

Summary

The aetiological fraction methodology and the associated fraction estimates enable estimation of the proportion of cases of an illness or injury that can be attributed to a risk factor. This report presents aetiological fraction estimates attributing deaths and hospital separations resulting from a range of specific illnesses or injuries to tobacco, alcohol and illicit drugs. The fractions represent a revision of the fractions originally presented by Holman et al. (1990) and later revised by English et al. (1995). Also presented here are estimates of 1998 mortality and 1997–98 hospital separations attributable to alcohol, tobacco and illicit drugs based on the revised fractions.

Deaths attributable to alcohol, tobacco and illicit drugs

In 1998 an estimated 19,019 people died in Australia as a result of tobacco smoking. A further 1,023 deaths can be attributed to illicit drugs. For tobacco smoking, the majority of deaths (14,799) occurred at ages 65 and over. However, because of the time lag between exposure to tobacco smoke and the onset of many diseases, particularly cancer and chronic obstructive pulmonary disease, many of these deaths represent the result of tobacco smoking at a much earlier age. The majority of deaths attributed to illicit drugs (649) occurred between the ages of 15 and 34 years.

The effect of alcohol consumption on illness and injury is more complex. In 1998 an estimated 3,271 people died as a consequence of hazardous and harmful levels of alcohol consumption. In addition to the harmful effects, however, when consumed at moderate levels alcohol appears to be associated with a decrease in heart disease and stroke. The number of people in Australia who drink at moderate levels far outweighs the number who drink at hazardous or harmful levels, so this apparent protective effect is greater for the overall population than the harmful effect for deaths, though not for potential years of life lost. Thus the estimated net reduction in deaths associated with alcohol consumption in 1998 was 2,371 but the estimated net potential years of life lost due to alcohol consumption in 1998 was 21,147.

The reason that alcohol appears to be associated with a net decrease in deaths but a net increase in potential years of life lost is because the decrease applies to illnesses which occur at older ages while the harmful effects apply across all ages. Deaths at younger ages contribute more potential years of life lost than deaths at older ages. Thus in 1998 the net effect of alcohol consumption at ages below 65 years was to cause an estimated 2065 deaths, leading to 47,887 potential years of life lost, while the net effect at ages 65 years and over was associated with a decrease of 4,436 deaths or 26,739 potential years of life lost.

Hospital separations attributable to tobacco, alcohol and illicit drugs

In 1997–98, 142,525 hospital separations in Australia were attributable to tobacco smoking and 14,471 to illicit drugs. For tobacco, the majority of separations (74,379) occurred at ages 65 and over; for illicit drugs the majority of separations (10,876) occurred at ages 15 to 34.

In 1997–98 an estimated 71,422 separations could be attributed to harmful and hazardous levels of alcohol consumption. However, once the estimate is adjusted for the decrease in heart disease and stroke associated with moderate alcohol consumption, the net overall number of separations was 43,033.

Revision of the aetiological fractions

Aetiological fractions depend on the prevalence of a risk factor and the associated relative risk of a particular illness or injury. The fractions presented here have been revised where possible—from the earlier reports of Holman et al. (1990) and English et al. (1995)—to incorporate the most recent estimates of the prevalence of use of tobacco, alcohol and illicit drugs. In addition, the relative risk estimates for some conditions have been revised to incorporate the results of recent research. The following conditions were selected for this detailed study and risk-ratio revision:

- in relation to alcohol—breast cancer, stroke, road injuries and fall injuries;
- in relation to tobacco—cervical cancer and peptic ulcer;
- in relation to illicit drugs—road injuries.

The relative risk of breast cancer associated with alcohol consumption was examined because recent research suggests that the risk varies with age. However, the analyses presented in this report failed to show a statistically significant difference in the risk for older women compared with younger women, and the overall risk-ratio estimate incorporating the results of recent research was similar to that derived by English et al.

The relative risk of stroke associated with alcohol consumption was examined because recent research suggests that the risk differs between ischaemic and haemorrhagic stroke. The analyses presented in this report support this, so different fractions were estimated for the two different types of stroke.

The relative risk of road injuries associated with alcohol consumption was examined using only Australian data—rather than a combination of Australian and international data, as used by English et al. Separate fractions were derived for motor vehicle drivers or motorcycle riders and for pedestrians, and these in turn were derived separately for hospital separations and deaths.

The relative risk of fall injuries associated with alcohol consumption was examined because of evidence that the risk varies with age. The analyses presented in this report support this, so separate fractions were derived for people aged 65 years and over and for people aged less than 65.

The relative risk of cervical cancer associated with tobacco smoking was examined because of recent research results on the causes of this cancer. Similarly, the relative risk of peptic ulcer associated with tobacco smoking was examined because of recent research results on the causes of peptic ulcer.

English et al. did not derive a fraction road injuries associated with illicit drug use because of the lack of suitable Australian data. More recent research has, however, provided such data so an estimate of this aetiological fraction is presented here.

In addition to using recent prevalence data and relative risk estimates, the aetiological fraction methodology for tobacco was revised in two ways. The first was to adjust for the time lag between tobacco exposure and the onset of related illnesses. English et al. used an estimate of current smoking prevalence in their calculation of aetiological fractions for tobacco. But for, many conditions there is a long time lag between exposure to tobacco smoke and the associated ill-effects—in the case of cancer it may be many decades. So for these conditions estimates of the current prevalence of smoking are not helpful in understanding the current associated disease burden.

We followed the Australian Burden of Disease Study (Mathers et al. 1999) in using the method proposed by Peto et al. (1992) to adjust for this time lag. Peto et al. proposed using an artificial compound prevalence measure of tobacco exposure, derived from a comparison between lung cancer rates in the country of interest and lung cancer rates among non-smokers observed in a large long-term follow-up study in the United States. This method was used here to determine tobacco exposure for cancer and chronic obstructive pulmonary disease. The mean time between tobacco exposure and the other illnesses and injuries discussed in this report is considerably shorter than that for cancer and chronic obstructive pulmonary disease, so estimates of current tobacco exposure were used for these other conditions.

The second modification to the methodology for tobacco involved the inclusion of estimates of passive exposure to tobacco smoke and its health effects in pregnancy and childhood. These estimates were based on data in the National Health and Medical Research Council's report on passive smoking (NHMRC 1997).

As with tobacco, current exposure to alcohol does not reflect the relevant exposure for some current outcomes, such as cirrhosis and cancers. There is, however, no equivalent of the method used by Peto et al. to adjust for this time lag. This report followed English et al. in using current prevalence estimates for alcohol consumption in calculating the aetiological fractions. The prevalence of alcohol consumption, particularly of heavy drinking, has declined in recent decades, so it is likely that these methods underestimate the true aetiological fractions of some current health outcomes attributable to alcohol consumption.

The final modification to the methodology of English et al. was to estimate the full attributable effect of alcohol consumption, including the apparent benefits of moderate consumption. English et al. calculated aetiological fractions for hazardous and harmful alcohol consumption (as defined by the NHMRC) relative to low alcohol consumption. These differed from the earlier estimates derived by Holman et al., which were calculated with abstinence as the reference category. Using low alcohol consumption as the reference category, English et al. sought to reflect more accurately the idea that unsafe drinking—as opposed to low alcohol consumption, which may be protective—is the cause for concern.

Even at low levels of consumption, however, alcohol raises the risk of some conditions. Further, the approach taken by English et al. does not allow for the quantification of conditions prevented as a result of the beneficial effects of low levels of alcohol consumption. This report followed the earlier approach of Holman et al. and derived fractions to reflect both the risks and benefits of alcohol at all levels of consumption relative to abstaining from alcohol. Hence the estimates of alcohol-related deaths and hospital separations represent the net effect of both the alcohol-related harm and the alcohol-related benefit. The only exception to this is the fraction for the effect of alcohol on road traffic accidents: although there is some evidence that low levels of alcohol consumption raise the

risk of road traffic accidents at some ages, we followed English et al. in deriving the aetiological fraction with the legal level of alcohol consumption in drivers as the reference level.

Public health efforts in Australia are directed towards reducing unsafe alcohol consumption, rather than alcohol consumption per se. Therefore, although the primary purpose of this report is to estimate the total effect of alcohol consumption, it also presents, as Appendix A, a separate calculation using the approach taken by English et al. These data represent the extra effect of alcohol consumption for the 'unsafe' drinker compared with the 'responsible' drinker (English et al. 1995, p. 58), where unsafe and responsible consumption are defined by the NHMRC guidelines for responsible drinking (NHMRC 1992).