Mortality attributable to drinking, drinking too much, or drinking too little: a comparison of methods

Ian R. White, Annie Britton, Kiran Nanchahal and Klim McPherson

Abstract

Background The existence of a U-shaped relationship between alcohol consumption and all-cause mortality complicates the calculation and interpretation of mortality attributable to alcohol consumption.

Methods We used the relationships between all-cause mortality and alcohol consumption from four British cohort studies. For each study we defined a ‘sensible drinking level’ as the level with lowest observed mortality. We estimated the fractions of deaths that were attributable to (1) any drinking (compared with not drinking), (2) drinking more than the ‘sensible level’, and (3) drinking less than the ‘sensible level’.

Results Data from the Doctors’ study suggest that on balance 22.3 per cent of deaths are prevented by alcohol consumption, yet the fractions of deaths attributable to drinking more than 8–14 units per week and less than 8–14 units per week are nearly equal (6.5 per cent and 6.4 per cent, respectively). In a sensitivity analysis we show that it is possible for alcohol consumption to prevent deaths overall yet for more deaths to be attributable to drinking above a sensible level than are attributable to drinking below the sensible level.

Conclusions The balance of deaths attributable to or prevented by alcohol consumption provides no information about the deaths attributable to drinking above or below sensible levels. Using all-cause data in this way is likely to exaggerate the protective effect of alcohol consumption, so our results are only illustrative.

Keywords: alcohol, attributable mortality

Introduction

The general acceptance that alcohol consumption can protect against coronary heart disease (CHD), and that the relationship between all-cause mortality and alcohol consumption is U-shaped, presents problems for research into the effects of alcohol consumption on all-cause mortality. The possibility that non-drinkers could be encouraged to drink for the sake of their health has been considered, but others are opposed to this.

Studies of mortality attributable to alcohol consumption still generally estimate what would happen if the mortality of all drinkers was changed to that of comparable non-drinkers – that is, no alcohol consumption is taken as the reference level. Where studies have allowed for a protective effect of CHD at all ages, they have typically found that the number of CHD deaths prevented by at all ages combined exceeds the number of deaths from other causes attributable to alcohol consumption.

When deaths at ages under 65 or person-years of life lost are considered, however, those attributable to alcohol consumption generally exceed the number prevented by alcohol consumption.

Holman and English have argued that, as the aim of public policy is not to eliminate alcohol consumption but to reduce unsafe alcohol consumption, studies of mortality attributable to alcohol consumption should estimate the mortality attributable to drinking more than some sensible level. The mortality attributable to drinking less than this level is also relevant to public policy if non-drinkers are to be encouraged to drink or indeed if light drinkers are to be encouraged to stop drinking. In other words, for both cases, some definition of ‘sensible drinking’ is taken as the reference level of alcohol consumption.

This paper uses data from four British cohort studies to estimate the mortality for men in England and Wales attributable to (1) any drinking compared with not drinking, (2) drinking above a ‘sensible level’ compared with drinking at the ‘sensible level’, and (3) drinking below the ‘sensible level’ compared with drinking at the ‘sensible level’.

This work assumes that the observed relationships between all-cause mortality and alcohol consumption are causal. This is broadly accepted, although it has been argued that the observed excess mortality in non-drinkers may be due to confounding and selection.

The use of all-cause mortality in studies of attributable

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mortality is problematic as the relationship between all-cause mortality and alcohol consumption varies with age, so it is unclear to what age-group the results apply. One major review found that using studies of all-cause mortality to estimate the total attributable mortality at all ages overestimated the beneficial effects of alcohol consumption. The aim of the present work is therefore not to produce definitive estimates but to explore the differences between the results using different levels of alcohol consumption as the reference category.

Methods

We used results reported by the four British cohort studies that have reported all-cause mortality for four or more categories of alcohol consumption: the Whitehall study, the British Regional Heart Study (BRHS), the Doctors study and the Scottish Heart Health Study (SHHS). The SHHS included men and women whereas the other studies included only men. Each study reported the number of subjects and an adjusted relative risk in each alcohol consumption category.

Using the results of each study in turn, we performed three calculations of attributable deaths. We first computed the proportion of deaths attributable to drinking, which is the proportion of observed deaths that would have been avoided if the risk of all drinkers were changed to equal the risk of (suitably matched) non-drinkers.

We then attempted to identify a level of alcohol consumption, termed the ‘sensible drinking level’, associated with low risk. For ease of exposition (and to avoid appearing to have solved this difficult question), we chose the category in each study whose observed risk was lowest; an alternative choice, for example selecting a level of 10 units per week, would have given similar answers for men.

We then computed the proportion of deaths that would have been avoided if the risk of all those drinking above the ‘sensible drinking level’ were changed to equal the risk of those drinking at the sensible drinking level, and similarly for those drinking below the sensible drinking level.

The calculations were carried out using the standard method, which is described in the Appendix. Where the total number of deaths would increase if the risk of all subjects drinking more than the reference level were reduced to the risk at the reference level, the number of attributable deaths is negative but is computed by exactly the same methods; dropping the negative sign gives the number of prevented deaths. We have not used the concept of the prevented fraction.

Results

Figures 1 and 2 show the relationship between all-cause mortality and alcohol consumption reported by each of the
studies. The width of each bar is proportional to the number of subjects in a category of alcohol consumption, and the height is proportional to the mortality risk. This form of display is useful because areas on the graph are exactly proportional to numbers of deaths. A ‘sensible drinking level’ in each study is taken as the level with the lowest observed risk. The numbers of deaths attributable to drinking above and below the ‘sensible level’ are proportional to the areas marked ‘A’ and ‘B’, respectively, lying between the observed relative risk for each category of alcohol consumption and the lowest relative risk of all categories. The number of deaths attributable to any drinking is not indicated by shading: it is proportional to the area between the observed relative risks and the dotted line, which is drawn at a relative risk of unity. Areas where the graph lies below unity represent deaths prevented by alcohol consumption.

Table 1 presents the corresponding values of the population attributable fraction (PAF). For drinkers compared with non-drinkers, negative values indicate an overall protective effect of alcohol consumption, whereas positive values indicate an overall harmful effect. The data from each study except the BRHS suggest that more deaths are prevented by drinking (compared with not drinking) than are attributed to it. However, the data from three of the studies suggest that slightly more deaths are attributed to drinking above a ‘sensible level’ than are attributed to drinking below this ‘sensible level’.

A total of 1584 subjects in the Doctors study who reported ‘less than weekly’ consumption were excluded from analysis because subjects who preferred not to state their intake were invited to give this response. A further 43 subjects who volunteered that they were ex-drinkers were excluded. To illustrate a further situation which could arise, we made the (implausible) assumption that these 1627 men were in fact drinkers of 43 or more units per week, increasing the size of this category from 8 per cent to 23 per cent of the doctors. The PAF for drinkers compared with non-drinkers was reduced to $-17.8\%$, still representing a substantial number of deaths prevented by drinking, but the PAF for drinking above the sensible level was now 10.8 per cent and far exceeded the PAF for drinking below the sensible level, which was 5.3 per cent.

### Discussion

Calculations of the mortality attributable to alcohol consumption are fraught with problems. They generally use the relative risks reported by observational studies (expressing the mortality risks at different levels of alcohol consumption). They must assume that these relative risks represent true causal effects of alcohol consumption and are not confounded by unknown or known factors, or biased by factors such as the misclassification of heavy drinkers as non-drinkers. They must extrapolate these relative risks to the population of interest. When the relative risks used compare all drinkers with non-drinkers, this extrapolation implicitly assumes that the distribution of drinking is the same in the population of interest and in the study population, whereas, for example, heavy drinkers may be under-represented in study populations. These calculations also typically assume that the relative risks are the same for men and women and for all ages, even though the majority of studies have concentrated on middle-aged men.

Even if all these problems are overcome, a further problem derives from the U-shaped curve: mortality has traditionally

### Table 1: Estimated population attributable fractions using the results of each cohort

<table>
<thead>
<tr>
<th>Study</th>
<th>Sensible level (units/week)</th>
<th>Drunk (%)*</th>
<th>Drinking above sensible level (%)†</th>
<th>Drinking below sensible level (%)†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Doctors²¹</td>
<td>8–14</td>
<td>−22.3</td>
<td>6.5</td>
<td>6.4</td>
</tr>
<tr>
<td>Whitehall¹⁹</td>
<td>0.1–26†</td>
<td>−26.2</td>
<td>5.2</td>
<td>15.9</td>
</tr>
<tr>
<td>BRHS²⁰</td>
<td>1–15</td>
<td>3.0</td>
<td>6.1</td>
<td>4.3</td>
</tr>
<tr>
<td>SHHS men²²</td>
<td>16–29</td>
<td>−5.6</td>
<td>7.1</td>
<td>5.3</td>
</tr>
<tr>
<td>SHHS women²²</td>
<td>1–2</td>
<td>−13.7</td>
<td>11.7</td>
<td>15.6</td>
</tr>
</tbody>
</table>

*Compared with not drinking.
†Compared with drinking at sensible level.
‡0.1–34 g/day.
been attributed to any drinking, but this is not an appropriate measure of the impact of drinking.

This paper used relationships between alcohol consumption and mortality risk from four British studies to estimate the fraction of deaths attributable to any drinking. This figure can be viewed as a balance of prevented deaths in lighter drinkers against attributable deaths in heavier drinkers, or alternatively as a balance of prevented deaths from CHD against attributable deaths from other causes.

The same relationships were also used to estimate the fractions of deaths attributable to drinking above a ‘sensible level’, and to drinking below this ‘sensible level’. In the absence of a consensus on the optimal level of drinking, we used the apparent optima from the separate studies. It would, however, be more realistic to recognize that the true optimal level of drinking varies by age and sex and between individuals.

Using the results of the British Doctors’ study, we estimated that 22.3 per cent of deaths were prevented by alcohol consumption – that is, that the total number of deaths would have been 22.3 per cent higher if the risk of all drinkers had been changed to equal the risk of non-drinkers. Eight to 14 units per week was taken as the ‘sensible level’ for all of the doctors, as it carried minimum risk of death. If the risk of all drinkers and non-drinkers were reduced to this minimum risk then total mortality would be reduced by 12.9 per cent and roughly equal numbers of deaths would be saved in those who were previously drinking above the sensible level and in those who were previously drinking below the sensible level. Put crudely, although the net effect of alcohol consumption is a large benefit, the public health benefit potentially achievable by encouraging heavy drinkers to drink less is as large as that potentially achievable by encouraging non-drinkers to drink and those drinking 1–7 units/week to drink more.

Similar patterns are seen using the results of the SHHS. The results of the Whitehall study differ in that more deaths are attributed to drinking below a sensible level than are attributed to drinking above this sensible level, whereas the BRHS differs in estimating a small positive fraction of deaths attributable to alcohol consumption. We used a final possibility for making the unlikely assumption that the doctors whose alcohol consumption was unclear (because of the design of the questionnaire in this study) were in fact all heavy drinkers. This showed that although any drinking is still associated with prevented deaths, twice as many deaths were attributed to drinking above a sensible level than were attributed to drinking below a sensible level.

The effect of alcohol consumption on all-cause mortality is likely to vary strongly with age and sex, being more protective in men and at older ages where there is more CHD. On the other hand, effects of alcohol consumption on cause-specific mortality may not vary with age. Thus methods using all-cause mortality are likely to overestimate the protective effect of alcohol consumption if they are applied to populations younger than those in the cohort studies from which the relative risks were derived or if the relative risks apply largely to men. Our results should therefore be taken as illustrative: definitive results would require the summation of cause-specific calculations.

As policy-makers begin to consider whether to encourage non-drinkers to drink and very light drinkers to drink more,1,25 it becomes important to know the fraction of deaths attributable to drinking below a ‘sensible level’ as well as the fraction attributable to drinking above this level. These fractions represent deaths potentially avoidable by changing the distribution of alcohol consumption, but the practical difficulties of implementing such a change are great and beyond the scope of this paper.

Policy-making must in addition take into account the distribution of deaths at different ages, perhaps through analysis of person-years of life lost, which tends to weight deaths attributable to alcohol consumption more heavily than deaths prevented. It must also, of course, take account of morbidity and social effects of alcohol consumption, which we have not considered here.

In conclusion, the growing body of literature that shows that more deaths are prevented by alcohol consumption than are attributed to alcohol consumption gives no grounds for complacency. It is more important to separately quantify deaths attributable to drinking above sensible levels; these are likely to outnumber the deaths attributable to drinking below sensible levels.

Acknowledgements
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Appendix: formulae for attributable fraction

Let us suppose that we have several categories of alcohol consumption, labelled \( i = 0 \) to \( I \), where \( i = 0 \) represents non-drinkers. The proportion of the population in group \( i \) is \( p_i \) and their risk (relative to non-drinkers) is \( RR_i \) (so \( RR_0 = 1 \)). Then the fraction of all deaths attributable to any drinking is

\[
\frac{\sum_{i=1}^{I} p_i (RR_i - 1)}{\sum_{i=1}^{I} p_i RR_i}
\]

whereas the fraction of all deaths attributable to drinking more than group \( r \) drink is

\[
\frac{\sum_{i=r+1}^{I} p_i (RR_i - RR_r)}{\sum_{i=0}^{I} p_i RR_i}
\]

and the fraction of all deaths attributable to drinking less than group \( r \) drink is

\[
\frac{\sum_{i=0}^{r-1} p_i (RR_i - RR_r)}{\sum_{i=0}^{I} p_i RR_i}
\]