

CMO Alcohol Guidelines Review

**A summary of the evidence of the
health and social impacts of
alcohol consumption**

A report for the Health Evidence Expert Working Group
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About this document

This document was prepared on behalf of the Secretariat to the Health Evidence Expert Group by the Centre for Public Health, Liverpool John Moore University. The Health Evidence Expert Group was established by the UK Chief Medical Officers to review the evidence on the health impacts from alcohol.

The purpose of this document is to provide an overview of the evidence on the health and social impacts of alcohol consumption mapped against the terms of reference of the Health Evidence Expert Group. The interpretation, analysis and views expressed are those of the authors (Lisa Jones and Mark Bellis) and not necessarily those of the Health Evidence Expert Group.

Acknowledgements

In addition to the authors of the report we would like to acknowledge Geoff Bates and Ellie McCoy (Centre for Public Health, Liverpool John Moore University) for their contributions to the map of systematic review level evidence that informed this document. We would also like to acknowledge the contribution that Michela Morleo (Centre for Public Health, Liverpool John Moore University) made to early drafts of this document.

We also thank the members of the Health Evidence Expert Group for their review and comments on earlier version of this overview and Professors Jürgen Rehm and Tim Stockwell for their thorough peer review and helpful comments on this overview.

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Background to the review of alcohol guidelines

Previous guidelines

Sensible Drinking guidelines (1987)

Drinking less than 21 units per week by men and less than 14 units per week by women was unlikely to damage health. (One units of alcohol being defined as 8g or 10ml of pure alcohol).

Sensible Drinking guidelines (1995)

In 1994, the Government announced that the 1987 guidelines would be reviewed in light of evidence indicating that alcohol consumption might provide protection from coronary heart disease (CHD).¹ An Inter-Departmental Working Group was established to consider the evidence and the main findings were as follows:

- They wished to move away from weekly drinking to enable people to set daily benchmarks and account for the harms associated with heavy episodic drinking.
- Men were advised that regular consumption of between 3 to 4 units per day would not accrue significant health risk, and women, regular consumption of between 2 to 3 units was advised. Consistently drinking more than the respective maximums (4 or more units a day for men and 3 or more units a day for women) was not advised as a 'sensible drinking level' because of the progressive health risk it carried. The maximum health advantages for men and women were thought to lie between drinking 1 and 2 units per day.
- However, sensible drinking guidelines are not appropriate to those aged under 16 and after an episode of heavy drinking, individuals should refrain from drinking for two days to allow physiological recovery. There are a number of occasions where individuals should be advised not to drink: before/during driving; before or during active sport (especially swimming); before using machinery, electrical equipment or ladders; before/during working and when taking medication (where alcohol is contraindicated).
- Middle-aged or elderly men and post-menopausal women may wish to consider the possibility that light drinking could benefit their health.

Review of guidelines in 2012

In January 2012, the House of Commons Science and Technology Committee published an inquiry examining the evidence base for alcohol advice in order to assess whether the guidelines needed to be updated.² The inquiry, which received submissions from a range of stakeholders, noted a number of concerns from experts in relation to the 1995 guidelines, in particular that:

- The move to daily drinking limits could have appeared to endorse daily drinking; with the suggestion that many people may not be aware that the advice was framed in terms of regular drinking.
- More recent analyses have questioned the robustness of the evidence related to the health benefits of alcohol consumption; the primary rationale for the shift to daily guidelines.

The report by the House of Commons Science and Technology Committee said that they were disappointed by the lack of a review of the evidence since 1995 and that concerns about the current Government guidelines indicated that a thorough review of the evidence would be worthwhile and timely and would increase public confidence in the guidelines. Thus, the UK Chief Medical Officers (CMOs) have established two expert working groups to review the evidence and develop joint UK wide alcohol guidelines. The Health Evidence Expert Working Group has been asked to consider: (i) the science around the effects of alcohol on health and to agree assessments of risk associated with various levels of alcohol consumption and, if possible, with different patterns of consumption; (ii) whether the evidence suggests that current alcohol guidelines should be revised; and (iii) the evidence in terms of a life-course approach, building on current guidelines for young people and pregnancy, and to examine the possibility of different guidance for different age groups.

Development of low risk drinking guidelines

Internationally, the development of new national guidelines has most recently been undertaken in Australia and Canada.^{3,4} Development of both guidelines was based on comprehensive reviews of published evidence but different approaches were used to derive the recommended low risk levels of consumption. A summary of both guidelines is presented in Appendix 1.

The Australian guidelines are based on the absolute risk of acute and chronic outcomes and daily drinking levels were estimated which would increase lifetime risk of death, injury or chronic illness by more than 1 in 100.⁵ The Canadian guidelines were mainly based on a relative risk approach and show how different levels of consumption change pre-existing levels of risk.⁶ Estimates of daily levels of average alcohol intake and their risk relationship with a range of diseases and injuries compared to lifetime abstinence were developed.³ The overall risk of experiencing an increased risk of premature death was identified from comprehensive reviews and meta-analyses that summarised the risk of all-cause mortality, again in comparison to lifetime abstinence. Risk of premature death was used as one way of estimating the point at which the potential risks and benefits balanced each other out.³ Dawson has argued that drinking guidelines should reflect relative levels of risk; thus focusing on “that proportion of risk that is attributable directly to alcohol consumption and not on the proportions that reflect social and biological influences”.⁷

Research questions

The Health Evidence Expert Working Group has been asked to consider:

- The science around the effects of alcohol on health and to agree assessments of risk associated with various levels of alcohol consumption and, if possible, with different patterns of consumption;
- Whether the evidence suggests that current alcohol guidelines should be revised; and
- The evidence in terms of a life-course approach, building on current guidelines for young people and pregnancy, and to examine the possibility of different guidance for different age groups.

Based on the Expert Working Group's terms of reference the following key research questions were developed:

- 1) What are the health consequences arising from regular consumption of alcohol?
 - a) How do the risks of alcohol change with different levels consumption? Is it possible to assign different degrees of risk (e.g. lower risk, higher risk) to particular levels of alcohol consumption?
 - b) What are the impacts, if any, of having alcohol free days (zero consumption) within a pattern of regular alcohol consumption?
- 2) What are the health consequences arising from heavy or episodic 'binge' drinking of alcohol?
- 3) What are the beneficial effects, if any, of low to moderate consumption of alcohol?
- 4) What are the effects, both beneficial and harmful, of alcohol consumption on social and individual well-being?
- 5) Are there any changes in the direction, form or strength of the evidence for health and social impacts of alcohol consumption since the 1995 guidelines?
- 6) Are there any changes in the direction, form or strength of the evidence on alcohol and pregnancy since the 2008 NICE review?
- 7) Are there any changes in the direction, form or strength of the evidence on young people and alcohol since the 2009 CMO for England's guidance?

As a background, and to provide context to, the international evidence discussed in this report, Appendix 2 provides a summary of how much people in the UK drink.

1 What are the health consequences arising from regular consumption of alcohol?

Globally alcohol represents the fifth largest single cause of premature mortality, loss of health and disability.⁸ In 2010, alcohol use resulted in 2.7 million deaths and accounted for around 4% of global disability-adjusted life years (DALYs*).⁸ In the UK, alcohol use is one of the top 5 leading risk factors, with a substantial fraction of the burden of disease falling on those younger than 55 years.⁹ Epidemiological studies provide empirical evidence that these adverse impacts of alcohol result from its combined relationships with a wide range of health harms.¹⁰

There is clear and consistent evidence from epidemiological studies that alcohol consumption is associated with the development of a number of diseases and health problems (see Appendix 3 for considerations regarding the evidence). Where sufficiently reliable studies are available, methodological developments have further enabled the relationship between alcohol consumption and disease to be characterised, with the dose-response relationship for some conditions characterised as linear (i.e. all levels of alcohol consumption are associated with an increased risk of harm). For other conditions, including ischaemic stroke, ischaemic heart disease and type II diabetes, U- and J-shaped relationships have been described, indicating a beneficial effect of alcohol at some levels of consumption (see Section 3) and a detrimental effect at others. Sufficient good quality evidence of an association with average volume of alcohol consumption and a number of diseases and health problems is available;¹⁰ a summary of these disease and health problems and their risk relationship with alcohol consumption is summarised in Table 1. Alcohol consumption is also strongly associated with a range of acute consequences, including both intentional and unintentional injury, and most notably traffic accidents. As well as the impact of average consumption on the risk of injury, the proportion of heavy or binge drinking occasions in the overall volume of drinking (see Section 2), the physical and social availability of alcohol and drinking context play a role.

There are other conditions for which currently, sufficient evidence of an association with alcohol consumption has yet to be established, but that have been explored in published systematic reviews and meta-analyses. These conditions include: HIV/AIDS (for which there is sufficient evidence of an association between alcohol consumption and the course of disease but not on incidence);¹¹ stomach cancer;^{12,13} lung cancer;^{12,14} prostate cancer;^{12,15} endometrial cancer;¹⁶ bladder cancer;^{17,18} nasopharyngeal carcinoma;¹⁹ renal cell carcinoma;^{20,21} Hodgkin lymphoma;²² pancreatic cancer;²³ epithelial ovarian cancer;²⁴ Alzheimer's disease and other dementias;²⁵ age-related macular degeneration;²⁶ psoriasis;²⁷ and osteoporotic fracture.²⁸

* The DALY is a summary measure used to give an indication of the burden of disease. One DALY represents the loss of the equivalent of one year of full health.

Table 1. Summary of risk relationship between alcohol consumption and conditions with sufficient evidence of an association

Condition	Risk relationship ^a based on average volume of consumption ^b per day	Source(s)
Tuberculosis	Threshold; harmful effects >5 units or diagnosis of alcoholism	Lönnroth et al., 2008 ²⁹
Oral and pharyngeal cancer	Monotonic; harmful effects >0 units	Corrao et al., 2004; ³⁰ Tramacere et al., 2010a
Oesophageal cancer	Monotonic; harmful effects >0 units	Corrao et al., 2004; ³⁰ Islami et al., 2011 ³¹
Colorectal cancer	Monotonic; harmful effects >0 units	Corrao et al., 2004; ³⁰ Fedirko et al., 2011
Liver cancer	Monotonic; harmful effects >0 units	Corrao et al., 2004 ³⁰
Laryngeal cancer	Monotonic; harmful effects >0 units	Corrao et al., 2004; ³⁰ Islami et al., 2010 ³²
Female breast cancer	Monotonic; harmful effects >0 units	Key et al., 2006; ³³ Collaborative Group on Hormonal Factors in Breast Cancer, 2002 ³⁴
Type II diabetes	Males: U-shaped; nadir 3 units; reversion point 7.5 units. Females: U-shaped; nadir 3 units; reversion point 6 units	Baliunas et al., 2010 ³⁵
Epilepsy	Monotonic; harmful effects >0 units	Samokhvalov et al., 2010 ³⁶
Hypertensive heart disease	Males: Monotonic; harmful effects >0 units. Females: J-shaped; nadir 0.5 units, reversion point 2 units	Taylor et al., 2009 ³⁵
Atrial fibrillation	Harmful effects >1.5–3 units; effects of lower levels of consumption are unclear	Samokhvalov et al., 2010; ³⁷ Kodama et al., 2011 ³⁸
Ischaemic heart disease – Mortality	Males: J-shaped; nadir 4 units; reversion point 8 units. Females: J-shaped; nadir 1.5 unit; reversion point 4 units	Roerecke & Rehm, 2012 ³⁹
Ischaemic heart disease – Morbidity	Males: J-shaped; nadir 8.5 units; no reversion point. Females: J-shaped; nadir 2 units; reversion point 7 units	Roerecke & Rehm, 2012 ³⁹
Ischaemic stroke – Mortality	Males: J-shaped; nadir 1.5 units; reversion point 4.5 units. Females: J-shaped; nadir 1.5 units; reversion point 5.5 units.	Patra et al., 2010 ⁴⁰
Ischaemic stroke – Morbidity	Males: J-shaped; reversion point 4.5 units. Females: J-shaped; reversion point 5.5 units	Patra et al., 2010 ⁴⁰
Haemorrhagic stroke – Mortality	Males: Monotonic; harmful effects >0 units. Females: J-shaped; inverse association ≤1.5 units	Patra et al., 2010 ⁴⁰
Haemorrhagic stroke – Morbidity	Males: Monotonic; harmful effects >0 units. Females: J-shaped; nadir 1.5 units; reversion point 4.5 units	Patra et al., 2010 ⁴⁰
Pneumonia	Harmful effects >3 units; effects of lower levels of consumption are unclear	Samokhvalov et al., 2010 ⁴¹
Liver cirrhosis – Mortality	Monotonic; harmful effects >0 units	Rehm et al., 2010 ⁴²
Liver cirrhosis – Morbidity	Males: Threshold; harmful effects >4.5 units. Females: Threshold; harmful effects >3 units	Rehm et al., 2010 ^{42,c}
Pancreatitis	Threshold; harmful effects >6 units	Irving et al., 2012 ⁴³
Injury	Risk increases non-linearly; methodological issues impact significantly on the magnitude of the effects.	Taylor et al., 2010; ⁴⁴ Zeisser et al., 2013 ⁴⁵

^a *Monotonic* = increasing risk as the average volume of alcohol consumption increases. *Nadir* = lowest point of the curve for conditions with a U or J-shaped relationship. *Reversion point* = point on the curve where alcohol consumption becomes detrimental. ^b Number of units approximated from grams of alcohol (1 unit ≈ 8 grams). ^c Stockwell et al. suggest that such findings in relation to a protective effect of low to moderate alcohol consumption noted in this meta-analysis are biologically implausible.

Based on risk estimates presented in the document *Mapping systematic review level evidence* for conditions with sufficient evidence of an association with alcohol consumption from Rehm et al.¹⁰

a) How do the risks of alcohol change with different levels of consumption? Is it possible to assign different degrees of risk (e.g. lower risk, higher risk) to particular levels of alcohol consumption?

As shown in Tables 1 and 2, alcohol consumption affects the risks of health conditions in different ways. At lower levels of consumption, studies suggest alcohol consumption is associated with both increased health risks for some conditions (e.g. cancers, liver cirrhosis) and decreased health risks for others (e.g. ischaemic heart disease, ischaemic stroke).

Table 2. Relative risk of harm associated with selected alcohol-related conditions for men and women by average units per day

	No. of studies	Relative risk estimate by average units per day		
		3 units	6 units	12.5 units
Oral and pharyngeal cancer	15	1.86	3.11	6.45
Oesophageal cancer	14	1.39	1.93	3.59
Laryngeal cancer	20	1.39	1.93	3.59
Colon cancer	16	1.05	1.10	1.21
Rectal cancer	6	1.09	1.19	1.42
Liver cancer	10	1.19	1.40	1.81
Female breast cancer	29	1.25	1.55	2.41
Hypertension	2	1.43	2.04	4.15
Ischaemic heart disease	28	0.81	0.87	1.13
Ischaemic stroke	6	0.90	1.17	4.37
Haemorrhagic stroke	9	1.19	1.82	4.70
Liver cirrhosis	9	2.90	7.13	26.52
Chronic pancreatitis	2	1.34	1.78	3.19
Injuries and violence	12	1.12	1.26	1.58

Based on relative risk estimates from Corrao et al., 2004.³⁰

Relative risk estimates from studies of all-cause mortality can provide some indication of the balance of the health risks and benefits associated with different levels of consumption. Meta-analyses of prospective all-cause mortality studies have demonstrated a J-shaped relationship between total mortality and average alcohol volume. In the a recent meta-analysis of alcohol consumption and all-cause mortality,⁴⁶ a low level of alcohol consumption (apparent from <1 unit/day, up to around 2 units/day for women and up to 5 units/day for men) was associated with a reduced risk of death (resulting mainly from the beneficial effects of alcohol consumption on ischaemic heart disease) and higher levels of consumption associated with an increased risk of death. However, there are problems with studies of all-cause mortality.^{47,48} The association between all-cause mortality and alcohol consumption depends on average volumes and patterns of drinking in the population under study. Most of the physiological mechanisms that have been suggested to explain the protective effect of moderate drinking only apply for cohorts with overall low levels of consumption and patterns of regular drinking that do not vary.⁴⁷ For cohorts that include drinkers with heavy or binge drinking occasions in their overall volume of drinking, light to moderate drinking has no protective effect. The distribution of age and causes of death in cohorts for studies of all-

cause mortality may also overestimate the protective effect of alcohol.⁴⁸ Compared to the general population, such cohorts generally have higher life expectancies and different causes of death. In the UK, as in other developed countries, mortality rates from cardiovascular disease have declined steadily during the late 20th century,⁴⁹ and cohorts may therefore historically be more influenced by deaths from cardiovascular disease. In addition, potential confounders that may influence the relationship between alcohol consumption and disease have been identified (see Section 3 and Gmel et al.⁵⁰ for further discussion; e.g. the sick-quitter hypothesis, the length of follow-up hypothesis, the cultural variation hypothesis and the age hypothesis) and further factors remain unexplained.

b) What are the impacts, if any, of having alcohol free days (zero consumption) within a pattern of regular alcohol consumption?

Recommendations on “drink free days” formed part of the 1995 Sensible Drinking guidelines,¹ which suggested that following an episode of heavy drinking “it is advisable to refrain from drinking for 48 hours to allow tissues to recover...” but noting that “such breaks are not required on health grounds for people drinking within the recommended benchmarks”. No systematic reviews or meta-analyses since 1995 have examined how alcohol free days within a pattern of regular consumption impact on the development of health or social harms and as such there is a lack of evidence on which to determine whether there is any beneficial effect from having days free from alcohol consumption. However, within a pattern of regular consumption it is plausible that adopting alcohol free days may be a useful way for drinkers to moderate their consumption.

With reference to populations of very heavy drinkers and those with alcohol dependency, findings from selected primary studies provide some evidence that drinking once or twice per week may be associated with a lower risk of developing liver cirrhosis than daily drinking.⁵¹⁻⁵³ For example, a Danish study⁵¹ of heavy drinkers[†] found that after adjusting for age, average number of drinks per day, duration of alcohol misuse and predominant type of alcohol consumed, men who reported pauses in their drinking of one or more days in the last month had a significantly lower risk of alcoholic cirrhosis mortality (RR 0.56; 95% CI: 0.37–0.85) than men who reported drinking every day. A trend towards lower risk was also found among women who reported pauses in their drinking of one or more days in the last month (vs. drinking every day: RR 0.55; 95% CI: 0.29–1.02) after adjusting for age, average number of drinks per day, duration of alcohol misuse, predominant type of alcohol consumed, marital status, housing status and work situation. A US study⁵⁴ that examined drinking patterns in relation to incidence of liver disease found a much greater magnitude of effect for the association between daily/near daily risk drinking[‡] (vs. never risk drinking: OR 4.76; 95% CI 2.29–9.88) than for engaging in risk drinking once or twice a week (vs. never risk drinking: 2.78; 95% CI 1.32–5.85). Further a Japanese study⁵⁵ that examined the “liver holiday” hypothesis found that a

[†]The mean number of drinks per day reported by participants corresponded to 30 units/day in men and 25.5 units/day in women.

[‡]Risk drinking was defined as drinking five or more alcoholic drinks (equivalent to 14 g) in a single day for men and four or more alcoholic drinks in a single day for women; corresponding to 70 units in a single day for men and 56 units for women.

significant relationship was apparent for heavy drinkers. Heavy drinkers showed an increased risk of all-cause mortality when alcohol was consumed over 5–7 days. Compared to occasional drinkers (those drinking on 1-3 days in a month), men drinking ≥ 37.5 units/week and on 5 to 7 days in the week showed a significantly increased risk of all-cause mortality (≥ 37.5 to < 56 units: HR 1.29; 95% CI 1.12–1.50; ≥ 56 units: HR 1.55; 95% CI 1.32–1.81). At lower levels of consumption there appeared to be no relationship between risk of all-cause mortality and frequency of alcohol intake.

2 What are the health consequences arising from heavy or episodic ‘binge’ drinking of alcohol?

Both average consumption and episodic drinking occasions play an important role in the development of alcohol-related diseases, with heavy drinking occasions particularly contributing to injury and other acute health problems. The now widely adopted definition of binge drinking is of an intake of large volumes of alcohol on a single occasion, but differences in operational definitions remain.⁵⁶ As discussed in Section 1b, patterns of consumption matter such that heavy or binge drinking on one occasion every month has different risks for the development of chronic diseases (such as liver cirrhosis) compared with heavy or binge drinking almost every day.⁵⁴ The association between heavy or binge drinking occasions and disease development has been examined in very few meta-analyses. A recent meta-analysis by Roerecke and Rehm found that any cardioprotective effects of moderate alcohol consumption were cancelled out by irregular heavy drinking occasions (≥ 7.5 units per occasion at least monthly) mixed with an average frequency of low to moderate consumption.⁵⁷ Heavy or episodic ‘binge’ drinking of alcohol during early pregnancy may be particularly harmful to the developing foetus,^{58,59} this is discussed further in Section 5.

While the relationship between alcohol consumption and increased risk of injury is well established, important methodological issues are commonly encountered in the literature.⁴⁵ For example, many studies (and consequently some meta-analyses) that examine the relationship between alcohol consumption and injury estimate the level of alcohol consumption from blood alcohol concentrations (BAC). As noted by Gmel et al.,⁵⁶ this presents a problem with distinguishing between single occasion binge drinking and chronic heavy drinking. Studies that have distinguished the impact of heavy episodic drinking indicate that it is more strongly related to injury than volume of drinking.⁶⁰ While all groups of drinkers, regardless of frequency and intake are at increased risk for alcohol-related injury, an emergency room study by Gmel et al.⁶⁰ showed that despite drinking approximately the same amount of alcohol in the 6 hours before injury, those who normally drank low levels of alcohol, but on occasion drank heavily[§], were at higher risk of alcohol-related injury than chronic heavy drinkers^{**}.

In a comprehensive meta-analysis of acute alcohol consumption and injury risk, Taylor et al.⁴⁴ found that the risk of injury increases non-linearly with increasing alcohol consumption. For every 10g (1.25 units) increment in consumption, the odds ratio (OR) increased by 1.24 (95% CI: 1.18–1.31) for motor vehicle accidents and by 1.30 (95% CI: 1.26–1.34) for non-motor vehicle injury. However, Zeisser et al. note that many of the studies included in this meta-analysis estimated the level of alcohol consumption from measured BAC.⁴⁵ Further, Zeisser et al.⁴⁵ have found that methodological issues with studies of alcohol and injury have significant effects on the magnitude of effect size estimates. For example, the use of case-crossover designs, which involves using patients as their

[§] Defined as drinking 5+ drinks for men and 4+ drinks for women.

^{**} Defined as drinking more than 7 drinks for women and 14 drinks for men a week as their usual volume, but no heavy episodic drinking in the past month.

own control, results in the overestimation of injury risk. Stockwell et al.⁶ also note that this type of study design fails to account for other confounding factors, and the role of context and extrinsic factors in alcohol-related injury. The paper by Zeisser et al. highlights findings from two 'appropriately designed' population case-control studies that did attempt to control for confounding.⁴⁵ Overall these two studies reported ORs for the risk of injury at any level of drinking of 3.74 (95% CI: 1.49–9.40)⁶¹ and 1.48 (95% CI: 1.26– 1.73).⁶²

3 What are the beneficial effects, if any, of low to moderate consumption of alcohol?

Meta-analyses have identified that drinking alcohol may have a protective effect at low levels of consumption compared to not drinking for a limited number of conditions; including, from published meta-analyses, extrahepatic bile system cancer;⁶³ renal cell cancer;^{20,21} type II diabetes;³⁵ dementia and cognitive decline;²⁵ hip fracture;²⁸ ischaemic heart disease;^{39,64,65} and ischaemic stroke.⁴⁰ In fact, Fekjær notes that studies provide ‘evidence’ that light to moderate drinking prevents 29 diseases and health problems.⁶⁶ However, unresolved confounding and other factors may explain the apparent protection observed. For example, it has been suggested that the misclassification of drinking groups,^{††} such as the failure to separate former and occasional drinkers from lifetime abstainers may create a strong bias towards less healthy individuals being more likely to reduce or quit drinking.^{67,68} A few meta-analyses have sought to account for such bias; for example a recent meta-analysis, which reported that light to moderate alcohol consumption was associated with a reduced risk of cardiovascular outcomes, included lifetime abstainers as a reference category in sensitivity analyses.⁶⁵ However, Stockwell et al. question the robustness of the conclusions generated from this literature, noting that all but two studies included in meta-analysis by Ronksley et al. had “serious methodological problems”.⁶⁹

Currently, based on the extent to which this misclassification of drinking groups and other biases can be accounted for, compared with lifetime abstainers, a protective association has been reported in meta-analyses of the association between alcohol consumption and type II diabetes,³⁵ ischaemic heart disease,³⁹ and ischaemic stroke.⁴⁰ However, age, ethnicity, gender, type of alcoholic beverage, and patterns of consumption may also influence the relationship. For example, a large cohort study of alcohol and mortality risk found substantial age differences in risk with apparent benefits from light drinking seen only among those aged 40 years or older.⁷⁰ Further reasons to suggest that the beneficial effects of alcohol consumption may currently be overestimated, include evidence of publication bias towards papers finding cardioprotective effects, and a failure to control for other lifestyle factors that are likely to reduce cardiac risk (e.g. healthy diet, regular exercise).⁷¹ Indeed, Fekjær observes that the majority of diseases and health problems for which alcohol has an apparent protective effect are the so-called ‘lifestyle diseases’.⁷² Alcohol consumption is an indicator of ‘optimal’ social status and studies show that abstainers are more likely to have unhealthy lifestyles and poorer psychosocial factors.⁷²

^{††} Misclassification of drinking groups include: (i) former drinkers being classified as abstainers; (ii) occasional drinkers being classified as abstainers; and (iii) occasional drinkers being classified as moderate drinkers. All three types of misclassification may make groups of moderate drinkers seem healthier.

4 What are the effects, both beneficial and harmful, of alcohol consumption on social and individual well-being?

While individuals derive pleasure from consuming alcohol and it can act as a catalyst in social interactions and leisure experiences, from a social and individual perspective, the consumption of alcohol has both benefits and consequences. The significance of the social benefits and harms arising from alcohol use have yet to be systematically examined,^{73,74} however, there is increasing recognition of the need to quantify the social consequences of alcohol consumption. Among young people, studies from Australia and the USA suggest that the association between alcohol consumption and drinking-related problems may increase as frequency and quantity increase but that strong associations exist at levels of consumption greater than two drinks per occasion (equivalent to between 2.5 to 3.5 units).^{75,76}

The social consequences of alcohol consumption also include social and economic costs to society and damage to third parties (termed ‘collateral damage’). Internationally, many studies have examined the social and economic costs of alcohol use. Table 3 summarises the estimated costs of alcohol-related harms in the UK from a review of available cost-of-illness studies in 2009.⁷⁷ Since 2009, the Department of Health has estimated the costs of alcohol-related harms in England as follows: NHS costs £3.5 billion per year at 2009–10 costs; lost productivity £7.3 billion at 2009–10 costs; and alcohol-related crime £11 billion per year at 2010–11 costs.⁷⁸

Table 3. Summary of various estimates of the costs of alcohol-related harms in the UK

Component	Country (Year of estimate)				
	England (2000/01)	Scotland (2001/02)	Scotland (2002/03)	Scotland (2006/07)	Scotland (2007)
Healthcare	1,383 – 1,683	96	110.5	405	267.8
Social care	-	85.9	96.7	170	230.5
Criminal justice system	11,940	267.9	276.7*	385*	727.1
Workplace	5,194 – 6,421	404.5	417.8	820	865.7
Human costs	-†	216.7	223.8	-†	1,464.6
Total (£ millions)	18,571-20,044	1,071	1,126	2,250	3,556

* Includes fire service expenditure. † No cost estimates presented.

The impact of alcohol on others is extensive, ranging from minor inconvenience to more severe impacts such as alcohol-related road traffic deaths and interpersonal violence.⁷⁹ Within communities, people may perceive that the alcohol use of others has an impact on their lives. For example, a study of alcohol and its impact on people and communities in the North West of England⁸⁰ showed that a high proportion of people had concerns about drunken behaviour and that a significant number felt the need to avoid town centres at night because of drunkenness. In Edinburgh, a

quarter of people thought street drinking or alcohol public disorder was a problem in their local area.⁸¹ Alcohol-related litter can also be a significant issue in communities.⁸² Alcohol use can disrupt family structures and functions.⁸³ Alcohol use is a contributory factor in intimate partner violence (IPV),⁸⁴ and a recent meta-analysis found clear evidence that alcohol use and IPV are associated for both males and females.⁸⁵ Parenting capacity is also affected by alcohol use and children living with parental alcohol misuse may experience neglect or abuse.⁸⁶ Internationally, studies have reported a rate of involvement of alcohol in 13-70% of substantiated child protection cases, with an Australian study showing that parental alcohol misuse is related to more intensive child protection outcomes.⁸⁷

Studies have begun to accumulate in recent years on the impact of the drinking of others. Two comprehensive reports have recently been produced that provide a broad overview of the problems faced from national perspectives.^{81,88} The first of these examined harms from an Australian perspective and estimated that in 2005, over 70,000 Australians were victims of alcohol-related assault, including 24,000 victims of alcohol-related intimate partner violence.⁸⁸ In addition, almost 20,000 Australian children were estimated to be victims of substantiated alcohol-related child abuse. The second report, on the impact of the drinking of others in Scotland, documented the wide range of harms experienced among the population both in public and private settings.⁸¹ The key findings of the survey are summarised in Box 1 below.

Box 1. Key findings from a Scottish survey of the second hand effects of drinking

- 1 in 2 people report one or more harms as a result of someone else's drinking.
- More than 1 in 3 report having heavy drinkers in their lives.
- People under 35 are four times more likely to report harm from others drinking in a public setting (street, public space, traffic, workplace).
- Those who know heavy drinkers are more likely to report harm from others drinking in private settings (home, family, friends, neighbours, private parties).
- Experiencing harm from other people's drinking is not related to whether the person affected by the harm drinks or not.
- Those who experience harm from someone else's drinking report lower life satisfaction compared to others.

Hope et al. (2013)⁸¹

The CMO Annual Report 2008 was concerned with raising awareness of the impacts of "passive drinking", highlighting the harms arising to the unborn fetus, acts of drunken violence, vandalism, sexual assault and child abuse, and the health burden on the NHS and friends and family affected by others alcohol use.⁸⁹ A selected summary of data from UK sources on the damage from alcohol on others, collated by Professor Bellis and colleagues at the Centre for Public Health, Liverpool John Moores University, is presented in Table 4.

Table 4. Damage from alcohol use: consequences for other people and communities

Measure	Total incidents	% alcohol-related	Estimated no. alcohol-related incidents/year	Source	Year	Coverage	Definition/Explanation
All violence	2,164,000	45%	947,000-991,730	British Crime Survey ⁹⁰	2007/08	England & Wales	Proportion of incidents where the victim believed the offender(s) to be under the influence of alcohol
Domestic violence	342,000	37%	126,540				
Violence against acquaintances	765,000	48%	367,200				
Violence against strangers	744,000	58%	431,520				
Mugging	391,000	17%	66,470	British Crime Survey ⁹¹	2006/07	England & Wales	Proportion of serious sexual assaults where the victim believed the perpetrator was under the influence of alcohol
All sexual assaults	528,485	Unknown	Unknown				
Serious sexual assaults	99,091	40%	39,225				
Children referred to children's social services	538,500	Unknown	Unknown	Department for Children, Schools & Families ⁹²	2008	England	All children referred to social services
	A study in London found 23% of social work cases involved parental alcohol misuse			Forrester & Harwin ⁹³	NA	London	All case files going for 'long-term allocation' with concerns noted about parental substance misuse
Drink driving casualties	7,910	100%	7,910	Department for Transport ⁹⁴	2006	UK	Casualties (slight, serious or fatal) in drink driving incidents who were not themselves the drunk driver*
Foetal alcohol syndrome	Unknown	100%	Unknown	Morleo et al. ⁹⁵	2007	England	No direct measure available for UK
Affected by maternal alcohol use	NA	100%	7.8 per 100,000 live births				
Vandalism	2,689,000	47%	1,263,830	British Crime Survey NEW-ADAM	2007/08	England & Wales	Incidents of vandalism reported in the British Crime Survey with % alcohol related taken from NEW-ADAM

*Includes pedestrians, cyclists, car drivers under the limit and car passengers, excludes car drivers over the limit, motorcyclists and others.

5 New evidence on alcohol and pregnancy

Currently all UK Chief Medical Officers' provide precautionary advice that women who are pregnant or trying to conceive should avoid alcohol. The guidelines were revised in 2007 stating that: '*Women who are pregnant or trying to conceive should avoid alcohol altogether. However, if they do choose to drink, to minimise the risk to the baby, we recommend they should not drink more than 1-2 units once or twice a week and should not get drunk.*' Additionally in England, updated NICE guidelines on the care and support that women should receive during pregnancy included the advice to avoid alcohol in order to minimise the risk of miscarriage in the first trimester.⁹⁶ Scotland's Chief Medical Officer subsequently developed a stronger precautionary message that drinking no alcohol during pregnancy is the best and safest choice.

The recent guidelines on the consumption of alcohol during pregnancy took into account the findings of a systematic review that evaluated the foetal effects of low to moderate prenatal alcohol exposure (equivalent to maximum 1.5 units or 12 g of alcohol daily) and binge drinking (most often ten defined as five or more drinks on any one occasion).⁹⁷ The review did not find consistent evidence of adverse effects from low-to-moderate prenatal alcohol consumption, however the authors noted that the evidence was not strong enough to rule out any risk.^{97,98} Most of the studies included in the review that examined risk of preterm birth, stillbirth and miscarriage found no association with low to moderate alcohol intake, and studies that did report increased risk had significant limitations. Gray and Henderson also examined the foetal effects of binge drinking finding that there were no consistently significant effects; with the exception of an indication of generally small effects on neurodevelopmental outcomes.^{97,99} They concluded that "at relatively low amounts of alcohol and infrequent occasions of binge-drinking, there is no consistent evidence of adverse effects. However, greater frequency of bingeing or higher levels of alcohol consumption may increase the risk of adverse foetal effects".⁹⁹ Taken together the evidence appears to suggest that the risk of foetal effects arising from single or rare episodes of binge-drinking not associated with a consistently high intake of alcohol may be small.^{56,99}

Since 2008, a meta-analysis has been published that investigated the effect of alcohol consumption during pregnancy on the risk of low birth weight, preterm birth and small for gestational age.¹⁰⁰ Alcohol consumption during pregnancy was associated with higher risk of developing all three complications. The meta-analysis shows that risk was elevated at consumption greater than 1-2 units per day and increased in a dose-dependence fashion thereafter. Both Henderson et al.⁹⁸ and Patra et al.¹⁰⁰ acknowledge weaknesses in the evidence base which preclude the assumption that consumption below these levels during pregnancy may be considered 'safe'. Henderson et al. suggest that one possible explanation for a lack of evidence of harm from small amounts of alcohol may be related to the 'healthy drinker effect'. That is, much like the sick quitter hypothesis (as discussed in Section 3), women with a poor obstetric history may be more likely to abstain from alcohol.⁹⁸

A summary of the evidence of the foetal effects of alcohol consumption in pregnancy is presented in Table 5 below.

Table 5. Summary of risk relationship between alcohol consumption in pregnancy and conditions originating in the perinatal period

Condition	Risk relationship ^a based on average volume of consumption ^b per day	Source(s)
Low birth weight	Threshold; harmful effects >1 unit; monotonic thereafter. Inadequate evidence for a causation of alcohol during pregnancy at levels of consumption below this.	Patra et al., 2011; ¹⁰⁰ Gray & Henderson, 2007 ^{97,98}
Preterm birth	Threshold; harmful effects >2 units. Inadequate evidence for a causation of alcohol during pregnancy at levels of consumption below this.	Patra et al., 2011; ¹⁰⁰ Gray & Henderson, 2007 ^{97,98}
Small for gestational age/Intrauterine growth restriction	Threshold; evidence of harmful effects >1 unit. Inadequate evidence for a causation of alcohol during pregnancy at levels of consumption below this.	Patra et al., 2011; ¹⁰⁰ Gray & Henderson, 2007 ^{97,98}
Miscarriage	Inadequate evidence for a causation of alcohol during pregnancy at low to moderate levels of consumption.	Gray & Henderson, 2007 ^{97,98}
Stillbirth	Inadequate evidence for a causation of alcohol during pregnancy at low to moderate levels of consumption.	Gray & Henderson, 2007 ^{97,98}
Malformations	Inadequate evidence for a causation of alcohol during pregnancy at low to moderate levels of consumption.	Gray & Henderson, 2007 ^{97,98}
Neurodevelopmental outcomes	Some evidence of a possible effect of binge drinking (five or more drinks on a single occasion; equivalent to 60g / 7.5 units).	Henderson et al., 2007 ⁹⁹

^a *Monotonic* = increasing risk as the average volume of alcohol consumption increases. *Nadir* = lowest point of the curve for conditions with a U or J-shaped relationship. *Reversion point* = point on the curve where alcohol consumption becomes detrimental. ^b Number of units approximated from grams of alcohol (1 unit ≈ 8 grams).

Based on risk estimates presented in the document *Mapping systematic review level evidence*.

6 New evidence on young people and alcohol

In 2009, the Chief Medical Officer for England published specific guidance on the consumption of alcohol by children and young people.¹⁰¹ Many factors play a part in the development of adolescent drinking and there is a large body of literature that has sought to establish the factors that put young people at risk of alcohol use, and also the factors that are protective against the early initiation of alcohol use and against problematic alcohol use later in adolescence. These factors are summarised in the epidemiological review of harms that accompanied the CMO guidance. The CMO guidance was based on evidence that an early age of drinking onset was associated with an increased likelihood of developing alcohol abuse or dependence in adolescence and adulthood, and also dependence at a younger age.^{102,103} Studies also showed that children and adolescents who begin drinking at a young age, typically below the age of 13, drink more frequently and in greater quantities than those who delay drinking, and are more likely to drink to intoxication. As with alcohol dependence and abuse, vulnerability to alcohol misuse in later adolescence appeared to be greatest among those who began drinking prior to age 13 years. Based on this evidence, the guidance therefore stated that an alcohol free childhood was the most desirable option and that drinking onset should be delayed for as long as possible (at least until the age of 15 years). The guidance further stated that if 15-17 year olds did choose to drink alcohol, that this should only occur under the supervision of a parent/carer, should not occur more than once a week, and should not exceed the maximum daily units for adults (females: 2-3 units; males: 3-4 units). Studies found that young people who binge drank in adolescence were more likely to experience negative outcomes in the transition to adulthood. Binge drinking at this age was linked to a higher likelihood of involvement in other substance use, crime, lower educational attainment and drug dependence.¹⁰⁴⁻¹⁰⁶

Since the publication of the CMO guidelines for young people, two systematic reviews of cohort studies have been published on the consequences of late adolescent drinking¹⁰⁷ and on age of first drinking.¹⁰⁸ Studies included in the review of late adolescent drinking provided evidence of associations with subsequent drinking in adulthood.¹⁰⁷ Those who drank heavily in late adolescent were likely to continue drinking heavily into adulthood and heavy drinking during late adolescent was associated with alcohol problems including dependence. However, the authors concluded that there was inadequate evidence to draw conclusions on the causal inferences of late adolescent drinking on broader health and social consequences, noting that the “evidence indicates that other factors indicative of heightened psychosocial risk more broadly are also implicated” in the development of drinking behaviours in adulthood. The review of age at first drinking found that there was a lack of evidence for a causal relationship. A recently published prospective study¹⁰⁹ adds to accumulating evidence that early onset of drinking tends to co-occur with other risk factors for drinking problems in later life such conduct problems and parental heavy drinking.¹¹⁰

References

1. Department of Health. *Sensible drinking - the report of an inter-departmental working group*. London: Department of Health; 1995.
2. House of Commons Science and Technology Committee. *Alcohol guidelines: eleventh report of session 2010-12*. London: The Stationery Office; 2012.
3. Butt P., Beirness D., Gliksman L., Paradis C., Stockwell T. *Alcohol and health in Canada: a summary of evidence and guidelines for low-risk drinking*. Ottawa: Canadian Centre on Substance Misuse; 2011.
4. National Health and Medical Research Council. *Australian guidelines to reduce health risk from drinking alcohol*. Canberra: National Health and Medical Research Council; 2009.
5. Rehm J., Room R., Taylor B. Method for moderation: measuring lifetime risk of alcohol-attributable mortality as a basis for drinking guidelines. *International Journal of Methods in Psychiatric Research* 2008; 17: 141-51.
6. Stockwell T., Butt P., Beirness D., Gliksman L., Paradis C. The basis for Canada's new low-risk drinking guidelines: a relative risk approach to estimating hazardous levels and patterns of alcohol use. *Drug and Alcohol Review* 2012; 31: 126-34.
7. Dawson D. A. Low-risk drinking limits: absolute versus relative risk. *Addiction* 2009; 104: 1303-4.
8. Lim S. S., Vos T., Flaxman A. D., Danaei G., Shibuya K., Adair-Rohani H. *et al.* A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2012; 380: 2224–60.
9. Murray C. J. L., Richards M. A., Newton J. N., Fenton K. A., Anderson H. R., Atkinson C. *et al.* UK health performance: findings of the Global Burden of Disease Study 2010. *Lancet* 2013; 381: 997–1020.
10. Rehm J., Baliunas D., Borges G. L. G., Graham K., Irving H., Kehoe T. *et al.* The relation between different dimensions of alcohol consumption and burden of disease: an overview. *Addiction* 2010; 105: 817-43.
11. Baliunas D., Rehm J., Irving H., Shuper P. Alcohol consumption and risk of incident human immunodeficiency virus infection: a meta-analysis. *International Journal of Public Health* 2010; 55: 159-66.
12. Bagnardi V., Blangiardo M., La Vecchia C., Corrao G. Alcohol consumption and the risk of cancer: a metaanalysis. *Alcohol Research & Health* 2001; 25: 263-70.
13. Tramacere I., Negri E., Pelucchi C., Bagnardi V., Rota M., Scotti L. *et al.* A meta-analysis on alcohol drinking and gastric cancer risk. *Annals Of Oncology* 2012; 23: 28-36.
14. Bagnardi V., Rota M., Botteri E., Scotti L., Jenab M., Bellocco R. *et al.* Alcohol consumption and lung cancer risk in never smokers: a meta-analysis. *Annals Of Oncology* 2011; 22: 2631-9.
15. Rota M., Scotti L., Turati F., Tramacere I., Islami F., Bellocco R. *et al.* Alcohol consumption and prostate cancer risk: a meta-analysis of the dose–risk relation. *European Journal of Cancer Prevention* 2012; 21: 350-9.

16. Sun Q., Xu L., Zhou B., Wang Y., Jing Y., Wang B. Alcohol consumption and the risk of endometrial cancer: a meta-analysis. *Asia Pacific Journal of Clinical Nutrition* 2011; 20: 125-33.
17. Mao Q., Lin Y., Zheng X., Qin J., Yang K., Xie L. A meta-analysis of alcohol intake and risk of bladder cancer. *Cancer Causes & Control* 2010; 21: 1843-50.
18. Pelucchi C., Galeone C., Tramacere I., Bagnardi V., Negri E., Islami F. *et al.* Alcohol drinking and bladder cancer risk: a meta-analysis. *Annals Of Oncology* 2012; 23: 1586-93.
19. Chen L., Gallicchio L., Boyd-Lindsley K., Tao X., Robinson K. A., Kim Lam T. *et al.* Alcohol consumption and the risk of nasopharyngeal carcinoma: a systematic review. *Nutrition and Cancer* 2008; 61: 1-15.
20. Bellocco R., Pasquali E., Rota M., Bagnardi V., Tramacere I., Scotti L. *et al.* Alcohol drinking and risk of renal cell carcinoma: results of a meta-analysis. *Annals Of Oncology* 2012; 23: 2235-44.
21. Song D. Y., Song S., Song Y., Lee J. E. Alcohol intake and renal cell cancer risk: a meta-analysis. *British Journal of Cancer* 2012; 106: 1881-90.
22. Tramacere I., Pelucchi C., Bonifazi M., Bagnardi V., Rota M., Bellocco R. *et al.* A meta-analysis on alcohol drinking and the risk of Hodgkin lymphoma. *European Journal of Cancer Prevention* 2012; 21: 268-73.
23. Tramacere I., Scotti L., Jenab M., Bagnardi V., Bellocco R., Rota M. *et al.* Alcohol drinking and pancreatic cancer risk: a meta-analysis of the dose-risk relation. *International Journal of Cancer* 2010; 126: 1474-86.
24. Rota M., Pasquali E., Scotti L., Pelucchi C., Tramacere I., Islami F. *et al.* Alcohol drinking and epithelial ovarian cancer risk. A systematic review and meta-analysis. *Gynecologic Oncology* 2012; 125: 758-63.
25. Anstey K. J., Mack H. A., Cherbuin N. Alcohol consumption as a risk factor for dementia and cognitive decline: meta-analysis of prospective studies. *American Journal of Geriatric Psychiatry* 2009; 17: 542-55.
26. Chong E. W.-T., Kreis A. J., Wong T. Y., Simpson J. A., Guymer R. H. Alcohol consumption and the risk of age-related macular degeneration: a systematic review and meta-analysis. *American Journal of Ophthalmology* 2008; 145: 707-15.
27. Zhu K. J., Zhu C. Y., Fan Y. M. Alcohol consumption and psoriatic risk: A meta-analysis of case-control studies. *Journal of Dermatology* 2012; 39: 770-3.
28. Berg K. M., Kunins H. V., Jackson J. L., Nahvi S., Chaudhry A., Harris Jr K. A. *et al.* Association between alcohol consumption and both osteoporotic fracture and bone density. *Journal of Medicine* 2008; 121: 406-18.
29. Lönnroth K., Williams B., Stadlin S., Jaramillo E., Dye C. Alcohol use as a risk factor for tuberculosis—a systematic review. *BMC Public Health* 2008; 8: 289.
30. Corrao G., Bagnardi V., Zambon A., La Vecchia C. A meta-analysis of alcohol consumption and the risk of 15 diseases. *Preventive Medicine* 2004; 38: 613-9.
31. Islami F., Fedirko V., Tramacere I., Bagnardi V., *al. e.* Alcohol drinking and esophageal squamous cell carcinoma with focus on light-drinkers and never-smokers: a systematic review and meta-analysis. *International Journal of Cancer* 2011; 129: 2473-84.

32. Islami F., Tramacere I., Rota M., Bagnardi V., Fedirko V., Scotti L. *et al.* Alcohol drinking and laryngeal cancer: overall and dose-risk relation--a systematic review and meta-analysis. *Oral Oncology* 2010; 46: 802-10.
33. Key J., Hodgson S., Omar R. Z., Jensen T. K., Thomson S. G., Boobis A. R. *et al.* Meta-analysis of studies of alcohol and breast cancer with consideration of the methodological issues. *Cancer Causes and Control* 2006; 17: 759-70.
34. Collaborative Group on Hormonal Factors in Breast Cancer. Alcohol, tobacco and breast cancer--collaborative reanalysis of individual data from 53 epidemiological studies, including 58,515 women with breast cancer and 95,067 women without the disease. *British Journal of Cancer* 2002; 87: 1234-45.
35. Baliunas D. O., Taylor B. J., Irving H., Roerecke M., Patra J., Mohapatra S. *et al.* Alcohol as a risk factor for type 2 diabetes: a systematic review and meta-analysis. *Diabetes Care* 2009; 32: 2123-32.
36. Samokhvalov A. V., Irving H., Mohapatra S., Rehm J. Alcohol consumption, unprovoked seizures, and epilepsy: a systematic review and meta-analysis. *Epilepsia* 2010; 51: 1177-84.
37. Samokhvalov A. V., Irving H. M., Rehm J. Alcohol consumption as a risk factor for atrial fibrillation: a systematic review and meta-analysis. *European Journal of Cardiovascular Prevention & Rehabilitation* 2010; 17: 706-12.
38. Kodama S., Saito K., Tanaka S., Horikawa C., Saito A., Heianza Y. *et al.* Alcohol consumption and risk of atrial fibrillation: a meta-analysis. *Journal Of The American College Of Cardiology* 2011; 57: 427-36.
39. Roerecke M., Rehm J. The cardioprotective association of average alcohol consumption and ischaemic heart disease: a systematic review and meta-analysis. *Addiction* 2012; doi:10.1111/j.1360-0443.2012.03780.x.
40. Patra J., Taylor B., Irving H., Roerecke M., Baliunas D., Mohapatra S. *et al.* Alcohol consumption and the risk of morbidity and mortality for different stroke types - a systematic review and meta-analysis. *BMC Public Health* 2010; 10: 258.
41. Samokhvalov A. V., Irving H. M., Rehm J. Alcohol consumption as a risk factor for pneumonia: a systematic review and meta-analysis. *Epidemiology and Infection* 2010; 138: 1789-95.
42. Rehm J., Taylor B., Mohapatra S., Irving H., Baliunas D., Patra J. *et al.* Alcohol as a risk factor for liver cirrhosis: A systematic review and meta-analysis. *Drug and Alcohol Review* 2010; 29: 437-45.
43. Irving H. M., Samokhvalov A. V., Rehm J. Alcohol as a risk factor for pancreatitis. A systematic review and meta-analysis. *JOP* 2012; 10: 387-92.
44. Taylor B., Irving H. M., Kanteres F., Room R., Borges G., Cherpitel C. J. *et al.* The more you drink, the harder you fall: A systematic review and meta-analysis of how acute alcohol consumption and injury or collision risk increase together. *Drug and Alcohol Dependence* 2010; 110: 108-16.
45. Zeisser C., Stockwell T. R., Chikritzhs T., Cherpitel C., Ye Y., Gardner C. A systematic review and meta-analysis of alcohol consumption and injury risk as a function of study design and recall period. *Alcoholism: Clinical and Experimental Research* 2013; 37: E1-E8.

46. Di Castelnuovo A., Costanzo S., Bagnardi V., Donati M. B., Iacoviello L., de Gaetano G. Alcohol dosing and total mortality in men and women. *Archives of Internal Medicine* 2006; 166: 2437-45.
47. Rehm J., Gmel G., Sempos C. T., Trevisan M. Alcohol-related mortality and morbidity. *Alcohol Research & Health* 2003; 27: 39-51.
48. Rehm J., Patra J. Different guidelines for different countries? On the scientific basis of low-risk drinking guidelines and their implications. *Drug and Alcohol Review* 2012; 31: 156-61.
49. O'Flaherty M., Buchan I., Capewell S. Contributions of treatment and lifestyle to declining CVD mortality: why have CVD mortality rates declined so much since the 1960s? *Heart* 2013; 99: 159-62.
50. Gmel G., Gutjahr E., Rehm J. How stable is the risk curve between alcohol and all-cause mortality and what factors influence the shape? A precision-weighted hierarchical meta-analysis. *European Journal of Epidemiology* 2003; 18: 631-42.
51. Kamper-Jorgensen M., Gronbaek M., Tolstrup J., Becker U. Alcohol and cirrhosis: dose-response or threshold effect? *Journal of Hepatology* 2004; 41: 25-30.
52. Parrish K. M., Dufour M. C. Drinking patterns and liver cirrhosis mortality. *Alcohol and Alcoholism* 1991; Suppl 1: 331-4.
53. Sorensen T. I., Orholm M., Bentsen K. D., Hoybye G., Eghoje K., Christoffersen P. Prospective evaluation of alcohol abuse and alcoholic liver injury in men as predictors of development of cirrhosis *Lancet* 1984; 324: 241-4.
54. Dawson D. A., Li T. K., Grant B. F. A prospective study of risk drinking: at risk for what? *Drug and Alcohol Dependence* 2008; 95: 62-72.
55. Marugame T., Yamamoto S., Yoshimi I., Sobue T., Inoue M., Tsugane S. Patterns of alcohol drinking and all-cause mortality: results from a large-scale population-based cohort study in Japan. *American Journal of Epidemiology* 2007; 1: 1039-46.
56. Gmel G., Kuntsche E., Rehm J. Risky single occasion drinking: Bingeing is not bingeing. *Addiction* 2011; 106: 1037-45.
57. Roerecke M., Rehm J. Irregular heavy drinking occasions and risk of ischemic heart disease: a systematic review and meta-analysis. *American Journal of Epidemiology* 2010; 171: 633-44.
58. Chen W. J., Maier S. E., Parnell S. E., West J. R. Alcohol and the developing brain: neuroanatomical studies. *Alcohol Research and Health* 2003; 27: 174-80.
59. Sulik K. K., Johnston M. C., Webb M. A. Fetal alcohol syndrome: embryogenesis in a mouse model. *Science* 1981; 214: 936-8.
60. Gmel G., Bissery A., Gammeter R., Givel J.-C., Calmes J.-M., Yersin B. *et al.* Alcohol-attributable injuries in admissions to a Swiss emergency room—an analysis of the link between volume of drinking, drinking patterns and preattendance drinking. *Alcoholism: Clinical and Experimental Research* 2006; 30: 501-9.
61. Watt K. D. M., Purdie D. M., Roche A. M., McClure R. Risk of injury from acute alcohol consumption and the influence of confounders. *Addiction* 2004; 99: 1262-73.

62. Stockwell T. R., McLeod R., Stevens M., Phillips M., Webb M., Jelinek G. Alcohol consumption, setting, gender and activity as predictors of injury: a population-based case-control study. *Journal of Studies on Alcohol* 2002; 63: 372–9.
63. Kan H. P., Huang Y. Q., Tan Y. F., Zhou J. Meta-analysis of alcohol consumption and risk of extrahepatic bile system cancer. *Hepatology Research* 2011; 41: 746-53.
64. Corrao G., Rubbiati L., Bagnardi V., Zambon A., Poikolainen K. Alcohol and coronary heart disease: a meta-analysis. *Addiction* 2000; 95: 1505-23.
65. Ronksley P. E., Brien S. E., Turner B. J., Mukamal K. J., Ghali W. A. Association of alcohol consumption with selected cardiovascular disease outcomes: a systematic review and meta-analysis. *BMJ* 2011; 342: d671.
66. Fekjær H. O. Second-class evidence for causality, not second-class science. *Addiction* 2013; DOI: 10.1111/add.12386.
67. Fillmore K. M., Kerr W. C., Stockwell T., Chikritzhs T., Bostrom A. Moderate alcohol use and reduced mortality risk: systematic error in prospective studies. *Addiction Research and Theory* 2006; 14: 101-32.
68. Shaper G., Wannamethee G., Walker M. Alcohol and mortality in British men: explaining the U-shaped curve. *Lancet* 1988; 2: 1267.
69. Stockwell T., Greer A., Fillmore K., Zeisser C. How good is the science? *BMJ* 2012; 344: doi: <http://dx.doi.org/10.1136/bmj.e2276>.
70. Klatsky A. L., Udaltsova N. Alcohol drinking and total mortality risk. *Annals of Epidemiology* 2007; 17: S63-S7.
71. Chikritzhs T., Fillmore K., Stockwell T. A healthy dose of skepticism: Four good reasons to think again about protective effects of alcohol on coronary heart disease. *Drug and Alcohol Review* 2009; 28: 441-4.
72. Fekjær H. O. Alcohol—a universal preventive agent? A critical analysis. *Addiction* 2013; 108: 2051-7.
73. Babor T. F., Caetano R., Casswell S., Edwards G., Giesbrecht N., Graham K. *et al.* Alcohol: no ordinary commodity. Research and public policy. New York: Oxford University Press; 2010.
74. Dodgson J., Bramley-Harker E., Spackman M., Aslam S., Barham L. *Alcohol in London: a cost-benefit analysis - a final report for the Greater London Authority*. London: NERA Economic Consulting; 2003.
75. Gruenewald P. J., Johnson F. W., Ponicki W. R., LaScala E. A. A dose-response perspective on college drinking and related problems. *Addiction* 2010; 105: 257-69.
76. Thompson K. D., Stockwell T., MacDonald S. Is there a 'low-risk' drinking level for youth? The risk of acute harm as a function of quantity and frequency of drinking. *Drug and Alcohol Review* 2012; 31: 184-93.
77. Jones L., Bates G., McCoy E., Tiffany C., Perkins C., Bellis M. A. *The economic and social costs of alcohol-related harm in Leeds 2008-09*. Liverpool: Centre for Public Health, LJMU; 2010.

78. House of Commons Health Committee. *Government's Alcohol Strategy. Third Report of Session 2012–13. Volume I: Report, together with formal minutes and oral and written evidence*. London: The Stationery Office Limited; 2012.
79. Giesbrecht N., Cukier S., Steeves D. Collateral damage from alcohol: implications of 'second-hand effects of drinking' for populations and health priorities. *Addiction* 2010; 105: 1323-5.
80. Cook P. A., Tocque K., Morleo M., Bellis M. A. *Opinions on the impact of alcohol on individuals and communities: early summary findings from the NorthWest Big Drink Debate*. Liverpool: Centre for Public Health, Liverpool John Moores University; 2008.
81. Hope A., Curran J., Bell G., Platts A. *Unrecognised and under-reported: the impact of alcohol on people other than the drinker in Scotland*. Glasgow: Alcohol Focus Scotland; 2013.
82. Forsyth A., Davidson N. The nature and extent of illegal drug and alcohol-related litter in Scottish social housing community: A photographic investigation. *Addiction Theory and Research* 2010; 18: 71-83.
83. Velleman R., Templeton L. Understanding and modifying the impact of parents' substance misuse on children. *Advances in Psychiatric Treatment* 2007; 13: 79-89.
84. World Health Organization. *Intimate partner violence and alcohol*. Geneva: World Health Organization; 2006.
85. Foran H. M., O'Leary K. D. Alcohol and intimate partner violence: a meta-analytic review. *Clinical Psychology Review* 2008; 28: 1222-34.
86. Cleaver H., Unell I., Aldgate J. *Children's needs - parenting capacity. Child abuse: Parental mental illness, learning disability, substance misuse and domestic violence*. Norwich: The Stationery Office; 2011.
87. Laslett A.-M., L., Dietze P. M., Room R. G. W. Carer drinking and more serious child protection case outcomes. *British Journal of Social Work* 2012; doi: 10.1093/bjsw/bcs052.
88. Laslett A.-M., Catalano P., Chikritzhs T., Dale C., Doran C., Ferris J. *et al. The range and magnitude of alcohol's harm to others*. Fitzroy, Victoria: AER Centre for Alcohol Policy Research, Turning Point Alcohol and Drug Centre, Eastern Health; 2010.
89. Donaldson L. *CMO Annual Report 2008: 150 Years of the Annual Report of the Chief Medical Officer*. London: Department of Health; 2008.
90. Kershaw C., Nicholas S., Walker A. *Crime in England and Wales 2007/08: findings from the British Crime Survey and police recorded crime*. London: Home Office; 2008.
91. Povey D., Coleman K., Kaiza P., Hoare J., Jansson K. *Homicides, firearms offences and intimate violence 2006/07*. London: Home Office; 2008.
92. Department for Children Schools and Families. *Referrals, assessments and children and young people who are the subject of a Child Protection Plan, England - year ending 31 Mar 2008*; 2008.
93. Forrester D., Harwin J. Parental substance misuse and child care social work: findings from the first stage of a study of 100 families. *Child and Family Social Work* 2006; 11: 325-35.
94. Department for Transport. *Road Casualties Great Britain: 2007*. London: Department for Transport; 2008.

95. Morleo M., Woolfall K., Dedman D., Mukherjee R. A. S., Bellis M. A., Cook P. A. Under-reporting of Fetal Alcohol Spectrum Disorders: an analysis of Hospital Episode Statistics. *BMC Paediatrics* 2011; 11: 1-6.
96. National Collaborating Centre for Women's and Children's Health. *Antenatal care: routine care for the healthy pregnant woman*. London: Royal College of Obstetricians and Gynaecologists; 2008.
97. Gray R., Henderson J. *Review of the fetal effects of prenatal alcohol exposure*. Oxford: National Perinatal Epidemiology Unit, University of Oxford; 2006.
98. Henderson J., Gray R., Brocklehurst P. Systematic review of effects of low–moderate prenatal alcohol exposure on pregnancy outcome. *BJOG* 2007; 114: 243-52.
99. Henderson J., Kesmodel U., Gray R. Systematic review of the fetal effects of prenatal binge-drinking. *Journal of Epidemiology and Community Health* 2007; 61: 1069-73.
100. Patra J., Bakker R., Irving H., Jaddoe V. W. V., Malini S., Rehm J. Dose–response relationship between alcohol consumption before and during pregnancy and the risks of low birthweight, preterm birth and small for gestational age (SGA)—a systematic review and meta-analyses. *BJOG* 2011; 118: 1411–21.
101. Donaldson L. *Guidance on the consumption of alcohol by children and young people*. London: Department of Health; 2009.
102. Dawson D. A., Goldstein R. B., Chou S. P., Ruan W. J., Grant B. F. Age at First Drink and the First Incidence of Adult-Onset DSM-IV Alcohol Use Disorders. *Alcoholism: Clinical & Experimental Research* 2008; 32: 1-12.
103. Grant B. F., Stinson F. S., Harford T. C. Age at onset of alcohol use and DSM-IV alcohol abuse and dependence: a 12-year follow-up. *JOURNAL OF SUBSTANCE ABUSE* 2001; 13: 493-504.
104. Hill K. G., White H. R., Chung I. J., Hawkins J. D., Catalano R. F. Early adult outcomes of adolescent binge drinking. Person- and variable-centered analyses of binge drinking trajectories. *Alcoholism: Clinical & Experimental Research* 2000; 24: 892-901.
105. Viner R. M., Taylor B. Adult outcomes of binge drinking in adolescence: findings from a UK national birth cohort. *Journal of Epidemiology & Community Health* 2007; 61: 902-7.
106. Renna F. The economic cost of teen drinking: late graduation and lowered earnings. *Health Economics* 2007; 16: 407-19.
107. McCambridge J., McAlaney J., Rowe R. Adult consequences of late adolescent alcohol consumption: a systematic review of cohort studies. *PLoS Medicine / Public Library of Science* 2011; 8.
108. Maimaris W., McCambridge J. Age of first drinking and adult alcohol problems: systematic review of prospective cohort studies. *Journal of Epidemiology and Community Health* 2013; doi:10.1136/jech-2013-203402.
109. Rossow I., Kuntsche E. Early onset of drinking and risk of heavy drinking in young adulthood – a 13 year prospective study. *Alcoholism: Clinical and Experimental Research* 2013; 37: E297-E304.

110. Toumbourou J. W., Stockwell T., Neighbors C., Marlatt G. A., Sturge J., Rehm J. Interventions to reduce harm associated with adolescent substance use: an international review. *Lancet* 2007; 369: 1391-401.
111. Ng Fat L., Fuller E. Chapter 6. Drinking patterns. In: Craig R, Mindell J, editors. *Health Survey for England 2011*. Leeds: Health and Social Care Information Centre; 2012.
112. Sharp C. Alcohol consumption. In: Rutherford L, Sharp C, Bromley C, editors. *The Scottish Health Survey Volume 1: Adults*. Edinburgh: The Scottish Government; 2012.
113. Welsh Government. *Welsh Health Survey 2011*. Cardiff: Welsh Government; 2012.
114. Northern Ireland Statistics and Research Agency. *Adult Drinking Patterns in Northern Ireland 2011*. Belfast: Department of Health, Social Services and Public Safety; 2012.
115. McAndrew F., Thompson J., Fellows L., Large A., Speed M., Renfrew M. J. *Infant Feeding Survey 2010*. Leeds: The Health and Social Care Information Centre; 2012.
116. Currie C., Zanotti C., Morgan A., Currie D., de Looze M., Roberts C. *et al. Social determinants of health and well-being among young people. Health Behaviour in School-aged Children (HBSC) study: international report from the 2009/2010 survey*. Copenhagen: WHO Regional Office for Europe; 2012.
117. Brooks F., Magnusson J., Klemra E., Spencer N., Morgan A. *HBSC England National Report. Health Behaviour in School-aged Children (HBSC): World Health Organization Collaborative Cross National Study*. Hatfield: University of Herfordshire; 2011.

Appendix 1. Summary of Australian and Canadian alcohol guidelines

Australian guidelines to reduce health risks from drinking alcohol

Guideline 1

Reducing the risk of alcohol-related harm over a lifetime

The lifetime risk of harm from drinking alcohol increases with the amount consumed.

For healthy men and women, drinking no more than two standard drinks* on any day reduces the lifetime risk of harm from alcohol-related disease or injury.

Guideline 2

Reducing the risk of injury on a single occasion of drinking¹

On a single occasion of drinking, the risk of alcohol-related injury increases with the amount consumed.

For healthy men and women, drinking no more than four standard drinks* on a single occasion reduces the risk of alcohol-related injury arising from that occasion.

Guideline 3

Children and young people under 18 years of age

For children and young people under 18 years of age, not drinking alcohol is the safest option.

- A Parents and carers should be advised that children under 15 years of age are at the greatest risk of harm from drinking and that for this age group, not drinking alcohol is especially important.
- B For young people aged 15–17 years, the safest option is to delay the initiation of drinking for as long as possible.

Guideline 4

Pregnancy and breastfeeding

Maternal alcohol consumption can harm the developing fetus or breastfeeding baby.

- A For women who are pregnant or planning a pregnancy, not drinking is the safest option.
- B For women who are breastfeeding, not drinking is the safest option.

* The Australian standard drink contains 10g of alcohol (equivalent to 12.5 mL of pure alcohol)

Canada's Low-Risk Alcohol Drinking Guidelines

Guideline 1

Do not drink in these situations:

When operating any kind of vehicle, tools or machinery; using medications or other drugs that interact with alcohol; engaging in sports or other potentially dangerous physical activities; working; making important decisions; if pregnant or planning to be pregnant; before breastfeeding; while responsible for the care or supervision of others; if suffering from serious physical illness, mental illness or alcohol dependence.

Guideline 2

If you drink, reduce long-term health risks by staying within these average levels:

Women	Men
0–2 standard drinks* per day	0–3 standard drinks* per day
No more than 10 standard drinks per week	No more than 15 standard drinks per week

Always have some non-drinking days per week to minimize tolerance and habit formation. Do not increase drinking to the upper limits as health benefits are greatest at up to one drink per day. Do not exceed the daily limits specified in Guideline 3

Guideline 3

If you drink, reduce short-term risks by choosing safe situations and restricting your alcohol intake:

Risk of injury increases with each additional drink in many situations. For both health and safety reasons, it is important not to drink more than:

- Three standard drinks* in one day for a woman
- Four standard drinks* in one day for a man

Drinking at these upper levels should only happen occasionally and always be consistent with the weekly limits specified in Guideline 2. It is especially important on these occasions to drink with meals and not on an empty stomach; to have no more than two standard drinks in any three-hour period; to alternate with caffeine-free, non-alcoholic drinks; and to avoid risky situations and activities. Individuals with reduced tolerance, whether due to low bodyweight, being under the age of 25 or over 65 years old, are advised to never exceed Guideline 2 upper levels.

Guideline 4

When pregnant or planning to be pregnant:

The safest option during pregnancy or when planning to become pregnant is to not drink alcohol at all. Alcohol in the mother's bloodstream can harm the developing fetus. While the risk from light consumption during pregnancy appears very low, there is no threshold of alcohol use in pregnancy that has been definitively proven to be safe.

Guideline 5

Alcohol and young people:

Alcohol can harm healthy physical and mental development of children and adolescents. Uptake of drinking by youth should be delayed at least until the late teens and be consistent with local legal drinking age laws. Once a decision to start drinking is made, drinking should occur in a safe environment, under parental guidance and at low levels (i.e., one or two standard drinks* once or twice per week). From legal drinking age to 24 years, it is recommended women never exceed two drinks per day and men never exceed three drinks in one day.

* A "standard drink" is equal to a 341 ml (12 oz.) bottle of 5% strength beer, cider or cooler; a 142 ml (5 oz.) glass of 12% strength wine; or a 43 ml (1.5 oz.) shot of 40% strength spirits (NB: 1 Canadian standard drink = 17.05 ml or 13.45 g of ethanol)

Appendix 2. How much do people in the UK drink?

Prevalence of drinking

As shown in Table 6 the prevalence of drinking among adults is high across all four countries in the UK. With the exception of Northern Ireland, which recorded the highest prevalence of non-drinkers, a similar prevalence of non-drinkers was recorded across England, Scotland and Wales in 2011.

Table 6. Summary of prevalence of drinking in the UK, by age and country

	England ^a	Scotland ^b	Wales ^c	Northern Ireland ^d
Men				
Drinkers	87%	89%	90%	78%
Non-drinkers	13%	11%	10%	22%
Lifetime abstainer	5% ^a	5%	-	-
Former drinker	6% ^a	6%	-	-
Women				
Drinkers	81%	83%	84%	72%
Non-drinkers	19%	17%	16%	28%
Lifetime abstainer	10% ^e	9%	-	-
Former drinker	9% ^e	9%	-	-

^a Health Survey for England 2011 ¹¹¹. ^b The Scottish Health Survey 2011 ¹¹². ^c Welsh Health Survey 2011 ¹¹³. ^d Adult Drinking Patterns in Northern Ireland 2011 ¹¹⁴. ^e Calculated based on data from the General Lifestyle Survey 2010.

Among current drinkers in 2011, drinking alcohol on five or more days in the previous week was reported by 18% of men and 10% of women in England and 9% of men and 8% of women in Wales. In Scotland, 13% of men and 10% of women reported drinking on more than five days a week, and in Northern Ireland, 8% of men and 5% of women drank daily or on most days. In England and Scotland, men and women aged 16-24 years drank on a lower mean number of days than men and women aged 75 years or older. For example, in England, men aged 16-24 years drank on a mean 2.1 days, compared with a mean 4.2 days among men aged 75 years or older.

Maximum daily consumption

Current government guidance recommends that men should not regularly drink more than 3–4 units of alcohol a day and women should not regularly drink more than 2–3 units a day. Table 7 summarises the proportion of adults who drank above the recommended levels on at least one day in the last week in 2011.

Table 7. Proportion of adults drinking above recommended limits on at least one day in the last week, by sex and country

	England ^a	Scotland ^b	Wales ^c	Northern Ireland ^d
Men				
More than 4 units	39%	41%	50%	76% ^e
More than 8 units	22%	25%	33%	35% ^f
Women				
More than 3 units	27%	34%	38%	81% ^g
More than 6 units	13%	17%	22%	25% ^h

^a Health Survey for England 2011 ¹¹¹. ^b The Scottish Health Survey 2011 ¹¹². ^c Welsh Health Survey 2011 ¹¹³. ^d Adult Drinking Patterns in Northern Ireland 2011 ¹¹⁴. ^e Drank more than or equal to 4 units. ^f Drank more than or equal to 10 units in one session. ^g Drank more than or equal to 6 units. ^h Drank more than or equal to 7 units in one session.

In England and Scotland, the proportion of men and women drinking more than twice the recommended levels was highest among 16-24 year olds. For example in Scotland, 32% of women aged 16-24 years old drank more than twice the recommended levels on at least one day in the last week compared to 12% of women aged 65-74 years or older.

Weekly consumption

The recommended levels for weekly drinking are currently no more than 21 units a week for men and no more than 14 units for women. As shown in Table 8, the proportions of men and women drinking more than the weekly recommend levels were broadly similar across the countries of the UK.

Table 8. Summary of weekly alcohol consumption in the UK, by sex and country

	England ^a	Scotland ^b	Wales ^c	Northern Ireland ^d
Men				
More than 21 units	23%	25%	-	26%
More than 50 units ^e	6%	-	-	7%
Women				
More than 14 units	18%	18%	-	20%
More than 35 units ^e	4%	-	-	3%

^a Health Survey for England 2011 ¹¹¹. ^b The Scottish Health Survey 2011 ¹¹². ^c Welsh Health Survey 2011 ¹¹³. ^d Adult Drinking Patterns in Northern Ireland 2011 ¹¹⁴. ^e NHS threshold for 'higher risk' drinking.

Men aged 55–64 years in England and men aged 45–54 years in Scotland had the highest estimates for weekly consumption. In both England and Scotland, women aged 45–54 years had the highest estimates.

Drinking during pregnancy

The 2010 Infant Feeding Survey ¹¹⁵ found that 81% of mothers in the UK had drunk alcohol in the two years before their pregnancy and that 40% had drunk during their pregnancy (Table 9). Among mothers who drank alcohol before pregnancy, 49% stopped drinking

during their pregnancy and 46% reported that they drank less alcohol. There was a clear association between drinking during pregnancy and mother's age; 28% of mothers aged under 20 drank during pregnancy compared with 52% of mothers aged 35 or over.

Table 9. Drinking before and during pregnancy, by country

	England	Wales	Scotland	Northern Ireland
Drank before pregnancy	80%	87%	87%	86%
Drank during pregnancy	41%	39%	35%	35%
Gave up drinking	48%	55%	59%	58%
Drank less	47%	42%	37%	38%
No change / drank more	2%	1%	1%	1%

Infant Feeding Survey 2010 ¹¹⁵

Drinking among young people

The Health Behaviours in School-aged Children (HBSC) survey collects data every four years on 11-, 13- and 15-year-old boys' and girls' health behaviours including alcohol use ¹¹⁶. The survey includes England, Scotland and Wales. In 2009/10, the prevalence of weekly drinking among young people was similar across boys and girls in England, Scotland and Wales (Table 10). In all three countries, the proportion of young people who were weekly drinkers exceeded the HBSC average.

Table 10. Proportion of young people who drink alcohol at least once a week, by sex and country

	England	Scotland	Wales	HBSC average ^a
Boys				
age 11 years	5%	4%	5%	5%
age 13 years	10%	10%	14%	10%
age 15 years	31%	29%	35%	25%
Girls				
age 11 years	1%	2%	2%	2%
age 13 years	10%	9%	14%	6%
age 15 years	22%	25%	29%	17%

^a Based on equal weighting of each region, regardless of differences in achieved sample size or country population. Figures highlighted in bold indicate a significant gender difference in prevalence.

HBSC survey 2009/2010 ¹¹⁶

The proportion of young people who had been drunk at least twice in England, Scotland and Wales also exceeded the HBSC average (Table 11). At age 15 years, in all three countries, the proportion of girls who had been drunk at least twice was higher than the proportion of boys.

Table 11. Proportion of young people who have been drunk at least twice, by sex and country

	England	Scotland	Wales	HBSC average ^a
Boys				
age 11 years	3%	3%	4%	3%
age 13 years	15%	14%	17%	11%
age 15 years	38%	40%	47%	34%
Girls				
age 11 years	1%	1%	2%	1%
age 13 years	15%	16%	18%	8%
age 15 years	43%	46%	50%	29%

^a Based on equal weighting of each region, regardless of differences in achieved sample size or country population. Figures highlighted in bold indicate a significant gender difference in prevalence.

HBSC survey 2009/2010 ¹¹⁶

Analysis of the England data ¹¹⁷ showed that at age 15, reported age of first alcoholic drink was significantly correlated with lifetime incidence of drunkenness among both boys and girls. The younger the age at which a young person first drank alcohol, the more times they reported having ever been drunk.

Appendix 3. Considerations regarding the evidence

All of the currently available research evidence on the relationship between alcohol and health is based on the findings of observational studies. Such studies are methodologically complex and study findings may potentially be influenced by various potential confounders, biases and other methodological issues. As noted below, issues with the quality of the research evidence may result in both over- and under-estimation of the risks associated with particular levels of alcohol consumption. The following points are important in considerations of the evidence on the health impacts of alcohol consumption.

- Published evidence is typically drawn from studies conducted outside the UK, and for some conditions (including epilepsy, atrial fibrillation, hypertensive diseases, liver cirrhosis and pancreatitis) none of the contributory data are from the UK. It is therefore not known how directly applicable some of this evidence is to patterns of alcohol consumption in the UK.
- The selection of control groups is important in studies of alcohol and health. The majority of meta-analyses have used non-drinkers as the reference group, and as such do not distinguish between lifetime abstainers and former drinkers who may have stopped drinking due to ill health. The inclusion of former drinkers potentially places the non-drinking reference group at a falsely higher level of risk than alcohol drinkers (known as the 'sick-quitter hypothesis'). Some, but not all, studies have used lifetime abstainers as the reference group to overcome this bias (see Section 3).
- The relationship between alcohol consumption and risk has generally been examined categorically (e.g. using pre-designated categories of alcohol consumption) however more recently, where sufficiently reliable studies are available, meta-analyses have sought to examine the dose-response relationship. Such studies are useful for determining whether there is any evidence for a threshold below which alcohol consumption may not have health consequences.
- Few published meta-analyses include a formal assessment of study quality but variability in the quality of the primary studies available has been noted by many review authors. For example, with regard to potential confounders, studies included in all of the reviews differed widely in their level of adjustment for potential confounding factors, with some studies not adjusting for any factors (including basic factors such as age or smoking).
- The vast majority of studies of alcohol consumption rely on self-reported alcohol use and underreporting of alcohol consumption poses problems for the interpretation of evidence from epidemiological studies of alcohol-related risks. The nature of underreporting is such that the risks associated with specific drinking levels in epidemiological studies may be overestimated.

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January 2014