feb;96 Suppl 1:S19–33.

71 Skog OJ. (2001). Alcohol consumption and mortality rates from traffic accidents, accidental falls, and other accidents in 14 European countries. Addiction. 2001 Feb;96 Suppl 1:S49–58.

72 Skog OJ. (2001). Alcohol consumption and overall accident mortality in 14 European countries. Addiction. 2001 Feb;96 Suppl 1:S35–47.

73 Ramstedt M. (2002). Alcohol-Related Mortality in 15 European Countries in the Postwar Period. European Journal of Population; 18:307–323, 2002.

74 Hemström O. (2001). Per capita alcohol consumption and ischaemic heart disease mortality. Addiction. 2001 Feb;96 Suppl 1:S93–112.

75 Ramstedt M. (2006). Is alcohol good or bad for Canadian hearts? A time-series analysis of the link between alcohol consumption and IHD mortality. Drug Alcohol Rev. 2006 Jul;25(4):315–20.

76 Kerr WC, Karriker-Jaffe K, Subbaraman M, Ye Y. (2011). Per capita alcohol consumption and ischemic heart disease mortality in a panel of US states from 1950 to 2002. Addiction. 2011 Feb;106(2):313–22.

77 Skog OJ. (1983). Methodological problems in the analysis of temporal covariation between alcohol consumption and ischemic heart disease. Br J Addict. 1983 Jun;78(2):157–72.

78 Pun VC, Lin H, Kim JH, Yip BH, Chung VC, Wong MC, Yu IT, Griffiths SM, Tian L. (2013). Impacts of alcohol duty reductions on cardiovascular mortality among elderly Chinese: a 10-year time series analysis. J Epidemiol Community Health. 2013 Jun;67(6):514–8.

79 Her M, Rehm J. (1998). Alcohol and all-cause mortality in Europe 1982–1990: a pooled cross-section time-series analysis. Addiction. 1998 Sep;93(9):1335–40.

80 Norström T. (2001). Per capita alcohol consumption and all-cause mortality in 14 European countries. Addiction (2001) 96(Supplement 1), S113–S128

81 Norström T. Per capita alcohol consumption and all-cause mortality in Canada, 1950–98. Addiction. 2004 Oct;99(10):1274–8.

82 Hvidtfeldt UA, Tolstrup JS, Jakobsen MU, Heitmann BL, Grønbaek M, O'Reilly E, Bälter K, Goldbourt U, Hallmans G, Knekt P, Liu S, Pereira M, Pietinen P, Spiegelman D, Stevens J, Virtamo J, Willett WC, Rimm EB, Ascherio A. (2010). Alcohol intake and risk of coronary heart disease in younger, middle-aged, and older adults. Circulation. 2010 Apr 13;121(14):1589–97.

83 Ronksley PE, Brien SE, Turner BJ, Mukamal KJ, Ghali WA.(2011). Association of alcohol consumption with selected cardiovascular disease outcomes: a systematic review and meta-analysis. BMJ 2011;342:d671.

84 Fuchs FD, Chambless LE, Folsom AR, Eigenbrodt ML, Duncan BB, Gilbert A, Szklo M. (2004) Association between alcoholic beverage consumption and incidence of coronary heart disease in whites and blacks: the Atherosclerosis Risk in Communities Study. Am J Epidemiol. 2004; 160: 466–74.

85 Kerr WC, Greenfield TK, Bond J, Ye Y, Rehm J. (2011). Racial and Ethnic Differences in All-Cause Mortality Risk According to Alcohol Consumption Patterns in the National Alcohol Surveys. Am J Epidemiol. 2011 Oct 1;174(7):769–78.

86 Zhou X, Li C, Xu W, Hong X, Chen J. (2010). Relation of alcohol consumption to angiographically proved coronary artery disease in Chinese men. Am J Cardiol. 2010 Oct 15;106(8):1101–3.

87 Schooling CM, Sun W, Ho SY, Chan WM, Tham MK, Ho KS, Leung GM, Lam TH. (2008). Moderate alcohol use and mortality from ischaemic heart disease: a prospective study in older Chinese people. PLoS One. 2008 Jun 4;3(6):e2370.

88 Roy A, Prabhakaran D, Jeemon P, et al. (2010) Impact of Alcohol on Coronary Heart Disease in Indian Men. Atherosclerosis 2010;210:531–5

89 Rizos EC, Ntzani EE, Bika E, Kostapanos MS, Elisaf MS. (2012). Association between omega-3 fatty acid supplementation and risk of major cardiovascular disease events: A systematic review and meta-analysis. JAMA, 2012. 308(10): p. 1024–1033.

90 Bergmann M, Rehm J, Klipstein-Grobusch K et al. (2013) The association of pattern of lifetime alcohol-use and cause of death in the European Prospective Investigation into Cancer and Nutrition (EPIC) study. Int J Epidemiol 2013; doi:10.1093/ije/dyt154.

91 Kerr WC, Ye Y. (2010). Relationship of life-course drinking patterns to diabetes, heart problems, and hypertension among those 40 and older in the 2005 U.S. National Alcohol Survey. J Stud Alcohol Drugs. 2010 Jul;71(4):515–25.

92 Liang, W. & Chikritzhs T. (2014) Alcohol consumption during adolescence and risk of diabetes in young adulthood. BioMed International, vol 2014 Article ID 795741, 6 pages. Doi: 10.1155/2014/795741.

93 Seike N, Noda M, Kadowaki T. (2008).Alcohol consumption and risk of type 2 diabetes mellitus in Japanese: a systematic review. Asia Pac J Clin Nutr. 2008;17(4):545–51.

94 Brien SE, Ronksley PE, Turner BJ, Mukamal KJ, Ghali WA. (2011). Effect of alcohol consumption on biological markers associated with risk of coronary heart disease: systematic review and meta-analysis of interventional studies. BMJ 2011;342:d636

95 Briel M, Ferreira-Gonzalez I, You JJ, Karanicolas PJ, Akl EA, Wu P, Blechacz B, Bassler D, Wei X, Sharman A, Whitt I, Alves da Silva S, Khalid Z, Nordmann AJ, Zhou Q, Walter SD, Vale N, Bhatnagar N, O'Regan C, Mills EJ, Bucher HC, Montori VM, Guyatt GH. (2009). Association between change in high density lipoprotein cholesterol and cardiovascular disease morbidity and mortality: systematic review and meta-regression analysis. BMJ. 2009 Feb 16;338:b92.

96 Schwartz GG, Olsson AG, Abt M, Ballantyne CM, Barter PJ, Brumm J, Chaitman BR, Holme IM, Kallend D, Leiter LA, Leitersdorf E, McMurray JJ, Mundl H, Nicholls SJ, Shah PK, Tardif JC, Wright RS; dal-OUTCOMES Investigators. (2012). Effects of dalcetrapib in patients with a recent acute coronary syndrome. N Engl J Med. 2012 Nov 29;367(22):2089–99.

97 Voight BF, Peloso GM, Orho-Melander M, Frikke-Schmidt R, Barbalic M, et.al. (2012). Plasma HDL cholesterol and risk of myocardial infarction: a Mendelian randomisation study. Lancet. 2012 Aug 11;380(9841):572–80.

98 Pletcher MJ, Varosy P, Kiefe CI, Lewis CE, Sidney S, Hulley SB. (2004). Alcohol Consumption, Binge Drinking, and Early Coronary Calcification: Findings from the Coronary Artery Risk Development in Young Adults (CARDIA) Study. American Journal of Epidemiology, Vol. 161, No. 5

99 Juonala M, Viikari JS, Kähönen M, Laitinen T, Taittonen L, Loo BM, Jula A, Marniemi J, Räsänen L, Rönnemaa T, Raitakari OT. (2009). Alcohol consumption is directly associated with carotid intima–media thickness in Finnish young adults. Atherosclerosis. 2009 Jun;204(2):e93–8.

100 Ding J, Eigenbrodt ML, Mosley TH Jr, Hutchinson RG, Folsom AR, Harris TB, Nieto FJ.(2004). Alcohol intake and cerebral abnormalities on magnetic resonance imaging in a community-based population of middle-aged adults: the Atherosclerosis Risk in Communities (ARIC) study. Stroke. 2004 Jan;35(1):16–21.

101 Kumari M, Holmes MV, Dale CE, Hubacek JA, Palmer TM, Pikhart H, Peasey A, Britton A, Horvat P, Kubinova R, Malyutina S, Pajak A, Tamosiunas A, Shankar A, Singh-Manoux A, Voevoda M, Kivimaki M, Hingorani AD, Marmot MG, Casas JP, Bobak M. (2014). Alcohol consumption and cognitive performance: a Mendelian randomization study. Addiction. 2014 Sep;109(9):1462–71.

102 Au Yeung SL, Jiang CQ, Cheng KK, Liu B, Zhang WS, Lam TH, Leung GM, Schooling CM. (2012). Evaluation of moderate alcohol use and cognitive function among men using a Mendelian randomization design in the Guangzhou biobank cohort study. Am J Epidemiol. 2012 May 15;175(10):1021–8.

103 Corrao G, Bagnardi V, Zambon A, La Vecchia C. (2004). A meta-analysis of alcohol consumption and the risk of 15 diseases. Prev Med. 2004 May;38(5):613–9.

104 Patel R, Lawlor DA, Whincup P, Montaner D, Papacosta O, et al. (2006) The detection, treatment and control of high blood pressure in older British adults: cross-sectional findings from the British Women's Heart and Health Study and the British Regional Heart Study. J Hum Hypertens 20: 733–741.

105 Fuchs FD, Chambless LE, Whelton PK, Nieto FJ, Heiss G (2001) Alcohol consumption and the incidence of hypertension : The Atherosclerosis Risk in Communities Study. Hypertension 37: 1242–1250.

106 Moore RD, Levine DM, Southard J, Entwisle G, Shapiro S (1990) Alcohol consumption and blood pressure in the 1982 Maryland Hypertension Survey. Am J Hypertens 3: 1–7.

107 Chen L, Davey Smith G, Harbord RM, Lewis SJ (2008) Alcohol Intake and Blood Pressure: A Systematic Review Implementing a Mendelian Randomization Approach. PLoS Med 5(3): e52. doi: 10.1371/journal.pmed.0050052

108 Holmes MV, Dale CE, Zuccolo L, Silverwood RJ, Guo Y, et.al.; InterAct Consortium. (2014). Association between alcohol and cardiovascular disease: Mendelian randomisation analysis based on individual participant data. BMJ. 2014 Jul 10;349:g4164.

BEER IS GOOD FOR THE HEART! WINE CAN CAUSE CANCER! – MEDIA ATTITUDES TOWARDS ALCOHOL RESEARCH

By Sara Nilsson

"Alcohol protects your heart."
"Alcohol linked to increased cancer risk."
"A little wine boosts your memory."

As readers, we encounter a veritable flood of contradictory news on the ways in which alcohol affects our health.

In the following article, news chiefs and reporters from Sweden's biggest newspapers discuss the way they regard the latest research on alcohol and their responsibility for the risk/benefit pictures painted by their news.



Readers encounter articles based on the latest research findings about the effects of alcohol on health in both the morning broadsheets and evening tabloids. A small dose is good for your sense of smell, drinking in the sun increases the risk of skin cancer, and alcohol is an effective means of preventing aneurysms, are just a few examples of the kind of articles readers see.

Sweden's biggest news website, aftonbladet.se, is no exception, and the latest findings on alcohol have a natural place here amongst the celebrity gossip, foreign news, and sports news.

"Alcohol plays a major part in our society and is consumed by so many people that writing about it is often of considerable interest to us", says Martin Schori, Assistant News Director at aftonbladet.se. According to Schori, the world of journalism and the tabloid evening papers has a history of glorifying alcohol consumption, but he believes that attitudes have changed at Aftonbladet.

"There's an increased awareness, nowadays, of the fact that we shouldn't romanticise alcohol when we write. But yes, things do slip through the net from time to time", says Schori.

Many people believe that the tabloid press will write anything at all in their hunt for clicks or newsstand sales. But Martin Schori points out that traditional newsworthiness standards still apply – what they write must be true, relevant and interesting – and he believes that they do pay attention to the picture they are painting of the benefits or risks of alcohol consumption.

"There's this prejudice that we never think twice before we publish, but that's just not the case. We're no longer a complementary medium that people read for entertainment purposes: we're the only news source for a lot of people. Which is why we've had to position ourselves and to rethink – including when it comes to the way in which we write about alcohol", says Schori.

According to Martin Schori, an expert is often asked to comment on the study, in order to assess its veracity. Alternatively, they use sources that are deemed to be credible, such as certain foreign newspapers, universities or publications.



Anna Bäsén, medical reporter for the Expressen newspaper, agrees that new research into alcohol is of interest to her newspaper because alcohol affects so many people. She also believes that that the newspaper has a responsibility for the picture they paint of the benefits or risks of alcohol consumption.

"Obviously, we have to try and provide balanced reporting. And there are undoubtedly times when we succeed very well in this respect, and times when we don't."

"I try to communicate the risks when I write. When the piece is about cardiovascular health and alcohol, for example, I try to make it clear that the group of people who might benefit is a small one – namely the elderly – and that for a lot of people, they really shouldn't be increasing the amount they drink. That, in fact, the opposite is true", says Bäsén.

Research-based news on the health effects of alcohol can, as noted above, be found in both the broadsheet morning press and the tabloid evening press and Anna Bäsén sees no great difference between them when it comes to the way in which the articles are put together.

"We report research news in pretty much the same way as the big morning papers. When people say there are huge differences, they're probably mainly thinking about our placards or lifestyle magazines", says Bäsén.

According to Bäsén, it is, in fact, the broadsheets who have moved closer to the tabloids, partly by picking up on the massive interest in health issues, and partly due to the fact that, nowadays, they write in a simpler and more populist style.

"They've also moved our way when it comes to making things more personal. They don't just include comments from a professor or expert in the article, they include someone directly affected. Someone who says, 'I lowered my blood pressure by drinking less alcohol'", notes Bäsén.

Anna Bäsén sees no problem with the fact that readers can one day be confronted by news saying that alcohol is good for your health, and another, by news that it is bad, and says it is a feature common to all forms of health reporting. As she sees it, there are both advantages and disadvantages to alcohol consumption, and the media must be able to present it that way.

"Researchers do, after all, disagree sometimes and we have to be able to show that. Our job is to report on the research findings and we have to be able to do that before the authorities or other organisations have reached their own conclusions on a given issue. Acting as a megaphone for Sweden's Public Health Agency or the National Board of Health and Welfare, or for anyone else, come to that, is not our job", says Bäsén.

She adds that the fact that contradictory research exists, or that a new study might show the complete opposite of what was previously believed, should also be borne in mind.

"Different studies can show different things", says Lena Karvik, Acting Head of the Newsroom at the broadsheet morning newspaper, Svenska Dagbladet. She also sees nothing wrong with the fact that the news with which readers are presented alternates between saying that alcohol is good and bad for your health.

"We're not promoting a particular scientific policy or line. We take a view on each thing we publish on a case to case basis, so that can happen. The important thing is that we demand certain standards of the research-related news we write about. We attempt, in every article we write, to give the readers everything they need to understand the news and we will always say if other studies point in the opposite direction. That's what we aim to do, anyway", says Karvik.

A fundamental requirement for publication in Svenska Dagbladet is that the studies about which they write must have been published in a scientific publication or come from an accepted source, such as a respected university or research group. The newspaper also looks at what type of study it is in order to determine how reliable it is, the method used, and the study's financing source.

Similar criteria apply at the Dagens Nyheter morning broadsheet, according to Maria Gunther, the paper's Science Editor and science reporter. The studies that the newspaper covers are generally those published in high-status scientific publications and which are adjudged to be of a high quality – the conclusions drawn must be plausible, and there should not be any other possible explanations. She admits that even the research published in scientific periodicals falls down on quality from time to time, but that the reputation of the publication and its status act as a filter for shoddy studies.

This approach applies to all research-related news in Dagens Nyheter, but studies that show positive health effects for alcohol enjoy a special position, according to Maria Gunther.

"Alcohol is an enormous public health problem, so we always try to include a 'facts and figures box' in which we state precisely which group the positive effects apply to, such as older women, and who it is harmful for, as well as stating it clearly in the main text", says Gunther.

Maria Gunther emphasises the paper's endeavours to give the reader as complete a picture as possible when it comes to health effects.

"We live in a complex world, which means the overall picture is complex too: a glass of wine might be good for your cardiovascular system, but then there are also a lot of hazards associated with alcohol. We have to try and give readers the full picture, even if that can be difficult when space is limited", says Gunther.

The desire to give their readers the full picture is something that is also mentioned by Magnus Persson, News Director at the TT Nyhetsbyrån news agency, which produces news for a number of Swedish editorial departments.

“If there’s serious research pointing in different directions, it’s important that we highlight that fact. That’s something we bear in mind at all times. Not because we feel any sort of social responsibility for public health, but because we attempt, as far as possible, to show the picture in all its complexity”, says Persson.

This might also involve pointing out any weaknesses in the study in question. Or of pointing out those cases when research that has shown one particular health effect for alcohol has only looked at a minor aspect of overall health.

“But then, the way media works is that we take a particular slant on something in the news and start talking about what’s new in the findings. Only then do we get to the ‘on the other hand, there is research that points in a completely different direction’ bit, and it’s possible that not everyone takes that latter part on board”, says Magnus Persson.

TT’s quality control procedure mandates that the studies must have been published in a scientific periodical.

THE ULTIMATE CURIOSITY JOB

By Sara Nilsson

Frida Dangardt, M.D., a researcher at the University of Gothenburg, is a new member of the group of alcohol researchers who have worked on this year's theme article on low-dose drinking.

Frida Dangardt's aim, through her research into cardiovascular disease risk factors, is to push decision-makers towards generating better preconditions for health.

"There's a lot of talk about how alcohol is supposedly good for your heart and circulatory system. But we quickly forget that there is no solid proof of this and that the levels in question are so low that virtually no one drinks in such small quantities – so all that's left is all the negative health effects", says Frida Dangardt, researcher at the University of Gothenburg and a physician at Queen Sylvia Paediatric Hospital in Gothenburg.

For Frida Dangardt, this is the first time she has been involved in the Alcohol & Society report, which sees a group of leading alcohol researchers working together to summarise the research position in a specific area within the overall field. This year, that area is low-dose drinking, and Dangardt says that the decision to accept the invitation to take part was an easy one.

"I'm interested in exerting an influence at society level and I'm keen to try and help the research that does exist become better known and to be presented in a way that makes it readily accessible and comprehensible to the public and decision makers", says Dangardt.

Frida Dangardt's research examines early factors in children that may influence the development of cardiovascular disease, such as exercise, diet, stress and alcohol use. She has shown, amongst other things, that overweight children display risk factors for future cardiovascular disease at an early stage in their lives, and that they can be helped by adding Omega-3 fatty acids to their diet.

"Without changing their overall diet or exercise levels – simply by giving the children an Omega-3 supplement – we were able to improve their vascular function. It's great that there are such simple things that we can do to influence the risk factors", says Frida Dangardt.



Frida Dangardt

These results will, however, have to be confirmed by larger studies before general dietary recommendations can be issued.

And it is precisely these opportunities to influence many people's health and reduce health inequalities that are the key driving forces for Frida Dangardt in her work. She responds instantly when asked what her research goals are.

"For people to feel better! Either through research that persuades people to change their lifestyle, or which persuades decision-makers to generate better preconditions for health – not least, children's health."

There is a lot that can be done at societal level to influence the lifestyle factors that she studies, according to Frida Dangardt, who mentions physical activity in schools as an example. Children of well-educated parents often participate in organised leisure activities

and get a lot of physical exercise, while children whose parents cannot afford these organised activities, for example, get virtually no exercise at all.

"In the past, children simply played outdoors anyway, but that's not the case these days. And as a result, there's a huge social difference in health that could be equalised with more physical education in schools", says Dangardt.

Frida Dangardt qualified as a doctor in 2005 and now works as a paediatric physiologist, but it was by no means a given that she would become a doctor at all. She started studying civil engineering, but dropped out, wanting to work more with people – and preferably children – and she thought about becoming a teacher. It was not until a study counsellor suggested training as a doctor that it struck her that this was one way of combining her interest in people with her curiosity about how the body works.

Inspired by an enthusiastic – and enthusing – paediatric physiologist, she began researching in this field while she was still training to become a doctor and defended her doctoral thesis in 2008. She now divides her time between clinical work and research and tuition at the University.

"I've always been curious and research is the ultimate curiosity job. The chance to choose which subject you want to know about and then to investigate it is just so much fun!" says Dangardt.

Frida Dangardt was awarded a public health scholarship and spent the last two years in England as a visiting researcher at University College London.

"I had the chance to work with some of the world's leading researchers in my field and to study massive populations – something we don't get to do in Sweden", says Dangardt.

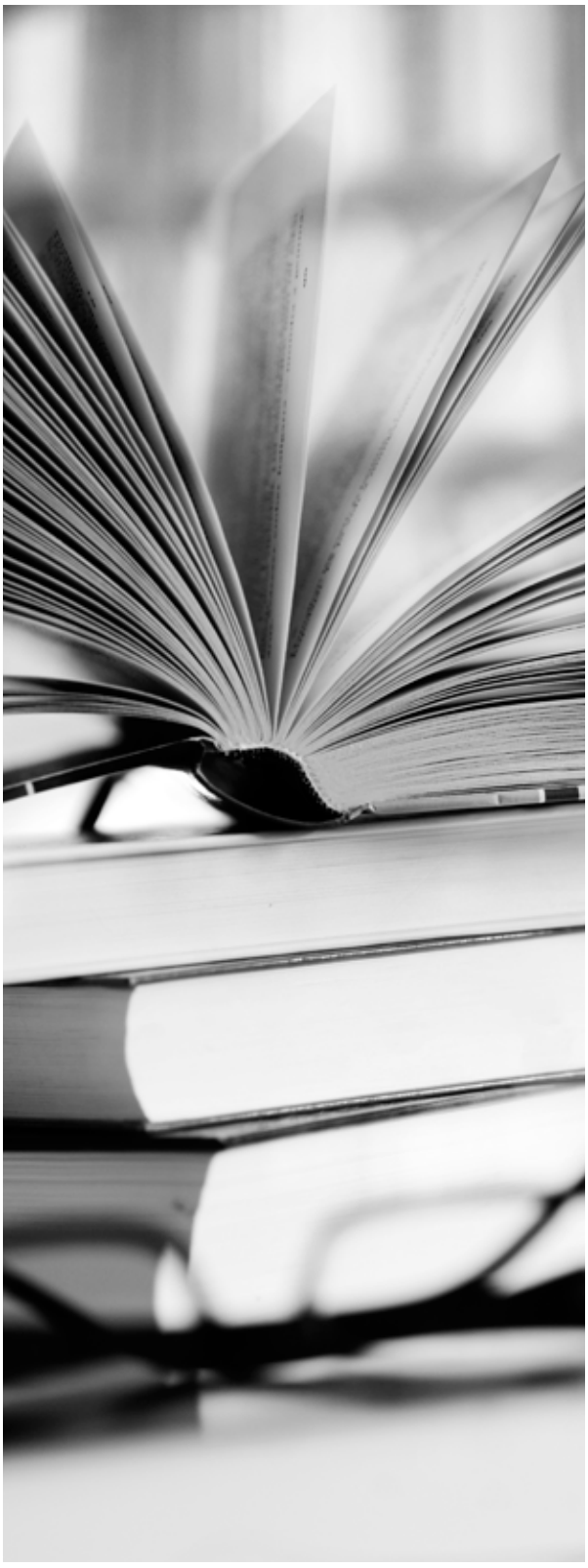
She worked, amongst other things, on a study of just over 14,000 children being monitored throughout their childhoods by drawing samples and completing extensive questionnaires about the children's dietary habits, and Dangardt was able, with the help of data from ultrasound investigations and answers to questions about alcohol habits from a group of children, to see how their circulatory systems are affected by alcohol consumption.

"Even at the age of 17, we were able to see that those who drink a lot in one go, even if they don't drink very often, had poorer circulatory function, which may predict a tendency to develop cardiovascular disease when they are older", says Dangardt.

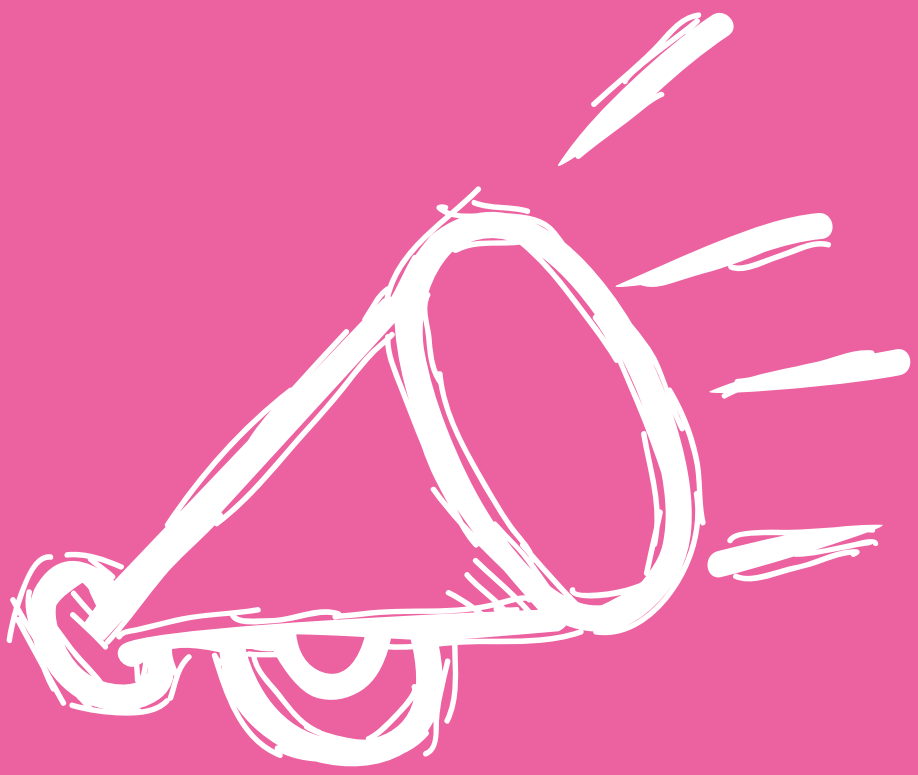
The results of the study to date have been presented at a scientific conference.

Back home in Sweden again now, Frida Dangardt has brought with her valuable, ongoing international partnerships and a track record of publication in prestigious scientific publications. She has also brought back insights into what appeals to her about the working climate, here in Sweden.

"In England, my colleagues and I just kept on working. We didn't each lunch together, we didn't take coffee breaks together – nothing. The social side of things is much nicer here in Sweden and, in my opinion, that is ultimately of benefit to the research as well", says Dangardt.



LATEST RESEARCH



SOCIOECONOMIC DIFFERENCES AND DEATHS ATTRIBUTABLE TO ALCOHOL

Low socioeconomic status, e.g. lower education, lower occupational status, lower income or unemployment, has been shown to be associated with substantial higher risk of death. The reasons for this are not clear. E.g. lifestyle as physical activity and smoking, or psychological factors like stress can explain only part of the differences.

In an effort to explain the socioeconomic differences in mortality by focussing on alcohol-related deaths, a group of researchers have summarized the results from 15 papers from European countries, together capturing about 133 million people, 3 741 334 deaths from all causes and 167 652 alcohol-attributable deaths.

The summarized result showed a 70 per cent higher risk for alcohol-attributable deaths for the group with lowest socioeconomic status compared to the highest. This increase in risk is over and above the general increased risk of death from all causes in lower socioeconomic groups.

Again, the reasons for these differences in alcohol-attributable deaths are not clear. Differences in consumption patterns have not been able to explain the difference and combination effects with smoking, e.g. mouth cancer, represent only a small part of the number of deaths. Access to primary care and dietary differences can be possible factors.

All alcohol-attributable harm is in principle avoidable. In Europe almost 14 per cent of all deaths in adult men and 8 per cent in adult women (aged 15–64 years) are estimated as attributable to alcohol. General alcohol policy measures like taxation, limitation of selling times and density of alcohol stores have been shown to be effective to reduce socioeconomic differences in alcohol-related harm. Social welfare spending has also been shown to reduce alcohol-attributable deaths, whereas rising health care spending was not. Nevertheless, screening and brief intervention in primary health care have been shown to be cost-effective measures to reduce alcohol consumption, related harm and mortality.

REFERENCE

Probst C, Roerecke M, Behrendt S, Rehm J. (2014). Socioeconomic differences in alcohol-attributable mortality compared with all-cause mortality: a systematic review and meta-analysis. Int J Epidemiol. 2014 Aug;43(4):1314-27



ALCOHOL AND ALCOHOL RELATED HARM IN CHINA

China is the world's most populous country. With its 1.35 billion inhabitants it represents almost one fifth of the world's population. The growing consumption of alcohol in China and its subsequent harm, as well as policy response, is described in an article in the Bulletin of the World Health Organisation.

Drinking alcohol has been traditional in China during major social events, such as the spring festival and wedding ceremonies. However, the rapid growth in the Chinese economy has been accompanied by noticeable changes in the drinking behaviour of the Chinese population.

Alcohol consumption in China is increasing faster than anywhere else in the world. Alcohol consumption per capita 15 years of age and older is estimated to have increased from 0.4 litres of pure alcohol in 1952 to 2.5 litres at the end of the Cultural Revolution in 1978 and to 4.9 litres in 2009. This figure does not include unregistered consumption which for the year 2000 was estimated to 1.7 litres per capita, approximately 30 per cent of total consumption.

A national survey of drinking in China for the year 2007 revealed that 55.6% of the men and 15.0% of the women were current drinkers, of which 62.7% of the men and 51.0% of the women reported excessive drinking. That would imply that e.g. more than 200 million Chinese men drink excessively.

Alcohol-related harm has not been systematically studied in China and reliable, nation-wide data are not available. According to World Health Organization estimates, rates of alcohol use disorders in China are 6.9% and 0.2% among men and women, respectively, corresponding to more than 26 million persons.

As in other countries, alcohol drinking is associated not just with health-related harm, but also with social harm, specifically traffic accidents, crime and child abuse, domestic violence and injuries of all types, including work-related injuries. The data are sparse however and most studies are based on case analysis or small clinical samples.

Alcohol policies in China are weak. For example, China has no enforceable legal drinking age and does not regulate when or where alcoholic products are sold. Weak alcohol policies lead to increased alcohol consumption and alcohol-related problems. Few things can be accomplished in China without the direct involvement and support of the central government. Unlike the use of tobacco and illicit drugs, alcohol use has received scant attention from Chinese policy-makers and public health officials. Some of them even argue that the alcohol industry is an important driver of national economic growth. Hence, China's government agencies for alcohol regulation are poorly coordinated. Several ministries share responsibility for different aspects of alcohol policy and no national supervisory or coordinating body exist.

The authors recommend that China's government officials follow WHO's Global alcohol strategy as a model for the planning and implementation of a public health approach to alcohol control. Many of the interventions in the strategy are universal measures intended to reduce the affordability by strengthening taxation, availability by licensing of sales or creating state retail monopolies for alcohol and accessibility of alcohol, e.g. by enforcing age controls and marketing restrictions.

REFERENCE

Tang YL, Xiang XJ, Wang XY, Cubells JF, Babor TF, Hao W. (2013). Alcohol and alcohol-related harm in China: policy changes needed. Bull World Health Organ 2013;91:270–276



DOES THE ALCOHOL POLICY ENVIRONMENT HAVE ANY EFFECT ON THOSE WHO DRINK EXCESSIVELY?

Most evaluation of alcohol policies look to changes in individual policies and relate those to level of consumption and harm. But the combined effect of several policies and the importance of the degree of implementation have received less attention from research. One earlier study has created an Alcohol Policy Index, taking into account both efficacy and stringency of implementation. The index was used across 30 OECD states and compared to per capita alcohol consumption as well as youth drinking.

A study published in American Journal of Preventive Medicine 2014, constructs a scale of alcohol policies across United States and correlates it to the level of excessive drinking, i.e. binge drinking or drinking above estimated levels of low-risk drinking.

Excessive drinking is a leading cause of disease, death, social problems, and economic costs in the U.S. Binge drinking alone accounts for approximately half of alcohol-attributable deaths, two thirds of years of potential life lost, and three fourths of economic costs.

For the study ten alcohol policy experts nominated policies to be included. Forty-seven policies were nominated and 29 were eventually selected for ratings of efficacy and implementation. As the aim was to study state policy environments, federal policies or those that did not vary across states or did not exist in the U.S. were excluded. Examples of excluded policies were blood alcohol concentration (BAC) 0.05 laws that do not exist in the U.S. and restrictions on mass media advertising which are not promulgated at the state level.

The Alcohol Policy Score, taking into account both efficacy and implementation of each of the included 29 policies, was constructed for each state and year. A higher score, i.e. more stringent alcohol policy environment, was associated with lower rate of excessive alcohol consumption.

A following study evaluated the effect of different categories of policies on binge drinking in the U.S. It found that policies that targeted the general population rather than the underage population, alcohol consumption rather than impaired driving, and raising the price or reducing the availability of alcohol had the strongest independent associations with reduced binge drinking. Alcohol taxes and outlet density accounted for approximately half of the magnitude observed for all policies.

REFERENCES

Naimi TS, Blanchette J, Nelson TF, Nguyen T, Oussayef N, Heeren TC, Gruenewald P, Mosher J, Xuan Z.(2014). A new scale of the U.S. alcohol policy environment and its relationship to binge drinking.. Am J Prev Med. 2014 Jan;46(1):10–6.

Xuan Z, Blanchette J, Nelson TF, Heeren T, Oussayef N, Naimi TS. The Alcohol Policy Environment and Policy Subgroups as Predictors of Binge Drinking Measures Among US Adults. American Journal of Public Health, published online ahead of print August 14, 2014



ARE ALCOHOL POLCIES EFFECTIVE IN LOW- AND MIDDLE-INCOME COUNTRIES?

Most of the research on alcohol policies has been conducted in high-income, western countries. This raises the question whether the policies like taxes, limited availability, marketing restrictions etc. are effective also in low- and middle-income countries. Lower living standards and different drinking cultures could conceivably change the effect of alcohol policies.

Data on alcohol consumption and policies from 15 low- and middle-income countries between 1998 and 2005 were compared in a study published 2014. The countries were mainly South American, African and Asian countries.

The analysis shows that policies that limit the physical availability or raise prices are effective also in these countries. For physical availability, policies that limit business hours or involve a licensing system for off-premises alcohol retail sales, as well as minimum legal drinking age, were the policies that most consistent lowered alcohol consumption. Higher alcohol price levels were also associated with lower drinking. Greater restrictions on alcohol advertising, particularly beer advertising, were also associated with lower alcohol consumption. However, policies that set legal blood alcohol concentration limits for drivers and random breath testing to enforce the limits were not associated significantly with alcohol consumption.

The authors conclude:

"Expansion of industrial production and marketing of alcohol is driving alcohol use to rise in emerging markets; cost-effective and affordable interventions to restrict alcohol-related harm exist, and are in urgent need of scaling up. With few civic organizations being present whose mandate is to reduce alcohol-related harms, there has been a lack of non-governmental organization engagement, while alcohol-industry funded organizations have promoted a 'partnership' role with governments to design national alcohol policies, as observed in some low-income countries. There is a need to develop public health infrastructures in those countries to develop, enact and then enforce comprehensive alcohol policies."

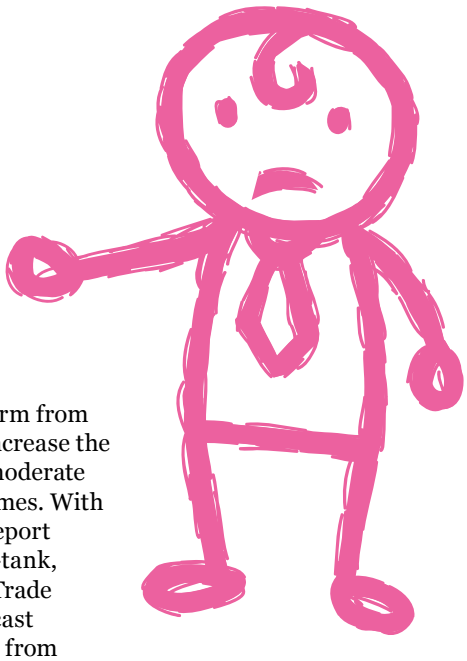


REFERENCE

Cook WK, Bond J, Greenfield TK. (2014). Are alcohol policies associated with alcohol consumption in low- and middle-income countries? Addiction. 2014 Jul;109(7):1081-90



UNDER THE INFLUENCE – AN INVESTIGATION INTO THE DEBATE ON MINIMUM PRICING FOR ALCOHOL IN ENGLAND



In March 2012 the British conservative prime minister, David Cameron, announced the government's decision to introduce a minimum price for a unit of alcohol. This would help reduce drinking in those who drink the most and a minimum price of 40 pence (approximately 5 Swedish kr), could mean 50 000 fewer crimes and 900 fewer alcohol-related deaths a year, according to the government. The prime minister acknowledged that the proposal wouldn't be universally popular, but said that "the responsibility of being in government isn't always about doing the popular thing. It's about doing the right thing."

A year later, in an extraordinary U-turn, the government announced that it had scrapped the proposal. The background to these events, the scientific evaluations and the lobbying efforts of the British alcohol industry, is described in a series of articles published in 2014, commissioned by British Medical Journal, BMJ, and written by the freelance journalist Jonathan Gornall.

The process started in 2008 when the Labour government commissioned a report from a research group at Sheffield University on the effect of different alcohol policy options, among them taxes and minimum prices. The results showed that an increase in level of minimum pricing would be followed by steep increases in reduction of harm. A 40p minimum price would reduce overall consumption by 2.6%, while 50p would cause a 6.9% drop in consumption. That population-wide measures, like general increases in prices, are the most effective to reduce harm is well known from research and the British Medical Association stated in a submission to the British government that "Measures need to be taken to reduce the mean level of consumption within the UK, as it is clear that the lower the mean level of consumption the fewer problems associated with alcohol, both for the individual and for society."

This is disapproved by the alcohol industry as such measures are potentially damaging to the companies' profits. In the industry campaign several reports from right-wing think-tanks were used. E.g. the Adam Smith Institute report "Minimal evidence for minimum pricing" declared that the Sheffield report was "entirely speculative and do not deserve the exalted status they have been afforded in the policy debate." The brewer SABMilller commissioned another report from the think tank The Centre for Economics and Business Research stating that minimum prices would make little difference to harm from alcohol misuse but increase the financial burden of moderate drinkers on low incomes. With the help of another report from the same think-tank, the Wine and Spirit Trade Association tried to cast doubts on the results from Sheffield University and launched a campaign with the message "Why should responsible drinkers pay more?"

This was combined with intense contacts from the industry with individual members of Parliament and governments departments. While the public health community had difficulties getting access to government officials for meetings industry representatives had almost daily contact with government departments. Jim McCambridge, researcher at the London School of Hygiene and Tropical Medicine have interviewed senior members of the industry for a study examining influence on health policy found that the industry lobbying was really about building long term relationships with key policy actors so influence can be exerted in very subtle ways and within these long term relationships you see quite astonishing levels of contact.

Strategies to counter demands for regulations were discussed within the alcohol industry as much as 30 years ago. In 1984 a senior executive in one of the drinks giants of the time, Grand Metropolitan, circulated a confidential memo where he proposed that the alcohol industry should learn from the mistakes of the tobacco industry. He wrote that "special interest groups" drew attention to the problems of alcohol abuse and demanding increased restrictions as higher taxes, restrictions on retail hours, advertising and marketing restrictions, and health warning and ingredient labels on drinks. The alcohol industry should instead promote the position that alcohol in moderation is good for the

consumer but in excess it is bad and he warned that if the media would concentrate on the harm of alcohol "voters will come to believe that alcohol is bad and the drinks industry irresponsible."

One way of achieving long term relations between political representatives and the alcohol industry are the all party groups in the British parliament. There are four such groups supporting and funded by the alcohol industry, for beer, for wine and spirits, for Scotch whisky and for cider. In total there are 472 all party groups in the parliament but the largest is the beer group with 300 MPs as members, almost half of all in the House of Commons, and 100 members from the House of Lords.

There is a big difference in resources and access to contacts with the public health community. John Holmes of Sheffield University's alcohol research unit, states that such groups "allow industry actors to . . . talk to lots of MPs in a way that just wouldn't be available to any public health group." Another disadvantage for the scientists is that the think tanks and the alcohol industry representatives are not really interested in whether their arguments are accurate or not. "It's all about creating doubt about what we're saying", say Holmes.

As a conclusion of the article series, Jonathan Gornall wrote "Five years on—and, by the government's estimate, hundreds if not thousands of avoidable deaths later— ... thanks to a combination of political self interest, libertarian ideology, and industry's unfettered ability to lobby the length and breadth of Westminster, 'big alcohol' had won the day."

REFERENCES

Gornall, J. (2014). Under the influence. BMJ 2014;348:f7646 doi: 10.1136/bmj.f7646 (Published 8 January 2014)

Gornall, J. (2014). Under the influence: 1. False dawn for minimum unit pricing, BMJ 2014;348:f7435 doi: 10.1136/bmj.f7435 (Published 8 January 2014)

Gornall, J. (2014). Under the influence: 2. How industry captured the science on minimum unit pricing, BMJ 2014;348:f7531 doi: 10.1136/bmj.f7531 (Published 8 January 2014)

Gornall, J. (2014). Under the influence: 3. Role of parliamentary groups, BMJ 2014;348:f7571 doi: 10.1136/bmj.f7571 (Published 7 January 2014)

Gornall, J. (2014). Under the influence: 4. Election prospects triumph over public health, BMJ 2014;348:f7610 doi: 10.1136/bmj.f7610 (Published 8 January 2014))











DOES REDUCTION IN DRINKING REDUCE RISK OF DEATH FOR INDIVIDUALS WITH ALCOHOL USE DISORDERS?

Alcohol use disorder is a medical diagnosis covering biological aspects, as tolerance to alcohol, withdrawal symptoms, psychological, like loss of control and behavioural aspects, as reduced occupational and recreational pursuits. Alcohol use disorders are among the most common mental disorders globally. In Europe it is the second largest mental disorder after depression, responsible for 6.2 per cent of all years lived with disability from chronic conditions.

A study summarizing 16 articles on cohort studies, i.e. groups of individuals that are followed several years, of persons with diagnosed with alcohol use disorders found that those who reduced their drinking reduced their risk of death substantially, compared to those who continued with heavy drinking. The studies were from USA, Europe, Sri Lanka and Japan. The mean time of follow up was almost nine years.

For those who reached abstention the risk of death was 35 per cent of the risk of death for those who continued heavy drinking. For those who substantially reduced their drinking without reaching abstention the risk of death was 61 per cent. Taken together the reduction in mortality was marked. Those who reduced their drinking had less than half the risk of those who continued their heavy drinking. Seen from the other perspective, people with continued heavy drinking had more than double the risk of death.

This result coincides with the results from studies on other and general populations. A reduction in alcohol consumption has been shown to result in a reduction of mortality in both aggregate-level and individual-level studies. People with alcohol use disorders also die from causes that are common in the general population, such as injury, heart disease, liver cirrhosis and cancer. The authors conclude that "while the magnitude of mortality reductions found in our meta-analysis may be specific to people with AUD [alcohol use disorders] from inpatient treatment settings, the underlying biological mechanisms may apply to other population groups".



REFERENCE

Roerecke M, Gual A, Rehm J. (2013). Reduction of alcohol consumption and subsequent mortality in alcohol use disorders: systematic review and meta-analyses. J Clin Psychiatry. 2013 Dec;74(12):e1181-9.





DOES ALCOHOL PRODUCTION AND CONSUMPTION INCREASE ECONOMIC GROWTH?

The production and sale of alcohol creates jobs in the alcohol trade and create revenue for the companies involved. It is therefore easy to imagine that the sale of alcohol is positive for the economy and economic growth. This is often stated by alcohol producers saying that the industry is a major contributor to the economy through job creation, wages and taxes. But a study from the US shows that this may not always be the case. For the economy as a whole, decreasing alcohol consumption, e.g. following an increase in taxes, increased total economic growth in the US during the period of study. This is the first study on the relation of alcohol consumption and total economic growth.

Labour productivity is one of the most important determinants of economic growth. Alcohol consumption lowers productivity in different ways. Drinking-related lost workdays can result from sickness absences, driving under the influence (DUI) arrests, alcohol-related injury work leaves, incarcerations due to alcohol-induced criminal activity, and alcohol-dependence-related withdrawals from the labour force. Several studies also show a direct association between alcohol use and reduced workplace performance measured through absenteeism, poor relations with co-workers, and accidents.

The importance of alcohol use for disease is also well documented. This includes injuries as well as chronic diseases and sexually transmitted infections. Diseases reduce the returns to human capital by lowering labour productivity as well as increasing lost workdays. Studies from different parts of the world show that alcohol use is costly to societies.

The US study used data from US states for the period 1971–2007, relating alcohol use to per capita gross domestic product (GDP) growth. The study focused on beer consumption as beer is the major source of alcohol consumption in the United States, making up approximately 55% of total alcohol consumption. Beer sales were therefore used as an indicator for alcohol consumption. When the researchers looked at total alcohol sales and economic growth the results were similar to those on per capita beer use.

The result for the study period is that a decrease in per capita consumption of one litre beer was associated with an increase of GDP growth of 1.7 per cent. An increase of beer tax, which lowers consumption, was hence associated to increase in economic growth.

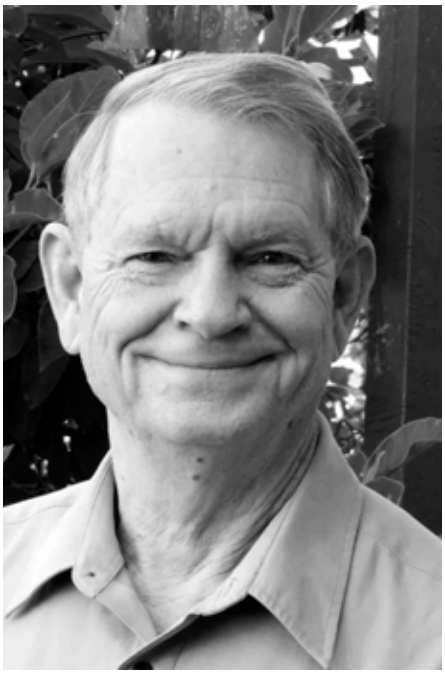
When looking at cigarette excise tax rate the study found strong relation to per capita cigarette consumption, but this did not have a significant impact on economic growth, possibly for the reason that smoking-induced costs have a much smaller effect on economic productivity than drinking.

The authors conclude that “the aggregate costs of the “bar tab” are indeed borne by society as a whole” and “Our results do, contrary to the claims of the Distilled Spirits Council and Beer Serves America, suggest that alcohol excise taxes do not harm economic activity at the aggregate level. If anything, our results show that beer excise tax rates are directly linked to economic growth.”



REFERENCE

Cesur R, Kelly IR (2014). Who pays the bar tab? Beer consumption and economic growth in the United States. Economic Inquiry, Volume 52, Issue 1, pages 477–494, January 2014



HAROLD HOLDER

Harold Holder, Chair of the research group. Harold D. Holder, Ph.D., is the former Director of the Prevention Research Center (PRC) of the Pacific Institute for Research and Evaluation, a national center for prevention research.

His published work has addressed a number of public policy studies including the impact of changes in retail sales of wine and spirits on drinking, alcohol-involved traffic crashes, and environmental strategies as part of comprehensive approaches to prevention. Dr. Holder has undertaken a series of collaborative studies in the Nordic Countries to study the effects of public policies. In addition, Dr. Holder has participated with prevention scientists from a dozen countries in international projects to document the effects of alcohol policy. The projects have produced three books in which he was a co-author, Alcohol Policy and the Public Good (1994), Alcohol: no ordinary commodity – Research and public policy (2003) and Alcohol: no ordinary commodity, second edition (2010).

Recently Dr. Holder chaired an international research group in an evaluation of Swedish research on alcohol, narcotics, doping, tobacco and gambling for the Swedish Council for Working Life and Social Research. The evaluation report was published in 2012.

Holder has published more than 150 scientific papers in refereed journals and collected volumes and was appointed to the National Advisory Council on Alcohol Abuse and Alcoholism, National Institutes of Health, effective 1998.



TANYA CHIKRITZHS

Professor Chikritzhs leads the Alcohol Policy Research team at the National Drug Research Institute. She has qualifications in epidemiology and biostatistics, some 15 years' experience in alcohol research and a national profile as an expert in her field. In 2012 she was awarded the prestigious Commonwealth Health Ministers Award for Excellence in Health and Medical Research.

Dr. Chikritzhs has published some 160 peer reviewed journal articles, reports and book chapters on evaluations of alcohol policy and alcohol epidemiology. Among these reports is the World Health Organization's International Guide for Monitoring Alcohol Consumption and Related Harm. She is Principal Investigator for high profile national projects such as the National Alcohol Indicators Project (NAIP) and the National Alcohol Sales Data project. The NAIP is Australia's central source of authoritative information on the epidemiology of alcohol in Australia and serves as a fundamental information base for the National Alcohol Strategies. Dr. Chikritzhs is regularly invited to contribute to public debate on alcohol issues in the media and has contributed to hundreds of radio and television articles.

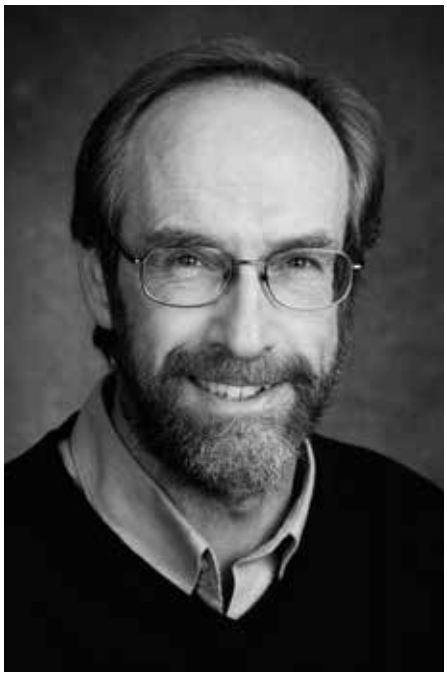


TIMOTHY NAIMI

Timothy Naimi, MD, MPH, is Associate Professor of Medicine at Boston University Schools of Medicine and Public Health. He is also active as physician at Boston Medical Center. Dr. Naimi's general fields of research are alcohol control policies, binge drinking, underage drinking and alcohol epidemiology.



Foto: emelie stachewsky





SVEN ANDRÉASSON

Sven Andréasson is the Adjunct Professor of Social Medicine at the Karolinska Institute's Department of Public Health Sciences. He is also a Senior Physician at the Stockholm Centre for Dependency Disorders and is in charge of operations at the Riddargatan 1 clinic.

Sven Andréasson's research involves both epidemiological studies of alcohol and narcotics usage in the population, and studies aimed at identifying the most effective methods of alcohol problem-related prevention.

TIM STOCKWELL

Tim Stockwell is Director of Centre for Addictions Research of British Columbia, Canada and Professorat the Department of Psychology, University of Victoria, BC, Canada.

Tim Stockwell has 35 years of experience as a researcher, administrator, educator and clinician in the UK, Australia and Canada. His research has covered many aspects of substance use policy, prevention, treatment methods, liquor licensing issues, taxation and the measurement of drinking patterns and their consequences. He has written over 200 peer reviewed journal articles and 100 books, chapters and reports. He has worked with international organisations (WHO, UNDCP) and many provincial, state and national governments.

He has also been Director of the Alcohol Education and Research Foundation in Australia and is a member of Canada's National Alcohol Strategy Advisory Committee and the WHO Technical Advisory Committee on Alcohol Epidemiology.

He holds degrees from Oxford University (MA Hons, Psychology and Philosophy), University of Surrey (MSc Clinical Psychology) and the University of London (PhD Institute of Psychiatry).

FRIDA DANGARDT

Dr Frida Dangardt is currently working as a physician and researcher at the Paediatric Physiology Department of the Queen Sylvia Paediatric Hospital in Gothenburg.

Frida Dangardt, who studied at the Sahlgrenska Academy at Gothenburg University, achieved her degree in medicine in 2005 and her doctorate in 2008. In 2012, she was awarded the AFA Försäkring insurance company's first ever post-doctoral scholarship in the field of public health. Between 2012 and 2014, she was a visiting researcher at the National Centre for Cardiovascular Prevention and Outcomes at University College London. Her particular area of interest is research into the development and prevention of cardiovascular disease in children and young people, focusing on childhood obesity, mental stress and alcohol consumption. She also conducts clinical research into the effects of chronic diseases on cardiovascular health in children.

Frida Dangardt's epidemiological research focuses on the effects of alcohol consumption on cardiovascular health and on how psychological well-being can affect the health of children, as part of UK's Avon Longitudinal Study of Parents and Children (ALSPAC), and in adults, as part of the UK-based 1946 Birth Cohort Study.

Frida Dangardt is currently also working on a number of clinical projects investigating cardiovascular changes in children with renal disease, both at the Great Ormond Street Hospital for Children and at Queen Sylvia's Paediatric Hospital in Gothenburg.



IOGT-NTO is Sweden's largest temperance organisation and its vision is of a society, a world, in which people are not prevented from living free and rich lives by alcohol and other drugs.

The Swedish Society of Medicine (SLS) is a non-profit organisation that is unaffiliated with any political party or trade union and whose primary mandate is to promote better healthcare for the patients of today and tomorrow.

IOGT·NTO

Street address: Gammelgårdsvägen 38, Stora Essingen
Box 12825, 112 97 Stockholm
Tel: +46 8 672 60 00
Fax: +46 8 672 60 01
Email: info@iogt.se
Website: www.iogt.se

Svenska Läkaresällskapet

Street address: Klara Östra Kyrkogata 10
Box 738, 101 35 Stockholm
Tel: +46 8 440 88 60
Fax: +46 8 440 88 99
Email: sls@sls.se
Website: www.sls.se