# The Minimal Evidence for Minimum Pricing

## The fatal flaws in the Sheffield Alcohol Policy Model

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# Executive summary

1. The Conservative Party and the Scottish National Party have both stated their intention of introducing a minimum floor price for alcohol, levied at around 50p per unit. Advocates of minimum pricing claim that the policy will significantly reduce alcohol consumption and the problems associated with hazardous drinking.

2. Estimates of how minimum pricing will affect health outcomes have overwhelmingly come from a single computer model—the Sheffield Alcohol Policy Model. This paper argues that the model is based on unreasonable assumptions which render its figures meaningless.

3. Amongst the problems with the Sheffield model is its false assumption that heavy drinkers are more likely to reduce their consumption of alcohol as a result of a price rise. Its calculations are based on controversial beliefs about the relationship between per capita alcohol consumption and rates of alcohol-related harm. Its assumptions about the relationship between price and consumption have frequently been refuted by real world evidence.

4. The Sheffield model provides figures without estimates of error and ignores statistical error in the alcohol-harm relationship. Data is drawn from different populations and applied to England and Scotland as if patterns of consumption and harm are the same in all countries. When data is not available, the model

resorts to what is essentially numerology. Insufficient data is provided for the model to be recreated and tested by third parties.

5. The model ignores the likely effects of minimum pricing on the illicit alcohol trade, it disregards the health benefits of moderate drinking and fails to take account of the secondary poverty created by regressive price rises. The decline in alcohol consumption seen in Britain in recent years has not led to the outcomes predicted by the model.

6. We conclude that predictions based on the Sheffield Alcohol Policy Model are entirely speculative and do not deserve the exalted status they have been afforded in the policy debate.

# Disposable statistics

by Christopher Snowdon

We live in an age of disposable statistics which are often better suited to justifying predetermined policies than to informing rational debate. Few would argue with the need to bring evidence to the table when decisions are made, but in the search for 'evidence-based policy', it is easy to mistake social science for hard science and wild speculation for verifiable fact. Predicting the outcome of an untried policy is at the softest end of social science and yet educated guesses about what people might or might not do in certain circumstances are afforded the same respect as the laws of nature. Human behaviour is not as predictable as the orbit of Halley's Comet and lazy sub-editors do their readers a disservice when they use the authoritative words "Scientists say..." to preface anything from the latest news from the Higgs boson project to the tentative results from a computer model designed by a junior sociologist at the University of East Rutland.

As anyone from ex-weatherman Michael Fish to the ex-directors of Northern Rock will tell you, a degree of humility is required if one is to forecast future events. Depending on where you get your news, you may have heard that minimum pricing will save 900, 3,393, "more than 1,000" or "nearly 10,000" lives a year in England. If you watch *Panorama*, you will have heard that the policy will save 50,000 lives in a decade amongst the over-65 age group alone, unless you watch the re-edited

iPlayer version in which the figure has been dropped to 11,150 (this sizeable discrepancy was attributed to 'human error' (Snowdon, 2012)). All these figures can be traced back to the Sheffield Alcohol Policy Model (SAPM) which has been used since 2008 to make predictions about what effect a minimum unit price for alcohol will have on everything from NHS expenditure to unemployment.

The most recent version of the SAPM model, as pertaining to England, predicts that a 50p unit price will lead to a 6.7 per cent reduction in alcohol consumption, which will lead to 521 fewer deaths in the first year and 3,060 fewer deaths per annum by the end of the first decade (Purshouse, 2009). At the heart of these projections is the 'single distribution' model, a theory first advanced by the French demographer Sully Ledermann in 1956, which assumes there to be a fixed relationship between the amount of alcohol consumed in a society and the amount of alcohol-related harm. According to this theory, "the top X% of consumers always consume Y% of the total alcohol market. Therefore, to reduce the consumption by the top X%, total alcohol consumption has to be reduced." (Amber, 2009; p. 174) If true, the Ledermann hypothesis suggests that policies aimed at reducing alcohol consumption across the general population will affect heavy drinkers via a statistical *deus ex machina*, and so policies aimed at heavy drinkers themselves are not required. Lowering average consumption will be enough.

As John Duffy explains below, we should not be surprised to find a correlation between average consumption and alcohol-related harm because harmful drinkers consume a vastly disproportionate quantity of alcohol. In Britain, 40 per cent of all alcohol is consumed by 10 per cent of the population (Meier, 2009). The number of heavy drinkers in a society naturally has a profound impact on average consumption, but it does not follow from this that changes in average consumption produce concomitant changes in the number of heavy drinkers. Attributing high rates of harmful drinking to high per capita consumption is to put the cart before the horse.

But even if the Ledermann Hypothesis does not confuse cause with effect, the policy prescriptions it inspires do not necessarily make sense. Average consumption data cover a multitude of sins. There may be more harmful drinking in a country in which per capita consumption is 8 litres a year than in one in which the rate is 10 litres a year. Imagine, for example, if half the population of the former country is teetotal, but everybody drinks in the latter. A relatively low rate of per capita alcohol consumption is no guarantee of better health outcomes if the lion's share is being

drunk by a handful of alcoholics. There is little to be gained by making moderate drinkers reduce their consumption from, say, seven beers a week to four, and yet, by the logic of the Ledermann Hypothesis, such an intervention would significantly reduce rates of liver cirrhosis, drink-driving, domestic violence and so forth.

The Ledermann Hypothesis appeals to temperance and public health campaigners because it offers a relatively simple solution to the problems of heavy drinking and alcoholism. If the answer lies in reducing alcohol consumption at the population level, campaigners need only to lobby for higher prices and restricted availability which, *ceteris paribus*, would be expected to lead to less alcohol being consumed per head of population. The complex psychological and societal factors which lead to alcoholism and alcohol-related violence can thus be side-stepped, and the responsibility for these problems can be passed to politicians and the drinks industry. But although the single distribution theory has been treated as an iron law by many alcohol control advocates since the 1970s, empirical tests have produced mixed results and there is ample real world evidence running counter to it. From the outset, as Grant notes, the "mathematical basis for Ledermann's curve was weak" and "the rationale for the prevention theory inspired by it was poorly developed and, in fact, contrived" (Grant, 1997; p. 133). Ledermann offered little documentary evidence to support his theory and relied instead on "some rather peculiar statistical arguments of doubtful validity, to say the least." (Skog, 2006)

Moreover, the belief that reducing the affordability of alcohol will inevitably reduce both alcohol consumption and alcohol-related harm has frequently been confounded. Alcohol consumption has fallen in most of Europe and the USA (though not the UK) in recent decades despite rising incomes which have made alcohol more affordable.* When the Institute of Alcohol Studies compared alcohol-related disease rates and alcohol prices across European countries, it found "no discernible relationship between affordability and harm" (Institute of Alcohol

* "Average alcohol consumption has gradually fallen in many OECD countries between 1980 and 2009 with an average overall decrease of 9%. The United Kingdom however, has seen an increase of 9% over these three decades." (Office for National Statistics, *Statistics on Alcohol: England, 2012*, 2012; p. 23). It should be noted that greater affordability rarely means lower prices when it comes to alcohol. Although politicians, journalists and pressure groups often claim that alcohol has become cheaper in Britain, the Office for National Statistics has found that the real price of alcohol has increased by 24 per cent since 1980. During the same period, alcohol became 45 per cent more affordable. The greater affordability has therefore come about entirely as a result of higher average incomes in spite of above-inflation rises in alcohol duty.

Studies, n.d.). Dramatic reductions in the price of alcohol in Scandinavia in 2003-04 provided a natural experiment for price elasticity models to be put to the test, but the results were surprising. Denmark reduced the tax on spirits by 45 per cent in 2003 without experiencing any increase in alcohol consumption (Mäkelä, 2008; Grittner, 2009). Instead, there was a decline in alcohol-related problems (Bloomfield, 2010). Prior to the Danish tax cut, it was predicted that alcohol consumption would soar in southern Sweden because Swedes would cross the Danish border to buy cheap booze. Many did, and yet alcohol consumption in the south *fell* overall while consumption in the distant north rose (Gustafsson, 2010).

The story is the same when it comes to availability. Although temperance campaigners have long assumed that restrictive licensing laws reduce per capita consumption and therefore—by their logic—alcohol-related harm, real world evidence offers only equivocal support. France, for example, has seen a dramatic decline in alcohol consumption since the 1960s despite availability remaining unchanged (Romelsjö, 2010). In the UK, the implementation of so-called '24 hour drinking' laws coincided with the start of a prolonged downturn in per capita alcohol consumption. Since the Licensing Act was introduced in 2005, average weekly alcohol consumption has fallen by nearly twenty per cent (Office for National Statistics, 2012).

There are, of course, examples of higher prices and restrictive licensing laws reducing alcohol consumption and alcohol-related harm. Wartime restrictions such as the 1914 Defence of the Realm Act had significant and lasting effects on drinking and drunkenness in Britain. Similarly, falling prices can lead to higher rates of consumption. Although Denmark and Sweden were strangely immune from the great Scandinavian price drop of 2003-04, Finland saw a rise in consumption after taxes on spirits were reduced by 44 per cent, albeit on a much smaller scale than the price elasticity models predicted (Room, 2004; Mäkelä, 2009). It would be quite wrong to assume that pricing has *no* effect on alcohol consumption, only that the effects of price interventions are highly unpredictable and the relationship between per capita consumption and harmful consumption is questionable.

For a price rise to reduce alcohol-related *harm*, we must be sure that (a) reducing average consumption will reduce harm (the Ledermann Hypothesis), and (b) higher prices will reduce average consumption. Both criteria must be fulfilled, but since there is a good likelihood that at least one of them will not be fulfilled, any claim to predict how a price rise with affect health outcomes can only be speculation. This poses a problem for those charged with forecasting the consequences of minimum

pricing, and yet the inherent uncertainty has not prevented such forecasts flowing out of Sheffield University with a confidence that borders on hubris.

Many of the flaws in the SAPM model will be discussed in the following section. I shall offer just one example which any non-statistician (like myself) will readily comprehend. The model assumes that minimum pricing will have more effect on the consumption patterns of heavy drinkers than on moderate drinkers because heavy drinkers are more price-sensitive. This is a convenient belief since it is heavy drinkers who cause and suffer the most alcohol-related harm, but can we really assume that someone with an alcohol dependency is more likely to be deterred by price rises than a more casual consumer? The SAPM model says that they are, and yet there is ample evidence to support the common sense view that heavy drinkers and alcoholics are *less* price-sensitive than the general population (eg. Gallet, 2007; Wagenaar, 2009). Indeed, research has shown that price elasticity for the heaviest drinkers is "not significantly different from zero"—they will, in other words, purchase alcohol at almost any cost (Purshouse, 2009; p. 76).

Heavy drinkers are more price-sensitive, but not in a way that is relevant to the minimum pricing debate. Research has shown that if the price of wine (for example) increases, heavy drinkers are more likely to stop buying it—or to buy less—than moderate consumers. This is not because they cut down their alcohol intake, but because they switch to cheaper drinks, such as whisky. And if the price of whisky increases, they are more likely to shift to vodka than is a whisky connoisseur. However, none of this is germane to the issue of minimum pricing because the minimum price will raise the cost of every type of drink. As economist Eric Crampton notes, a price elasticity estimate for a particular *type* of alcohol product "tells us *zilch* about what harmful drinkers do in response to a price increase for the entire product category; it would be misleading to use this kind of data to claim that harmful drinkers are the most price responsive. They're *most* price responsive when their preferred brand or product changes in price but they're also *least* responsive to aggregate changes in alcohol prices." (Crampton, 2012; italics in the original.) By wrongly assuming that heavy drinkers are more sensitive than the general population to changes in the price of alcohol *as a product category*, the Sheffield model not only overestimates the putative health benefits to be derived from minimum pricing, but also overestimates the drop in overall consumption that is likely to take place (since heavier drinkers consume a disproportionate quantity of alcohol). Moreover, it underestimates how much poorer heavier drinkers will be as a result.

Upon such faulty assumptions is the SAPM based. Mention should also be made of what is *not* in the model. It does not, for example, factor in the possibility that raising the floor price of spirits will fuel the market for illegally produced alcohol; counterfeit alcohol is already a growing problem in the UK and unregulated spirits pose a greater risk to health than the legitimate product. Nor does the model take account of the effects of secondary poverty created by people on low incomes spending a greater share of their wages on drink. Minimum pricing could indirectly damage the health of drinkers who make cuts in other parts of the family budget, such as food and fuel. Also conspicuous by its absence in the SAPM is any acknowledgement of the mountain of evidence showing alcohol to have medicinal benefits and that teetotallers have, on average, shorter lifespans and higher rates of cardiovascular disease than moderate drinkers (eg. Doll, 1997; Rimm, 2007; Holahan, 2010, Ronksley, 2011).* Is it possible that minimum pricing will turn some moderate drinkers into teetotallers? If so, what are the health implications of this? One searches the Sheffield research in vain for an answer.

In summary, the Sheffield research does not give a prediction of what will happen under a minimum pricing regime. At best, it offers a shaky guesstimate of what *might* occur under a minimum pricing regime *if* the Ledermann hypothesis is correct, *if* harmful drinkers are more price-sensitive than moderate drinkers, *if* there is no illicit alcohol market and *if* there are no health benefits to be derived from moderate alcohol consumption. Since the first two of these assumptions are highly questionable and the latter two assumptions are demonstrably false, the Sheffield research has no practical merit and does not deserve the exalted status it has been afforded in the policy debate.

In the era of evidence-based policy, it seems that speculative statistics are considered superior to no statistics and a wrong answer is better than no answer. We argue that this is a mistake. The aura of scientific certainty, or even mild confidence, in computer-generated numbers based on dubious assumptions is misplaced. Minimum pricing might reduce alcohol harm, or it might increase it, or it might bring about other unexpected consequences, good or bad (Donald Rumsfeld's 'unknown unknowns'). There is no shame in saying that we simply do not know. An admission that the evidence base is, to all intents and purposes, non-existent is less likely to mislead decision-makers than a spurious prediction.

* The kernel of truth in the Ledermann Hypothesis is that if people never start drinking, they cannot become harmful drinkers or alcoholics. However, few hope or expect to see a teetotal society and taxes on alcohol are not intended to bring about total abstinence.

There is one further reason why the Sheffield research should not be seen as a prediction. In a real sense, we know what would happen to alcohol-related mortality if the SAPM's assumptions are correct. The data used in the model to calculate alcohol consumption come from 2006 and, as already noted, alcohol consumption has fallen by close to twenty percent since then. All the projections in the SAPM are based on a 50p minimum price reducing per capita alcohol consumption by 6.7 percent *from the 2006 level*. But we know exactly what would happen if alcohol consumption fell by 6.7 per cent from the 2006 level because we have lived through it. Indeed, the decline in per capita consumption since that year has been closer to what the SAPM predicts would happen under a 70p per unit regime (ie. a 17.5 per cent decline). According to the model, the kind of reduction in alcohol consumption that Britain *has already experienced* should have reduced the number of alcohol-related deaths by 1,273 (28.3 per cent) in the first year, rising every year until 7,263 deaths (62.4 per cent) are prevented each year by 2015 (Purshouse, 2009; pp. 109-111). None of this has happened. We are thus in the unusual position of being able to empirically disprove a prediction about a policy which has not yet been introduced.

# The Sheffield Alcohol Policy Model

by John C. Duffy

For about 25 years since 1970 I worked as a research statistician in psychiatric epidemiology. In the mid-1970s I became involved in alcohol research and have remained active in that field on and off ever since. When I started, the 'control of consumption' or 'single distribution model' was being advanced by researchers in many countries, mostly those with alcohol control systems such as the Nordic countries, Canada and some states of the USA.

The idea was simple—average consumption correlated with rates of alcohol-related problems, so if average consumption could be controlled, the problems would be reduced in proportion to the reduction in per capita consumption. There is a tautology in this relationship which has never been properly addressed. If one area has more alcohol-related problems than another then it probably (almost certainly) has more people drinking heavily. Therefore it will have a higher average consumption. Seen this way, average consumption is a product of the drinking culture, and not a determining variable. Strenuous efforts were made to ignore this aspect, or to pretend that statistical models offered a way round the problem, and I made myself fairly unpopular by pointing out this fundamental flaw.

For the most part, the research community continues to insist that increased availability of alcohol leads to more drinking, which leads to more problems. Symmetry, however, implies that it works the other way around and despite the prophets of doom, experience in the UK with increases in availability (albeit limited), usually in terms of licensing and hours of sale did not have the effects predicted by the 'researchers', who were quick to develop *ad hoc* arguments to explain why not. My first experience of this was with my analysis of Scottish licensing liberalisation back in the 1980s. When a before and after comparison of Scottish liberalisation in 1976 (using England & Wales as a control) failed to show that increased availability led to increased consumption and problems (Duffy, 1986) the work was criticized on the grounds that Scotland had been adversely affected by economic recession (Eagles, 1986; Prichard, 1986). However, examination of unemployment rates and disposable income per capita showed no divergent trends between Scotland and England & Wales, so this 'explanation' or rescue attempt for the availability hypothesis failed (Duffy, 1992). Even if the availability hypothesis were true it does not follow that symmetry would apply—that is that decreased availability must lead to decreased consumption and problems.

While discussing this I think it is worth pointing out that while availability may in some instances be a correlate of consumption it is most definitely not a cause. It is a condition—in that if there is no availability there can be no consumption. This is not just a pedantic distinction, as it goes some way towards explaining why availability-based models always fail at some point or another and have to be rescued *ad hoc*.

Alcohol epidemiology suffers from a number of disadvantages. Most obviously, the survey method which is a basic tool of epidemiology isn't very good for alcohol. We know that individuals under-report their consumption in surveys, which is a problem when we are trying to estimate the risk of some health or social outcome as a function of individual consumption. We also know that many individuals drink different amounts in different time periods. This is a problem because survey methods that try to be very accurate about recording amounts consumed have to be based on a relatively short period of time in order to (presumably) elicit more accurate information. Because of the variability in drinking behaviour, the shorter the time period, the more extreme behaviour (drinking a lot or drinking nothing or very little) is over-represented. So a survey doesn't provide an accurate description of an individual's long-term consumption habits. Per capita alcohol consumption figures based on the amount sold divided by the population, which are available for many countries, are in principle reasonable estimates of average consumption but

do not help in epidemiological studies which require individual level data.

In terms of alcohol-related harm, there are two types of harm that most researchers distinguish—harm related to high per-occasion consumption (e.g. accidents, crime, etc.) and harm related to longer-term consumption (liver damage for example). Most of the relationships that can be investigated with any degree of accuracy involve the second type of harm, but even here relationships are poorly estimated and imprecise because many of the studies used as source information are not mainly interested in alcohol-related harm, and measure alcohol consumption very crudely.

So I was surprised when I began to read in the press that, for example, banning supermarkets and shops from selling drinks for less than any duty and VAT owed on them would 'only save 21 lives a year, according to an alcohol expert' (Whitehead, 2011). Why 21? Why not 20? Or none? And anyway, how could you tell? In England & Wales the annual number of deaths is just under half a million, so the 95% error bars (for random variation in the number of deaths) are about 1,400 either side. So even if the expert's prediction was correct it could never be demonstrated.

Similarly in Scotland the minister responsible, Nicola Sturgeon claimed that a minimum price of 50p per unit would save 60 lives a year (Scottish Government, 2012). Again, though, the standard error of the number of deaths per year in Scotland is considerably more than 60—and a prediction that cannot be tested is no prediction at all. Another way of expressing this is that the predicted effect is not statistically significantly different from zero and may actually be zero.

Looking more closely at the argument for minimum pricing and the associated predictions, it becomes clear that the forecasts are based on existing data, heroic assumptions and computer number-crunching in something called the Sheffield Alcohol Policy Model (SAPM). The results of the number-crunching are quoted without estimates of error, and in particular the model completely ignores statistical error in the alcohol-harm relationship. Far from being evidence based it would be more accurate to describe these predictions as assumption based.

As my background is in statistics and epidemiology I don't propose to comment in detail on the economic aspect of the model, other than to point out that the calculations are based on elasticities and cross-elasticities which are local linear quantities (the derivative of the demand curve)—that is they apply to small changes,

not necessarily to large ones. These quantities are not eternal constants but change from time to time and from place to place. This makes the wholesale use of economic data from other time periods or other geographical areas inappropriate and the conclusions suspect. An example is the use of economic data for England in the Scottish version of the model, and in general where appropriate data (in terms of geography and time period) are not available the model is just run with whatever data the modellers can find. Since elasticity is an average phenomenon, there may be differences in elasticity between different subgroups. For example, if heavy drinkers are less price-sensitive than the rest of us then even the most basic model predictions will fail.

On the epidemiology side, it turns out that the risk relationships between consumption and harm used in the model are in general not known for the populations to which the model purports to predict. Most studies attempting to quantify risk and attributable risk of particular adverse health outcomes involve recording exposure to risk factors either in a healthy population followed over time for development of the health outcome (a cohort study), or in a sample of cases of the health outcome and a control sample (a case-control study), but for the vast majority of the outcomes considered there are no such studies. One way the SAPM deals with this for some conditions is to use estimates for other populations obtained 'from the literature' (and there is really no reason to believe these will apply to different populations). When there are no such estimates the model is based on what amounts to little more than numerology—making an assumption about the form of the risk, using survey data for consumption and then computing a risk function. There is no reason to think that the assumed form of any of these risk functions (for they are different for different conditions) is correct. So there is no reason to believe that the predictions of the model are correct.

The Sheffield team's approach to problems related to single instances of high consumption is even less convincing. A statistical model is used to estimate peak consumption from average consumption, for use in analyses relating adverse results of individual episodes of heavy drinking. From the available description of this model it appears to be a poor fit to such data as there is, and, as noted earlier, the drinking pattern in one week (or similar short time period) is not necessarily representative of longer periods. I am not going to go through the details, but it adds yet another imponderable into the mix.

"All models are wrong but some are useful" is a famous quotation in statistics attributed to George E.P. Box. So even if the SAPM is wrong it might still be useful. Certainly it is useful to those who want to lobby for control of alcohol consumption, but how useful is it as what it purports to do—predicting the impacts of prices on consumption and harm?

This is an empirical question, and since the model has been around for a good few years now, one might have expected the accretion of new data on price, consumption and harm to have led to testing of the model. So far the authors have preferred not to do this, instead they 'update' the model to fit the new data.

It's rather like a racing tipster who claims to use an evidence-based weighted sum of form variables to predict that Likely Lad will win the 3.30 at Uttoxeter. When in fact Lively Lady wins, he tells you that he's changed the weights (again, 'evidence-based') so that he would have predicted Lively Lady to win. He wants to sell you a tip for the next race. I wouldn't buy it.

A strange aspect of the model publications is that they provide reams of data in appendices to show some of the model inputs and outputs, but oddly not enough information for a third party to rerun bits of the model. Whatever the reason for this, it also means that third parties can't check if the model fits the changes that have occurred. Furthermore, the anomalous pattern of risk functions, and changes in these, which are noted in the following stats section pass without comment in the Sheffield publications. This and the supply of implausible estimates to *Panorama* suggest a lack of critical consideration of the model and its components by the Sheffield group.

The SAPM is certainly complex and takes many factors into account. However it is also full of unknowns, as it isn't known how these factors actually relate to consumption and price, so dubious assumptions are used to construct the relationship. It seems unlikely that many people have read the whole description of the model critically, being content to be impressed by its apparent complexity and comprehensiveness.

Of course, a supporter of the model will ask me 'If you're so smart, what's your model – what do you predict?' My answer is that I don't have a model and therefore I won't make a prediction. There is not enough information around to produce a reliable model and I won't invent one that is engineered (by undemonstrated assumptions)

to fit the prevailing facts and pretend that it is of any use for prediction. As Taleb says in *The Black Swan* about those who attempt to justify worthless predictions because 'that's their job'—get another job.

# Appendix

This appendix lists some of the contentious issues related to application of the SAPM to Scotland in the publication *Model-based appraisal of alcohol minimum pricing and off-licensed trade discount bans in Scotland using the Sheffield alcohol policy model (v 2): Second update based on newly available data* (January 2012). Page and section numbers refer to that document.

**Economic/consumption data**

On-trade price data are not available for Scotland (p. 16). There is no reason to believe that 'adjustment' of the data for England will adequately represent the position in Scotland.

Price elasticities are not available for Scotland, so values for England are used (p. 99). Again there is no reason to expect these to be applicable to Scotland.

Various surveys are used to provide inputs to the model (Sections 2.1 and 2.2). The following aspects relating to validity of the survey data are in general ignored:

- Sampling variability (chance variation due to the individuals selected for sampling)

- Non-response—the response rate in the Living Costs and Food Survey is 53% (which may also lead to bias if, say, particular types of consumers are less likely to respond)

- Response bias (deliberate misreporting)

- Bias in estimated distributions arising from the length of the time period covered—the diary in the Living Costs and Food Survey covers two weeks, and it is known that where there is temporal variability (ie. respondents don't drink exactly the same amount each week) use of a short survey period leads to over-estimation of the proportion of low and high consumers and corresponding under-estimation of consumers in the middle of the range

Section 2.2.2 gives a brief description of the process used to estimate peak daily consumption from average consumption. This measure is used later, itself as a proxy for heavy episodic drinking, in analyses relating adverse results of individual episodes of heavy drinking. The regression models used require empirical verification, and the drinking pattern in one week (or similar short time period) is not necessarily representative of longer periods. The low values of $R^2$ obtained indicate that the regression model is not a good fit. Predictive $R^2$ which assesses the fit of a regression model in terms of predicting the y-value would be even lower, and it cannot be concluded that this aspect of the model is reliable.

**Health risks and other forms of harm**

Section 2.3 deals with the relationship between consumption and various forms of adverse health consequences. There are no direct estimates of the relationships between individual drinking habits and consequences in the population covered by the report. While there are summary statistics on harmful health outcomes 'wholly or partially attributable to alcohol' and estimated statistics on amounts drunk, the risk functions relating different amounts of consumption to different degrees of harm are not based on direct data where an individual's consumption and his/her health outcome is known. For some outcomes no relevant studies have ever been published in the literature, and as a result this crucial aspect of the model is based entirely upon assumptions.

Use of risk functions 'from the literature' (p. 33) for partially attributable chronic conditions implies an assumption that average risk functions derived from a range of

studies in various countries may appropriately be applied to the Scottish population. While the epidemiological evidence is in general strong for alcohol increasing the risk of the conditions considered, there is no evidence that the risk function values apply to the Scottish population. In fact there is strong evidence that risk functions relating alcohol to harm may differ between populations (heterogeneity in the jargon of meta-analysis), and there are obvious reasons why this should be the case. Patterns of drinking, beverage choice, varying levels of association with other risk factors and genetic aspects of the population studied are just a few reasons why apparent risk relationships might show statistically significant and meaningful differences between populations.

The methods used to estimate the risk functions between alcohol consumption and other forms of harm—crime and 'workplace harms' (sections 2.5 and 2.6) again do not relate to associations observed at the individual level, but are based on various forms of attribution including self-report attribution (which may of course be incorrect) and then modelling these in much the same way as for the acute conditions, but using peak consumption rather than mean consumption. There is thus no direct statistical evidence to support these estimates.

As an example of the problems of assuming the form of a risk function and then estimating its parameters from the attributable total, consider the estimated slope of the risk functions for mental and behavioural disorders due to alcohol. The estimates for mortality are in Table A4.1 and for morbidity in Table A4.2, and in Tables A5.1 and A5.2 of the previous version of the model (Purshouse *et al.*, 2009). The estimates do not appear to follow common sense patterns in relation to sex and age differences and trends over time. For example, for age 45-54 the mortality slope for females is about 30% greater than that for males, while for age 55-64 the mortality slope for females is 25% less than that for males. Again, in age group 45-54 the morbidity slope for females is nearly 40% less than that for males. And if we compare the 2009 and 2012 estimates we see that the mortality slope for males aged 45-54 decreased by 30%, while the mortality slope for females of this age increased by 30%.

Figure 3.10 illustrates the inadequacy of the modelling approach to attribution of criminal behaviour to alcohol consumption, the lines showing gross differences between the 'results' using different basic assumptions. Predictions cannot seriously be sustained when differences of factors of five result from different assumptions required to be made due to the lack of direct evidence.

Section 2.6 describes sensitivity analyses and adjustments of survey data. The sensitivity analyses should not be confused with allowing for sampling and other sources of statistical error. The basic assumptions that underpin the approach are not questioned in these analyses.

**Limitations – admitted and not admitted**

The authors of the report present a number of limitations in section 4.2. In the section on consumption they fail to mention sampling variation, variability of individual consumption over time and response issues related to the surveys used. They do not mention the assumptions required in constructing the relationship between the price distribution and the consumption values of the respondents to the surveys.

In the section on elasticities they acknowledge that there is no detailed longitudinal study of purchase and consumption of alcohol, and that the methodology may be inadequate (p. 99).

In section 4.2.2 they acknowledge considerable uncertainty regarding the relationship between alcohol and crime, but do not mention the difficulties associated with quantifying risk functions for the Scottish population for other consequences.

The above is not an exhaustive list of the difficulties associated with the SAPM, but demonstrate that the results of the model reflect speculation (or assumptions) made regarding the model components. No degree of statistical confidence can be placed in the values produced. The models relating health consequences to consumption cannot be considered reliable as they are based almost entirely on assumptions with no direct individual level data relating consumption and consequences. They are not fit for the purpose of estimating rates of consequences, far less changes in these following policy changes.

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