
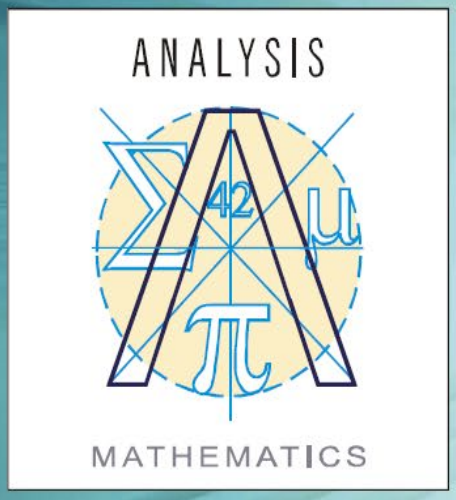


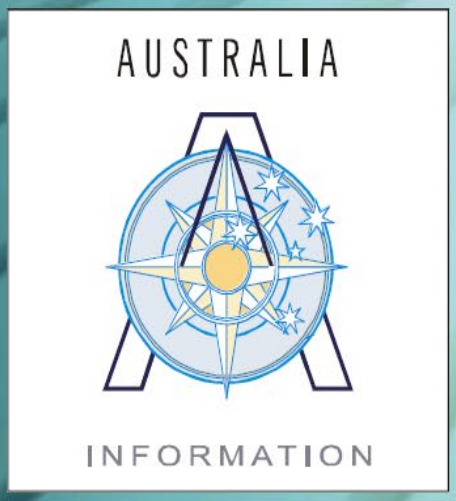


# Review of Draft Alcohol Guidelines

March 2020

*Project:* ABA/1

STRATEGIC INFORMATION CONSULTANTS

# Review of Draft Alcohol Guidelines

March 2020

Client: Alcohol Beverages Australia

*Project*: ABA/1

*[NHMRC has removed personal information]*

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# Executive Summary

Data Analysis Australia was commissioned by Alcohol Beverages Australia to conduct on independent review of the statistical aspects of the *Draft Australian Guidelines to Reduce Health Risks from Drinking Alcohol* released by the NHMRC in December 2019 (the Report). Since the first proposed guideline is the only one that is quantitative in nature, the review focused on that guideline. This guideline reads:

> ***To reduce the risk of harm from alcohol-related disease or injury for healthy men and women, drink no more than 10 standard drinks per week and no more than 4 standard drinks on any one day.***
>
> ***The less you choose to drink, the lower your risk of alcohol-related harm. For some people not drinking at all is the safest option.***

As is outlined below, the review is heavily constrained by time and lack of access to relevant information but it has been possible to draw several significant conclusions:

**Finding 1. The methodology behind the guideline relies heavily upon modelling conducted (apparently under contract) by the Sheffield Alcohol Research Group (SARG) using an adaptation of the Sheffield Alcohol Policy Model (SAPM). This model has not been made available for independent review and hence does not represent open and transparent science.**

Published information on this model mainly refers to earlier versions. Indeed, it appears that the SAPM was not made available to the NHMRC Alcohol Working Committee who authored the Guidelines. Our investigation of the information published on the model and the responses by SARG to our questions strongly suggests that the model contains numerous assumptions that are not fully documented.

For example, the model appears to assume certain relationships between the likelihood of binge drinking and average alcohol consumption, whereas the data claimed to be used in this part of the model, the National Drug Strategy Household survey 2016 does not contain data that could establish such a relationship. It would appear that information or models from other jurisdictions has been used. Whether this is reasonable or not is impossible to determine without proper access to the model or the detailed model outputs.

**Finding 2. The SAPM was primarily developed to assess the possible effects of certain alcohol control measures, particularly those relating to prices. These aspects of the model are not necessarily relevant to the context of the guideline and it is not clear whether they impact upon the utility of the model for the current context.**

It is notable that the model was not used to evaluate the risks associated with the existing guideline. Guidance to us from SARG is that the scenario of averaging 14

drinks per week and never having more than four drinks in any one day is "approximately" one of the scenarios presented but it is not clear how approximate this is.

Some of these issues appear to arise from the SAPM being developed to model a population, while a guideline is meant to communicate to an individual.

**Finding 3. There appears to be a substantial disconnect between the outputs of the Sheffield model and the guideline. In particular the draft guidelines (a) lose the difference between males and females, and (b) for people who avoid binge drinking, they understate the average number of drinks that might be considered low risk.**

The most obvious problem of the Report is that they do not clearly separate out the impacts of average numbers of drinks and binge drinking, particularly as the latter is much more likely to lead to acute health conditions. Instead the guideline represents a simplified summary of one set of behaviours that might be considered to have an acceptable risk, while not presenting different behaviours that might have similar or lower levels of risk. Hence, the guideline does *not* encourage informed decision making despite the statement that "understanding the risks helps Australians make informed choices about their health."

The reason given for having the same guideline for men and women – that men tend to drink more anyway – confuses the science with a marketing message. While we recognise that public health issues almost always need to consider both, this should not mean incorrectly portraying the science.

**Finding 4. The model scenario SA1 that assumes that low levels of alcohol consumption has no protective effect does not exist has a major effect on the model that substantially increases the aetiological fraction mortality attributable to alcohol. This effect is not explicitly presented and no evidence is presented that the aetiological fraction thus calculated is reasonable.**

Modifying the risk functions in the complex model to remove thresholds or to remove the protective effects effectively increases the risks of adverse health incomes. The complex calibration in the model has complex effects. It is not apparent what the implications of this actually are.

**Finding 5. The Report has an inconsistent attitude towards the protective effects of low levels of alcohol consumption. The guideline is fundamentally based upon the assumption that there is a protective effect – if there was not then the methodology would lead to a recommended level that is a small fraction of the guideline. However at the same time it is stated that "all alcohol consumption comes with some degree of risk" and the guideline includes the statement "the**

**less you choose to drink, the lower your risk of alcohol-related harm."**

The possible protective effect of low levels of alcohol consumption has been recognised for many years. At the same time, the causal relationships are not well understood as most of the data is observational. Our assessment is that there are reasoned arguments both for and against such a causal relationship and that this question is unlikely to be answered definitively for some time. However many of these arguments apply to all aspects of estimating the effects of alcohol and it would appear inconsistent to apply such cautions to only this aspect.

While some claims are made that methods such as Mendelian randomisation can answer this, our assessment is that they are a long way from providing definitive estimates for what might be complex relationships.

**Summary**

The Guidelines do not, in our opinion, provide statistically valid evidence for a significant change in the guidelines for alcohol consumption (outside the areas of children and pregnant/nursing mothers). For this reason it is difficult to understand why a change is being suggested. In fact, the form of the first guideline is a very crude characterisation of the outputs of the model used to justify it. In that sense, it does not provide genuine information that assists drinkers in making sensible choices.

Having said that, the overall approach of modelling the likely harms that may result from given drinking behaviour has much to recommend it. It is unfortunate that this has been done in a far from transparent manner and that the results of the modelling are used in a confusing manner.

**Perhaps more importantly, this review must be regarded as an interim review. Without proper access to the most important modelling work in the Report, it is impossible to have confidence in the accuracy or relevance of the central parts of the Report. We strongly recommend that either the model be made freely available or it not be used until a satisfactory independent review is conducted.**

# Table of Contents

# 1. Introduction

Data Analysis Australia was commissioned by Alcohol Beverages Australia to independently review the *Draft Australian Guidelines to Reduce Health Risks from Drinking Alcohol* released by the NHMRC in December 2019.[1] The review was undertaken by [*NHMRC has removed personal information]* of Data Analysis Australia with assistance from *[NHMRC has removed personal information]* also of Data Analysis Australia.

In conducting the review Data Analysis Australia followed as far as was possible the guidelines used by an Expert Witness in a legal matter, with the primary responsibility being to give an unbiased assessment rather than to try to support one side or the other.[2]

## 1.1. The Guidelines

The Report *Draft Australian Guidelines to Reduce Health Risks from Drinking Alcohol* (which we will refer to as "the Report") in conjunction with a number of subsidiary commissioned reports presents three proposed new guidelines and presents evidence for their acceptance.

The guidelines are as follows:

**Guideline One:**

> To reduce the risk of harm from alcohol-related disease or injury for healthy men and women, drink no more than 10 standard drinks per week and no more than 4 standard drinks on any one day.
>
> The less you choose to drink, the lower your risk of alcohol-related harm. For some people not drinking at all is the safest option.

**Guideline Two:**

> To reduce the risk of injury and other harms to health, children and young people under 18 years of age should not drink alcohol.

**Guideline Three:**

> To reduce the risk of harm to their unborn child, women who are pregnant or planning a pregnancy should not drink alcohol.
>
> For women who are breastfeeding, not drinking is safest for their baby.

The latter two guidelines are essentially non-quantitative and it is clear that the precautionary principle is being applied in a way that few would argue with. We have therefore focused the review on the first Guideline.

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[1] See https://www.nhmrc.gov.au/health-advice/alcohol#download

[2] See for example the Expert Witness of the Federal Court of Australia at https://www.fedcourt.gov.au/law-and-practice/practice-documents/practice-notes/gpn-expt

## 1.2. Guideline 1

The Report centres the argument for the proposed Guideline 1 on the results of modelling carried out by the Alcohol Research Group of the University of Sheffield. This is described in an associated report ("the Sheffield Report").[3] This model, derived from earlier work mainly concerning pricing policies for alcohol, examines the effect of alcohol on the morbidity and mortality of the community.

An example of the output of the model is shown in Figure 1.

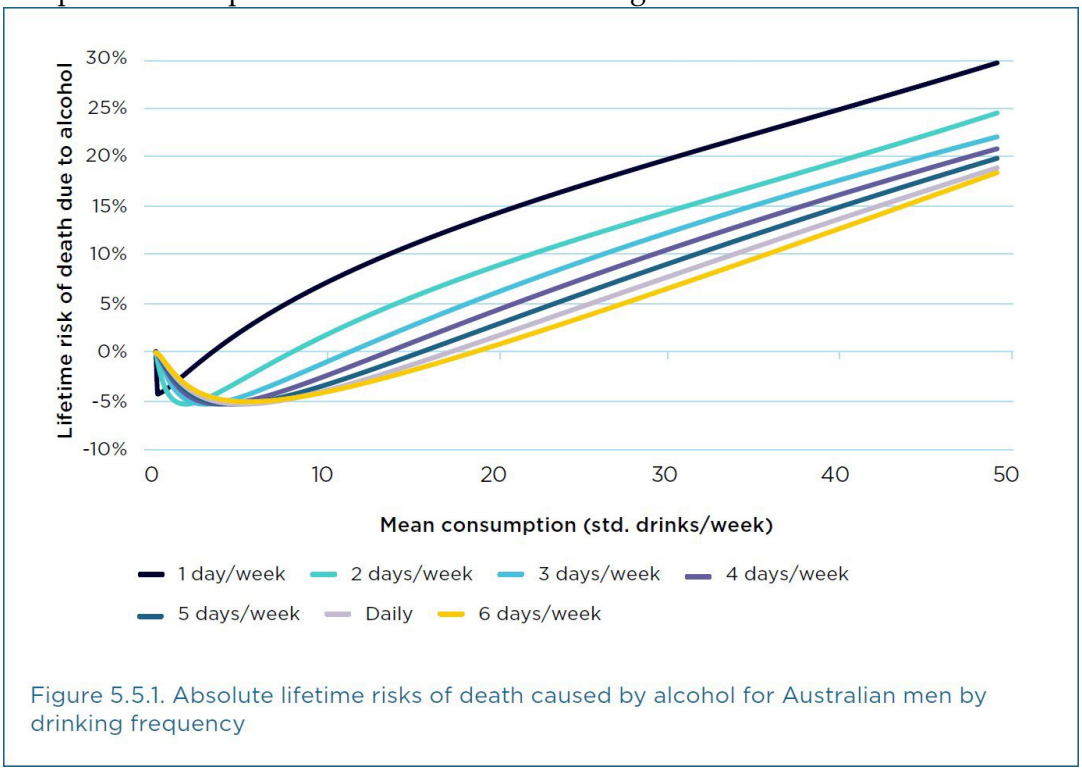


Figure 5.5.1. Absolute lifetime risks of death caused by alcohol for Australian men by drinking frequency

**Figure 1. Output from the Sheffield model for Australian men as presented in the Report. Note that the labels for "Daily" and 6 days/week" are incorrectly interchanged.**

Several features are worth noting:

- For various levels of alcohol consumption, measured in terms of the average number of drinks per week, the heightened risk of dying from an alcohol related health condition is presented.
- The separate curves distinguish between different drinking patterns, summarised by the number of days per week the drinking is spread over. Above about five drinks a week, for any average level of drinking the risk is lower is the consumption is more spread through the week.

[3] Angus, C., Henney, M., Meier, P., Brennan, A. and Holmes, J., Mortality and morbidity risks from alcohol consumption in Australia: Analyses using an Australian adaptation of the Sheffield Alcohol Policy Model (v2.7) to inform the development of new alcohol guidelines Final report August 2019, University of Sheffield.

- For low levels of consumption, the heightened risks are actually negative. This is due to the protective effects of alcohol in regard to several heath conditions.

If an "acceptable" level of risk is chosen, the model then gives possible acceptable behaviours, that is, points on the lines below the acceptable risk level. The Report considers several levels of absolute risk of death due to alcohol related causes – 0.1%, 0.2%, 1% and 2%. These risk levels are displayed in Figure 2. It can be seen that these risk levels have only a minor effect on the points where the risk curves intersect them – that is, over the range of risk levels considered they have minimal effect upon recommendations for drinking, in the order of 2 or 3 per week.

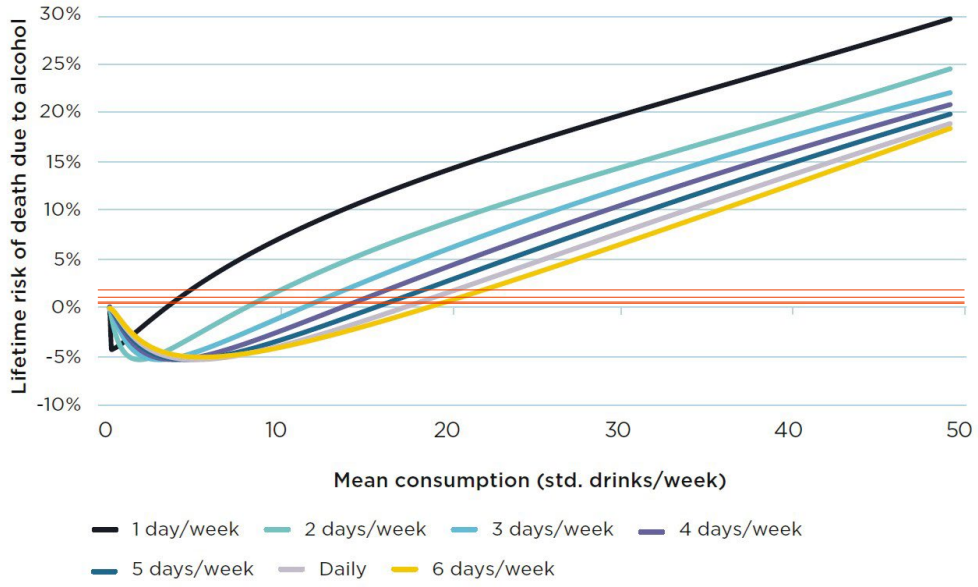


Figure 5.5.1. Absolute lifetime risks of death caused by alcohol for Australian men by drinking frequency

**Figure 2. The risk curves for Australian men with the four levels of risk considered in the Report overlaid in red.**

A second issue is that each curve assumes not just a uniform drinking behaviour but rather a certain pattern of behaviour. In particular it assumes a relationship between the average consumption and the frequency of binge drinking. Indeed, it is likely that this aspect of the model is the major contributor to why the seven curves in Figure 1 or Figure 2 differ from each other.

A third issue with such an approach is which curve or curves should be used in developing the guideline. One approach would be to develop a guideline that uses all the curves so that drinkers can effectively decide which best applies to them. The Report takes the approach of choosing the "3 days/week" curve, based upon less than ideal survey evidence on what might be typical drinker behaviour.[4]

[4] See the Report, page 60. It is acknowledged there that such measures are somewhat imprecise, in part due to the well-known problem of under self-reporting of drinking in such surveys.

A fourth issue is that the evidence strongly supports a difference between men and women in the risks of alcohol, with women generally being more susceptible to harm. This is illustrated most clearly in Figure 3 that is taken from the Report. The guideline does not reflect this difference.

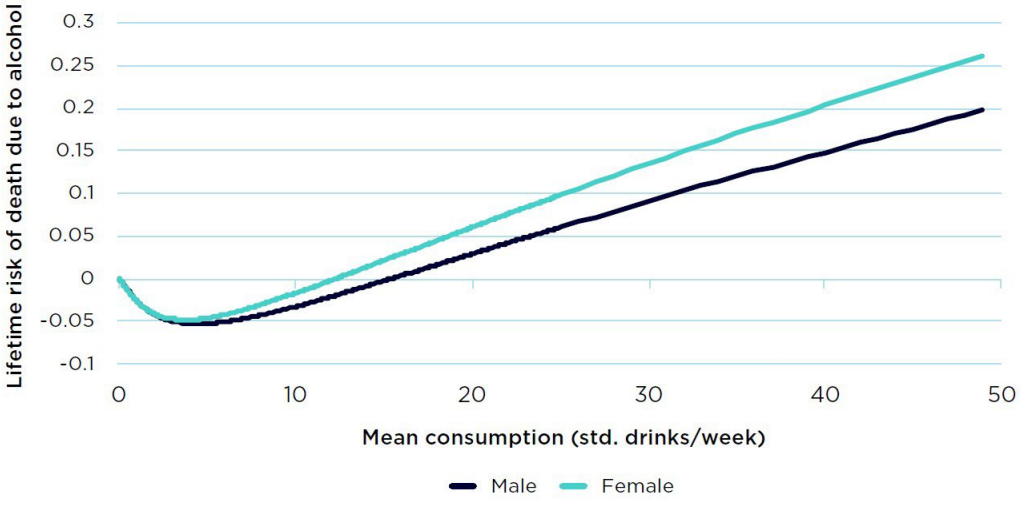


Figure 5.5.3. Absolute lifetime alcohol-attributable mortality risk for adults who drink spreading their consumption evenly across 5 days per week.

**Figure 3. Comparison of males and females according to the Sheffield model for persons drinking across five days a week. (Source: the Report.)**

The modelling approach has certain elements to recommend it, but caution must still be exercised. The outputs are only as meaningful as the assumptions in the model, the data used to calibrate it and its relevance to the issues being addressed.[5] To that end, this review emphasises the application of this model and questions whether the model is fit for the current purpose and whether it has been used appropriately.

## 2. The Sheffield Model

The Sheffield Alcohol Research Group (SARG) developed the Sheffield Alcohol Policy Model (SAPM) over a number of years. It is probably best described as an evolving project. The initial Version 1 was created in 2008 and for the current project Version 2.7 was used, with adaptations to Australian conditions.

It must be pointed out that the SAPM was designed for a more all-encompassing perspective, looking at the effects of policy changes and interventions. For example, the most widely quoted application has been to examine the possible effects of "minimum unit prices" for alcohol, a policy change that the SARG has become closely associated with. The section of the model relevant to the current context is that which goes from alcohol consumption to possible harms.

[5] The classic statement by the highly respected statistician George Box is pertinent here: "All models are wrong but some models are useful".

The use of an existing model to answer a new problem is often an efficient step. However the decision must consider whether the model is sufficiently suitable – typically it was designed with other constraints, data or assumptions in mind and how well it can be adapted will vary.

A fundamental potential problem with the SAPM is that it was designed to estimate population level effects while the guidelines are intended to give advice at personal level. Even if SAPM is able to give meaningful estimate of the effects of population level interventions such as pricing policies, that does *not* mean that one of its internal modules can be readily applied at a personal level.

## 2.1. Reviewing the Model

Documentation for the SAPM is not readily available. There are some published papers[6] and some internal reports[7] that are publically available. Not surprisingly these sources cover various versions of SAPM and, most critically, do not cover Version 2.7 or, perhaps more importantly, the adaptations for Australian conditions (also referred to as SAPM-AU). The report of the Sheffield group on their Australian work[8] describes some of the modifications at a high level, but omits many details.

Our understanding is that the NHMRC does not have access to a copy of the SAPM, and in particular not a copy of the version as used here (SAPM-AU). To our knowledge the SAPM-AU has not been independently reviewed, and it is not clear that it is capable of being independently reviewed. This naturally raises great concern – from our perspective **the model is effectively a black box that is claimed to function in certain ways by the developers but which comes with no independent evaluation of its correctness or appropriateness for the task at hand**.

In our opinion this does not represent open science and hence it does not represent good science. We strongly urge the NHMRC to remedy this situation, either by making the model openly available or by refraining from using it.

Once commissioned to conduct the review, Data Analysis Australia began the process of attempting to gain access to the SAPM-AU and the data used to calibrate it, with the first request being made to the NHMRC on 20 December 2019. We were informed that the NHMRC did hold a copy of the model and we would need to contact SARG directly. We did so on 24 December, but not surprisingly we ran into

[6] Most notably Brennan A, Meier P, Purshouse R, et al. The Sheffield Alcohol Policy Model: A Mathematical Description. *Health Econ* 2014; **24**(10): 1368-88.

[7] The report Purshouse, R., et al, *Modelling to access the effectiveness and cost effectiveness of public health related strategies and interventions to reduce alcohol attributable harm in England using the Sheffield Alcohol Policy Model version2.0*, November 2009, and its associated appendices perhaps give the greatest insight to the SAPM, albeit as it was in 2009.

[8] Angus, C., Henney M., Meier P. Brennan A., Holmes J., *Mortality and morbidity risks from alcohol consumption in Australia: Analyses using an Australian adaptation of the Sheffield Alcohol Policy Model (v2.7) to inform the development of new alcohol guidelines Final report*, August 2019

issues of the Christmas-New Year break. In preparation for receiving the model Data Analysis Australia also obtained from the Australian Institute of Health and Welfare (AIHW) the data used in the model.

The first meaningful response (apart from acknowledgements of our request) was received on 21 January 2020, declining to give us access. The reason given was that "the Sheffield Alcohol Policy Model (SAPM) is a large and complex model that is not designed to be used by independent third parties. … It would be extremely difficult for any third party, even one with significant statistical expertise, to use the model to accurately replicate or interrogate our results or understand and assess the modelling methods without substantial support from our team, which we are not in a position to provide."

A request for reconsideration of this decision was on 24 January, emphasising the capability of Data Analysis Australia, but was rejected on 30 January. The SARG has responded to questions put to them, but at the time of writing, not providing what we regard as a sufficient level of technical detail.

## 2.2. Model Structure

Without full access to the model we are only able to hypothesise on some aspects of the model, being guided by the Sheffield Report and the responses to questions sent to the Sheffield Group. However it seems that for a given gender, behaviour and possibly age, it calculates the probability of an adverse health outcome for each of 42 health conditions. The aggregate of such probabilities is then summarised into results such as those presented in Figure 1. We presumably that the risk-dose curves for each of the health conditions were combined with Australian mortality and morbidity data (provided by the AIHW via the NHMRC) to calculate lifetime risks.

The original SAPM defined several groups of drinkers, based upon how heavily they drank – Moderate Drinkers, Hazardous Drinkers and Harmful Drinkers. For each group there is a range of alcohol consumption and a range of probabilities of binge drinking. It appears that "both the likelihood and scale of the binge (how much is drunk on each occasion) are strongly associated with mean consumption".[9] It appears that SAPM-AU takes a similar approach although the details are not clear.[10] The division of drinkers according to how many days per week they spread their drinking over clearly is a move in the direction of moving from a unidimensional description of drinking to two dimensional, and as such must be acknowledged as an improvement, but it is not without its faults:

- There appears to have been an intention to characterise current drinking behaviour. For example, if a drinker consumes $x$ drinks per week over $y$ days, then it assumes that they will have probability $z$ of binge drinking. This might be sensible if the aim is to model population level issues, but it is not appropriate if

[9] Purshouse, R., et al, *Modelling to access the effectiveness and cost effectiveness of public health related strategies and interventions to reduce alcohol attributable harm in England using the Sheffield Alcohol Policy Model version2.0*, November 2009, page 21.

[10] We have requested but yet to receive this information from SARG.

the aim is to develop sensible guidelines for individuals. For example, it does not help develop a guideline that might emphasise the avoidance of binge drinking.

- Our understanding is that the models for the relationship between average consumption and binge drinking were adapted for Australian conditions but not based solely on Australian data. In particular, the only Australian data referenced in this regard is the National Drug Strategy Household Survey 2016 from the AIHW. Our examination of the questionnaire and the data dictionary[11] indicates that this does not contain ideal information by itself to establish such relationships by itself. It appears that some aspects of the model have been derived from other data. In addition, such survey data tends to significantly understate actual alcohol consumption.[12]
- The second dimension – the number of drinking days per week – does not necessarily assist in setting guidelines as this, as seems to be implemented in the model, embodies both the typical consumption on a drinking day and the propensity to binge. Indeed, it is not clear to what extent harm as estimated by the model is due to average consumption levels, typical consumption on drinking days or binge drinking.

It is notable that the model does not appear to have been used to evaluate the harm for an individual who follows the existing guidelines.[13] In that sense, there appears to be no attempt to demonstrate a quantitative need to change the guidelines.

## 2.3. Risk Functions

The relationships between alcohol consumption and heath conditions are the subject to continual study as both the causal mechanisms are better understood and as quantitative epidemiological studies attempt to estimate the precise form of the relationships. The SAPM-AU appears to have compiled a reasonably large body of information on these relationships and used them in the model. As expected, these relationships are estimated by a number of different authors. They are displayed in Figure 5 of the Sheffield Report. We have not been able to review all of these but have no reason to believe that there are significant problems in them.

Having said that, the curves must be all treated with a small degree of caution:

- They are all estimated from studies of varying size and quality. All these studies are observational and hence while efforts would have been taken to eliminate or adjust for confounding variables, they cannot be regarded as definitive. (The medical gold standard approach of a randomised clinical trial would be both unethical and impractical.)
- This is particularly the case where the risks are low, the situation that generally occurs with low levels of alcohol consumption since the risks must be estimated

[11] Both obtained from the AIHW.

[12] See for example, Livingston, M. and Callinan, S.,2015, Underreporting in Alcohol Surveys: Whose Drinking Is Underestimated?, *Journal of studies on alcohol and drugs* 76(1):158-167.

[13] Personal communication, *[NHMRC has removed personal information]*, SARG.

by relatively few cases. In such cases, the precise form of the functions used can be somewhat arbitrary. The SAPM-AU appears to have largely used smooth functions – typically low order polynomials – but in many cases these are statistically indistinguishable from threshold curves where the risk is assumed constant up to some threshold. We do not believe it to be fruitful to argue over such details.

- A more important issue is to ensure that models that are too simple have not been fitted. It is far from unknown for risk functions to be linearly interpolated between the relative risk of 1 for non-drinkers and the high risks associated with high levels of alcohol consumption.

We note that the Sheffield Report investigated the effect of assuming thresholds below which it is assumed that the risks are the same as for non-drinkers (their scenario SA2). This is effectively changing the risk curves without providing a data or evidence driven reason.[14] We see no scientific merit in this and believe that such considerations should not influence the Australian guideline. In any event, the effect on possible guidelines appears minimal.

The Sheffield Report also explored the effect of assuming that there are no protective effects (their scenario SA1). We recognise that there is some scientific debate about the extent or indeed the existence of such protective effects:

- All studies for the effect of alcohol are essentially observational in nature and hence subject to possible interference from confounding factors or selection effects. For example, it is sometimes suggested that the apparent protective effect may be because the cohort of non-drinkers has been affected by previous heavy drinkers who stopped drinking due to the effects on their health. It is not unreasonable to accept that there are some such effects, but it is not appropriate to assume that the effects predominantly act in a certain direction and are enough to explain the apparent protective effect.
- Some authors have suggested that new methods of analysis, particularly using Mendelian randomisation, that overcome the problems of observational studies, giving the benefits of a randomised clinical trial. In our opinion the claims for such methods are somewhat optimistic. In particular the application of Mendelian randomisation using a gene (ALDH2) that strongly affects a person's tolerance of drinking alcohol can only provide a crude estimate of a risk function, one that is insufficient to seriously explore its precise form.

Mendelian randomisation is a statistical method of analysis that uses an additional genetic variable that is considered to be closely related to the exposure variable (the level of drinking) and not related to the outcome variable (health outcome) except through the exposure variable. Since the genetic variable can be considered truly random, if it appears to be related to the outcome then this implies that the exposure

[14] We understand that this was done in some of the British modelling, setting the threshold to match what was at the time their recommended guidelines.

variable must also be related to the outcome. The statistical justification for doing this is that it eliminates confounding and selection effects.

There are several difficulties that can arise in such an analysis:

- The assumption that the genetic variable has no effect on the outcome except through the exposure may be incorrect. Even when there is no known direct or alternative effect, doubts will always remain that there might be such an effect. In that sense, it replaces doubts about confounders with doubts about causal pathways.
- The statistical methodology of Mendelian randomisation (and the more general method of instrumental variables) involves projecting both the exposure variable and the outcome variables onto the space spanned by the genetic variable. If only a single gene is used as the genetic variable (such as ALDH2) which takes three possible values corresponding to the three phenotypes, this effectively reduces the data to three points. It is impossible to fit a complex risk function to three points.
- Increasing the number of genes used can reduce the second problem but is likely to increase the first problem.

For these reasons Mendelian randomisation analyses must be treated with great caution, particularly when estimating risk functions.

The Sheffield Report has an additional problem in its SA1 scenario. The resulting absolute risks are uniformly higher across all drinking levels than for the base case. This implies a greater number of alcohol related deaths. This is effectively implying that there is substantial undiagnosed or unobserved alcohol related mortality. No reason is given for this.

We strongly believe that the results of the scenario SA1 in the Sheffield Report have little value. Fortunately the (NHMRC) Report gives only brief reference to this scenario. It also points out that if SA1 was well supported then the methodology would lead to guidelines recommending less than three drinks per week. Such a scenario would lead to a serious questioning of the risk levels considered acceptable in the Report.

At the same time the Report suggests that any reduction in alcohol consumption is good. This suggests that the Report does *not* accept that the protective effect may exist.

## 3. Applying the Model to give Guidelines

The principal outputs of the model are the charts convenience reproduced below as Figure 4 and Figure 5. While recognising that there are substantial limitations to and uncertainties in the model, it can be seen that for a drinker who spreads their drinking over seven days in the week then risks only become significantly larger than for non-drinkers when about 20 drinks is exceeded for men or 14 for women.

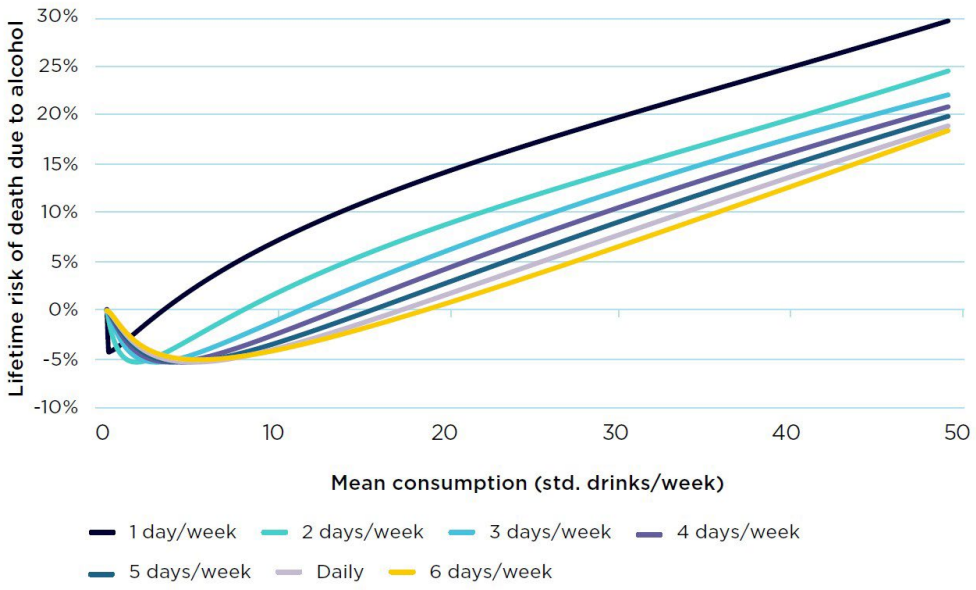


Figure 5.5.1. Absolute lifetime risks of death caused by alcohol for Australian men by drinking frequency

**Figure 4. Output from the Sheffield model for Australian men as presented in the Report. Note that the labels for "Daily" and 6 days/week" are incorrectly interchanged.**

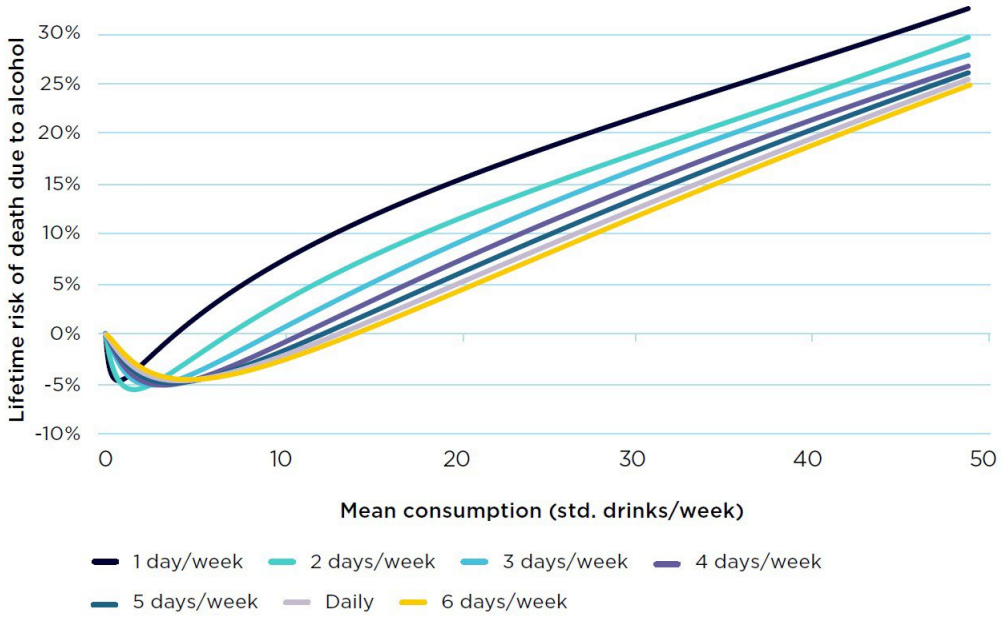


Figure 5.5.2. Absolute lifetime risks of death caused by alcohol for Australian women by drinking frequency

**Figure 5. Output from the Sheffield model for Australian women as presented in the Report. Note that the labels for "Daily" and 6 days/week" are incorrectly interchanged.**

These conclusions from the model appear to be more consistent with the existing guidelines (up to 14 drinks a week and no binge drinking) than with the proposed guidelines. We note that no attempt appears to have been made to directly compare risks with the proposed guideline and the existing guideline.

The Report suggests focusing on the curve corresponding to three drinks per week. In our opinion this is not appropriate. Quite apart from the uncertain data on just how often per week people drink, it is an over simplification of the model outcomes that sends out a crude message.

We cannot comment upon how messages to the community may need to be communicated, but as statisticians it is our belief that decisions should be informed choices. An over-simplification of the message reduces the information.[15]

# 4. Conclusions

**Finding 1. The methodology behind the guideline relies heavily upon modelling conducted (apparently under contract) by the Sheffield Alcohol Research Group (SARG) using an adaptation of the Sheffield Alcohol Policy Model (SAPM). This model has not been made available for independent review and hence does not represent open and transparent science.**

**Finding 2. The SAPM was primarily developed to assess the possible effects of certain alcohol control measures, particularly those relating to prices. These aspects of the model are not necessarily relevant to the context of the guideline and it is not clear whether they impact upon the utility of the model for the current context.**

**Finding 3. There appears to be a substantial disconnect between the outputs of the Sheffield model and the guideline. In particular the draft guidelines (a) lose the difference between males and females, and (b) for people who avoid binge drinking, they understate the average number of drinks that might be considered low risk.**

**Finding 4. The model scenario SA1 that assumes that low levels of alcohol consumption has no protective effect does not exist has a major effect on the model that substantially increases the aetiological fraction mortality attributable to alcohol. This effect is not explicitly presented and no evidence is presented that the aetiological fraction thus calculated is reasonable.**

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[15] In this context it is relevant to note that in the Royal Statistical Society submission to the United Kingdom guideline consultation, the first point was made was "Communications should genuinely reflect the principle of informed choice". We would be in complete agreement with that statement.

**Finding 5. The Report has an inconsistent attitude towards the protective effects of low levels of alcohol consumption. The guideline is fundamentally based upon the assumption that there *is* a protective effect – if there was not then the methodology would lead to a recommended level that is a small fraction of the guideline. However at the same time it is stated that "all alcohol consumption comes with some degree of risk" and the guideline includes the statement "the less you choose to drink, the lower your risk of alcohol-related harm."**

Overall the Report has numerous shortcomings including the lack of transparency and the lack of clear connection between the model results and the proposed guideline. In our opinion it is not acceptable in its current form. It is also very possible (we cannot say likely) that the modelling may have some substantial errors or make assumptions that are not appropriate in the Australian context. Hence we recommend substantia further work be done to correct these deficiencies.