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THE FLAWED THEORY BEHIND ALCOHOL CONTROL POLICIES

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About the authors

John C. Duffy was a non-clinical scientist in the Medical Research Council Unit for Epidemiological Studies in Psychiatry between 1971 to 1990 and a senior lecturer in Statistics at the University of Edinburgh. After the closure of the MRC Unit, he was appointed a director in the Alcohol Research Group in the Department of Psychiatry supported by a grant from the Portman Group to the University of Edinburgh. In 1996 he was seconded to the Scottish Office Department of Health and from 1999 to 2001 was director of the statistical consultancy service of the University of Edinburgh. From 2001 to 2003 he was Head of Statistics in the Department of Primary care, University of Birmingham, and from 2003 until his retirement in 2012 was a deputy director at the Scottish Funding Council.

Duffy's work on alcohol dates from the mid 1970s and he has always been critical of the theories around the distribution of alcohol consumption. From 1994 to the present he has been the subject of ad hominem attacks from alcohol researchers on the grounds that he has acted as a consultant to the alcohol beverage industry, and the Alcohol Research Group was funded by the Portman Group. The first example was related to the publication of 'Alcohol Policy and the Public Good' edited by Griffith Edwards and Peter Anderson. The most recent criticism appeared in the *British Medical Journal* earlier this year, paid for by a grant from the European Union under the FP7 research program to support an alcohol project called ALICE-RAP – headed by Peter Anderson.

Christopher Snowdon is an author, journalist and researcher who focuses on lifestyle freedoms, prohibition and dodgy statistics. He is the Director of Lifestyle Economics at the Institute of Economic Affairs and regularly appears on TV and radio discussing social and economic issues. He wrote *Velvet Glove, Iron Fist: A History of Anti-Smoking* (2009) and *The Spirit Level Delusion* (2010). His most recent book is *The Art of Suppression: Pleasure, Panic and Prohibition since 1800* (2011) which looks at the prohibition of alcohol, drugs and tobacco. Born in North Yorkshire, he now lives with his wife and daughter in Sussex.

Summary

Alcohol policy in Britain and many other countries aims to reduce per capita alcohol consumption in the belief that this will inevitably reduce heavy and harmful drinking. The cornerstone policies of this approach are advertising bans, licensing restrictions and higher taxes.

Campaigners cite the 'Total Consumption Model' as justification for implementing policies that affect all drinkers, rather than just the heavy drinking minority. The theory was devised in the 1950s based on a statistical correlation between average alcohol consumption and rates of harmful drinking.

As researchers have long recognised, this theory is deeply flawed and has little predictive power. Per capita alcohol consumption largely depends on the amount of heavy drinking in a population, not vice versa. The mathematical model is simply wrong. Numerous real world examples, including the UK in recent years, show that alcohol-related harm does not necessarily correlate with overall alcohol consumption.

Empirical evidence supports neither the Total Consumption Model nor the policies upon which it is based. These policies bear costs on moderate drinkers while being largely ignored by at-risk drinkers.

Alcohol policy would be more effective and equitable if it targeted excessive drinkers, alcoholics and those who require help, rather than the whole population.

Drinking patterns and alcohol-related harm

Health campaigners in the alcohol field argue that per capita consumption of alcohol in a population is the determining factor in levels of alcohol-related harm and that governments should therefore introduce policies to control or reduce *average* consumption in order to reduce *problematic* consumption. This is known as the Total Consumption Model or the Single Distribution Theory. Colloquially, it might be described as 'punishing the majority for the sins of the minority' since the policies it inspires - tax rises, licensing restrictions and marketing bans - have significant general welfare costs.[1]

The argument traditionally takes the form that individuals consuming large amounts of alcohol experience high levels of alcohol-related harm and that the proportion of these heavy drinkers in a population is positively correlated with average consumption. From this, it is assumed that reducing average consumption will reduce heavy drinking and that 'we need alcohol policies for the whole population. If we all drink less, then harms will come down across the board' (Alcohol Focus Scotland, n.d.). Together with the equally spurious claim that average consumption is driven by availability and advertising - defined to accord with the policy being criticised or advanced, from licensing liberalisation (they disapprove) to price increases and advertising bans (they approve) - this constitutes the 'scientific' basis for the prohibitionist paradigm.

1 These include the deadweight costs of taxation, the welfare cost of being unable to drink at chosen times and search costs incurred by limitations on advertising.

When one of us (JD) first became interested in this area in the 1970s while working for the Medical Research Council, on a routine visit to my GP I mentioned that I was working on the relationship between average alcohol consumption and alcohol-related problems, he said, 'Well, *of course* average consumption is correlated with problems, as it's the people with problems who push up the average.' That common-sense remark was and remains my view, but it has never been popular with researchers.

It is well known that a relatively small number of drinkers consume a disproportionately large proportion of alcohol. In Britain, more than 40 per cent of alcohol is consumed by ten per cent of the population. Close to 70 per cent is consumed by one fifth of the population. This distribution is not unusual in markets - the Pareto principle gives the rule of thumb that 80 per cent of sales come from 20 per cent of customers - but it indicates the extent to which per capita consumption depends on the drinking patterns of a minority. It is amongst this minority that most of the health problems associated with excessive drinking occur.

It is no surprise that there are many examples of alcohol-related health problems rising and falling in line with per capita consumption. Often-cited examples include the USA during Prohibition and France during the Second World War when rationing was in place. In both instances, rates of liver cirrhosis fell very quickly after the respective policies were implemented. The association between overall consumption and heavy drinking in such examples is real, *but it is tautological rather than causative*. Or, more precisely, the causation runs in the opposite direction to that assumed by many public health campaigners.

In these examples, per capita consumption was bound to fall when the government made it more difficult for heavy drinkers to acquire the amount of alcohol to which they were accustomed. The question today is whether the same effect takes place in less extreme circumstances when government policy explicitly aims to reduce overall (per capita) consumption as a means of reducing excessive drinking. If it is predominantly moderate drinkers who contribute to

the decline in alcohol consumption, there is no reason to expect a fall in diseases such as liver cirrhosis. Evidence from England and Wales in the last decade demonstrates that a significant decline in alcohol consumption does not necessarily lead to a commensurate improvement in associated health outcomes if it is the ‘wrong’ people who are reducing their consumption.

The rise of the total consumption model

In the 1970s, the Total Consumption Model was being pushed in the literature by workers at the Addiction Research Foundation (ARF) in Toronto, quite conveniently for policymakers as the Ontario Government was committed to alcohol control. The ARF had been founded to continue the research work of the Liquor Control Board of Ontario. Their work was based on a statistical theory developed by the French researcher Sully Ledermann in the 1950s (Ledermann, 1956), and widely (and we would argue, wisely) ignored until the ARF work. When alcohol research became fashionable in the UK (in the early to mid 1970s) it was perhaps natural that the Canadian work should become influential as it was written in English. Among the usual reporting of correlations between consumption and various types of harm, notably liver cirrhosis, the Canadian researchers tabulated 'proportions of excessive drinkers' corresponding to various levels of average consumption (de Lint & Schmidt, 1970).

Ledermann theorised that the distribution of alcohol consumption in a population (that is the classification of the members of the population by their amounts consumed – rather like a *smooth histogram*[2]) was of a particular mathematical form. The graph below shows a histogram of simulated data for a sample of 2000 drinkers. If the reader visualises a smooth curve through the tops of the bars,

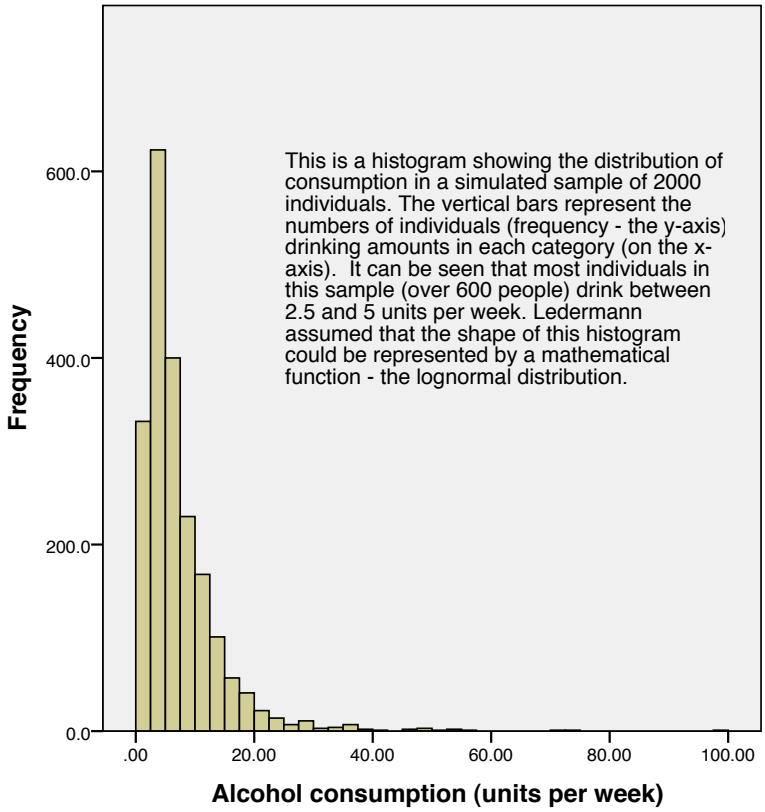
2 A graphical display of data using bars of heights representing the number of observations in the ranges to which the bars relate.

it looks like what is known in statistics as the lognormal distribution, which is the form Ledermann assumed for the distribution of alcohol consumption. In a way, the lognormal distribution is a particularly simple form to describe a variable which cannot have values less than zero, and where there are some (but relatively few) very large values. The relationship between the well-known normal distribution (the 'bell curve') and lognormal distributions is simple – the lognormal is the distribution of a variable whose logarithm is normally distributed.

Ledermann further assumed that the entire distribution could be determined by the average consumption, using a further relationship (mathematically speaking, a constraint) that requires the proportion of drinkers consuming more than one litre of alcohol per day (in beverage equivalent) to be constant in all populations. In terms of this graph, this constraint is equivalent to assuming that the area under the curve from the point on the x-axis corresponding to one litre per day to infinity is always the same, so although the curve will differ for different average consumption values, that tiny bit of area will always be the same.

The distributional assumption was based on the general finding that the empirical distribution of consumption was *positively skew*[3] (not particularly surprising, as distributions where all values are greater than zero and there are instances of extremely large values usually are positively skew – variously power law, Pareto, Zipf etc), and the lognormal fit was generally adequate to the centre of the distribution. However it is impossible to understand why Ledermann thought that the proportion of consumers drinking more than the beverage equivalent of a litre of absolute alcohol per day should be a universal constant, or why the distribution should apply at all at extremely high values.

3 Asymmetrical, with a long tail to the right.



By the mid 1970s, one Nordic researcher (Skog, 1972) in particular realised that the constraint was ludicrous (although he didn't use that word), but failed to recognise the much more basic problem that the idea that the amounts people drink should always follow a particular mathematical distribution is equally untenable. Nor has it been generally recognised that the distribution of consumption (if one is interested in it) requires to be defined in time, and that distributions covering short periods of time, as in surveys, cannot just be scaled up to estimate distributions over longer periods unless everyone's drinking habits are completely regular and show no temporal variation.

In statistical analysis it is often assumed that a particular quantity follows a particular distribution, and it is known that in many cases such as estimating a *mean*[4] or testing a statistical hypothesis the assumption does not lead to undue error. That is, it is a mathematical approximation to the actual distribution, for convenience of analysis. Such a distributional assumption is not generally good enough to estimate the proportion of a population in the extreme of the distribution with any accuracy. And of course consumption in different populations would have varying degrees of fit to any postulated distribution, so there isn't and never was a 'single distribution of alcohol consumption'.

Since the last point was not then appreciated, researchers in the Nordic countries argued that the dispersion of the distribution of consumption, a measure of the variability in the population, was constant (a new constraint), despite empirical evidence (even presented in a graph by them and misinterpreted) that it clearly was not (Bruun et al, 1975).

4 The arithmetic average.

The fall of the total consumption model

The popularisation of the Total Consumption Model owes much to the involvement of the anti-establishment Finnish sociologist Kettil Bruun who reacted against the post-prohibition model of alcohol control in the 1970s by proposing interventions targeted at the whole of society, rather than alcoholics specifically. Between 1919 and 1932, Finland had American-style alcohol prohibition and the anti-alcohol measures that were pursued after repeal were largely targeted at problem drinkers. Alcoholics were forcibly sent to asylums and prisons in their thousands and laws against public drunkenness were strictly enforced (Sulkunen and Warsell, 2012).

Bruun saw these 'individual interventions' as a form of social control which breached the alcoholic's civil rights. He wanted to replace them with 'environmental strategies' which replaced individual responsibly with collective responsibility (Tigerstedt, 1999). The Ledermann hypothesis had obvious appeal as a theoretical justification for this approach. It also suited the times. A new public health movement movement was emerging in the 1970s which tended to blame governments and institutions, rather than individuals, for personal health problems and took the position that 'Dangers are everywhere, and they concern all; they are external to and outside the control of the individual' (ibid.: 211). Bruun's policy proposals for greater state control over the price and availability of alcohol were aimed at removing stigma from individuals while reducing per capita consumption. Although clearly less draconian

than prohibition, his approach was less liberal than that practised in most countries outside of Scandinavia, but has spread far beyond the Nordic countries in the decades since. It has become the orthodox public health position.

However, by the late 1970s, the assumption of a single distribution was beginning to be questioned. In the European home of alcohol control (Scandinavia) Ledermann's theory had already been found to be deficient, but as the research was published in Norwegian and Finnish it took some time before this was recognised in the UK. An argument based on collective adjustment (Skog 1985) formed the next attempt to rescue the single distribution theory and the primacy of average consumption by Nordic researchers. The original article developed the theory on the basis of verbal arguments, with no mathematics, very little empirical data, and an 'Alice in Wonderland' use of words to mean what the author wanted them to mean. '...the whole population move (sic) upwards along the consumption scale when the mean increases'. Obviously the population does not 'move' at all, but what is presumably meant is that changes in consumption are in the same direction for everyone. If this were true then it would not be possible for an individual to decrease their consumption during a period of increasing consumption. It hardly needs to be pointed out that this is contradicted by everyday experience.

The single distribution theory assumes that the population is homogeneous in a statistical sense. This is contradicted by countless studies which show particular subgroups drinking at quite different levels, and changing their consumption levels in different directions at the same time. Similarly the link between consumption and harm is different in different subgroups, with wealthier members of populations drinking more but suffering fewer consequences than more deprived subgroups.

As Timo Alanko, a Finnish statistician, wrote: 'why and how would the individuals in a society collectively adjust their consumption so as to exactly imitate fairly simple mathematical formulae?' (Alanko, 1997). To illustrate the absurdity of the hypothesis, consider the

case of obesity. It is self-evident that average body weight in a population will rise as the number of obese people rises. The only way this outcome could be avoided would be if non-obese people lost weight at the same rate as obese people gained it, a highly unlikely scenario. Similarly, it is tautologically true to say that a rise in the number of people who consume high levels of salt can be expected, *ceteris paribus*, to result in a rise in per capita salt consumption. But in neither case would we expect to see a decline in the number of 'deviants' (to use the slightly derogatory technical term) from a decline in body mass and salt consumption amongst moderate consumers.

Almost incredibly, both the examples above were put forward as serious propositions by the epidemiologist Geoffrey Rose as an extension of the Ledermann hypothesis. Rose observed that the prevalence of obesity, heavy drinking and high sodium intake correlated closely with average body weight, average alcohol consumption and average sodium intake respectively. He concluded that these correlations had 'profound implications' for policy and claimed that the 'close link between mean and prevalence implies that to help the minority the "normal" majority must change' (Rose and Day, 1990: 1034). Society had, he wrote, a 'collective responsibility' to reduce harmless consumption at the middle of the distribution curve in order to prevent harmful consumption at the tail. He dismissed concerns about *false* correlation by pointing to the strength of the statistical associations but ignored the real problem of *reverse* causation. Elsewhere, he expanded his theory further by speculating that many traffic accidents were the result of very large numbers of people drinking very small amounts of alcohol - which, as research has shown, 'is decidedly not the case' (Harper, 2009: 1744). Moreover, the rise in obesity in the USA is far larger than would have been predicted based on the change in average body weight (ibid.). Put simply, 'deviancy' is not mathematically fixed to 'normalcy'. Averages do not cause extremes, but extremes have a profound effect on averages.

The plain fact is that the alcohol and harm relationship in the UK and elsewhere does not 'work'. Poorer people drink less, but

experience higher rates of 'alcohol-related harm'. Cross-sectional studies (essentially correlational) between liver cirrhosis rates and average consumption do not invariably show the expected positive relationship (e.g. Ramstedt 2001) – and there is no doubt that liver cirrhosis really is caused by heavy drinking. Overall consumption can be a *indicator* of heavy drinking but it is not, and cannot be, the *cause* of heavy drinking. It is true that successful attempts to reduce heavy drinking are very likely to reduce overall consumption, but attempts to reduce overall consumption will not necessarily reduce heavy drinking.

Correlations between average consumption and harm are clearly affected by the tautological issue referred to earlier – that is a high proportion of heavy consumers leads to both high average consumption and high levels of harm. They have another inbuilt problem, generally unrecognised - spurious correlation - identified by Karl Pearson (1897). Both the average consumption and the rates of illness or harm are based on quantities, each pair of which are divided by the same number (the population size, which of course varies from population to population and over time) and this division calculation itself is sufficient to produce positive correlations between unrelated quantities. An interesting discussion of this fallacy (including discussion and analysis of the correlation between stork populations and human birth rates) is given in Kronmal (1993).

Confirmation bias and seeing what one wants to see are persistent features of the analyses adduced as evidence in this area. Two papers published in the 1990s (Rose and Day, 1990 and Colhoun et al., 1997) attempted to deal with the tautology problem by correlating the proportion of heavy drinkers in population samples with the average consumption of everyone else excluding the heavy drinkers. One paper was actually titled 'The population mean predicts the number of deviant individuals'.[5] When analysing the data as described above, that is correlating the mean excluding the heavy drinkers with the proportion of heavy drinkers, in both papers the

5 Another researcher begged the question by titling his paper 'The impact of per capita consumption on Swedish cirrhosis mortality'. The clear implication was that cirrhosis is caused by per capita consumption rather than by heavy drinking (Norström, 1987).

authors naturally found a correlation much smaller than the correlation between the mean of the whole population and the proportion of heavy drinkers. But they did not demonstrate prediction, they merely calculated a correlation. To validate their predictions they could have used subsequent data from the same areas, or contemporary data from other areas. Another approach using the same data reported in the work would have been to use cross-validation and calculate a statistical quantity called PRESS which would have given an idea of how well or badly the prediction works. They also failed to notice the possibility of spurious correlation induced by the proportion of heavy drinkers forming part of the denominator of the amount consumed by the rest of the population.

‘Paradoxes’

Campaigners point to rough correlations between alcohol consumption and liver cirrhosis as supporting evidence for the Total Consumption Model. These correlations are often very rough indeed. Between 1980 and 2000, alcohol consumption rose only slightly in the UK (from 9.6 to 10.4 litres per person) while liver cirrhosis mortality nearly doubled (from 4.6 to 9.7 per 100,000). In Sweden, liver cirrhosis more than halved while alcohol consumption fell by only 15 per cent, and in the Netherlands there was a fall in consumption but no change in liver cirrhosis. In Norway and Ireland, meanwhile, rates of liver cirrhosis fell while per capita consumption rose (Bentzen and Smith, 2011).

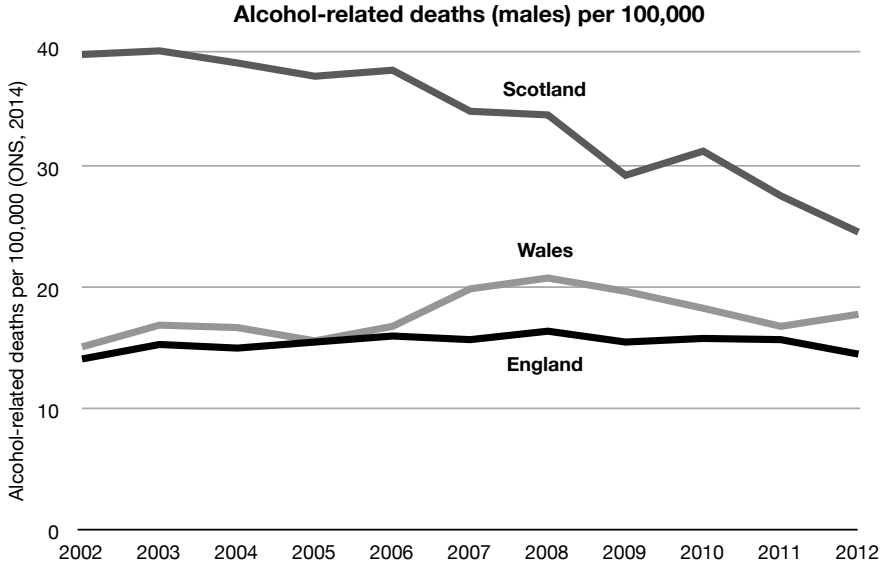
The association between per capita consumption and alcohol-related harm is therefore far from being an iron law; it depends on which drinkers are increasing or reducing their intake. Since the Ledermann hypothesis is based on statistical correlations between per capita consumption and alcohol-related harm, exceptions to the ‘rule’ are viewed by its proponents as ‘paradoxes’. The US Centers for Disease Control noted in 1986 that ‘The reason for the decline in cirrhosis mortality since 1973 is not clear - especially since deaths from alcoholism and per capita consumption have not shown a similar decline’ (CDC, 1986). The explanation for this ‘paradox’, which has also been observed in Canada, is that increased spending on the treatment of alcoholism breaks the statistical link between per capita consumption and deaths from liver cirrhosis - ‘with treatment controlled, changes in cirrhosis

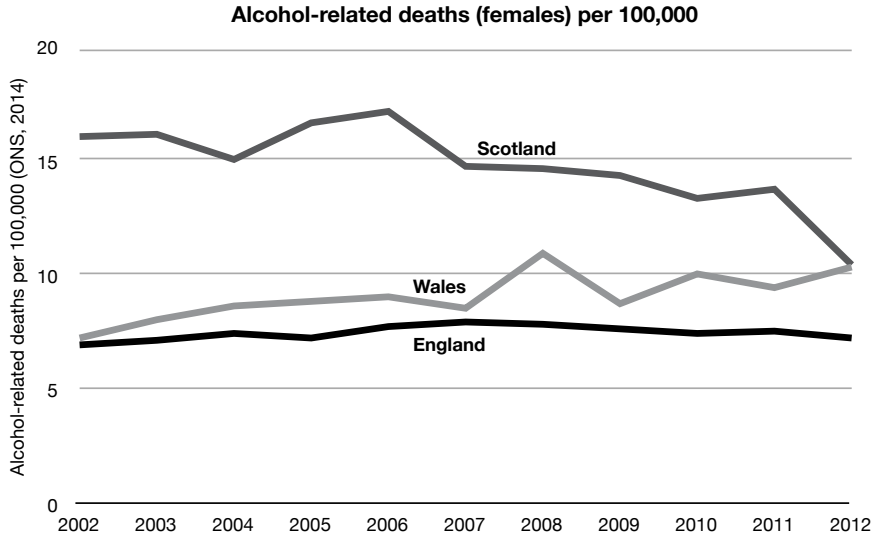
mortality are independent of consumption' (Holder and Parker, 1992). Put simply, treatment works.

The recent history of the UK throws up a further paradox. If NHS data are taken at face value, there was a 135 per cent rise in alcohol-related hospital admissions between 2002/03 and 2011/12, a period in which per capita alcohol consumption fell by 18 per cent (Alcohol Concern, 2013). A close study of the home nations raises further questions. England, Scotland and Wales share the same tax regime, have similar alcohol prices, similar licensing laws and identical advertising restrictions. Despite being comparable on almost every measure, Scotland has seen a 37 per cent decline in alcohol-related deaths amongst males and a 35 per cent decline amongst women since 2002 (see graphs below). England, by contrast, has seen little change amongst either sex and Wales has seen a rise in alcohol-related deaths, particularly amongst women (ONS, 2014). Looking specifically at alcoholic liver disease, which makes up the bulk of alcohol-related deaths (63 per cent), mortality increased by 18 per cent in England and Wales between 2002 and 2012 despite a major decline in per capita alcohol consumption (ONS, 2014b).

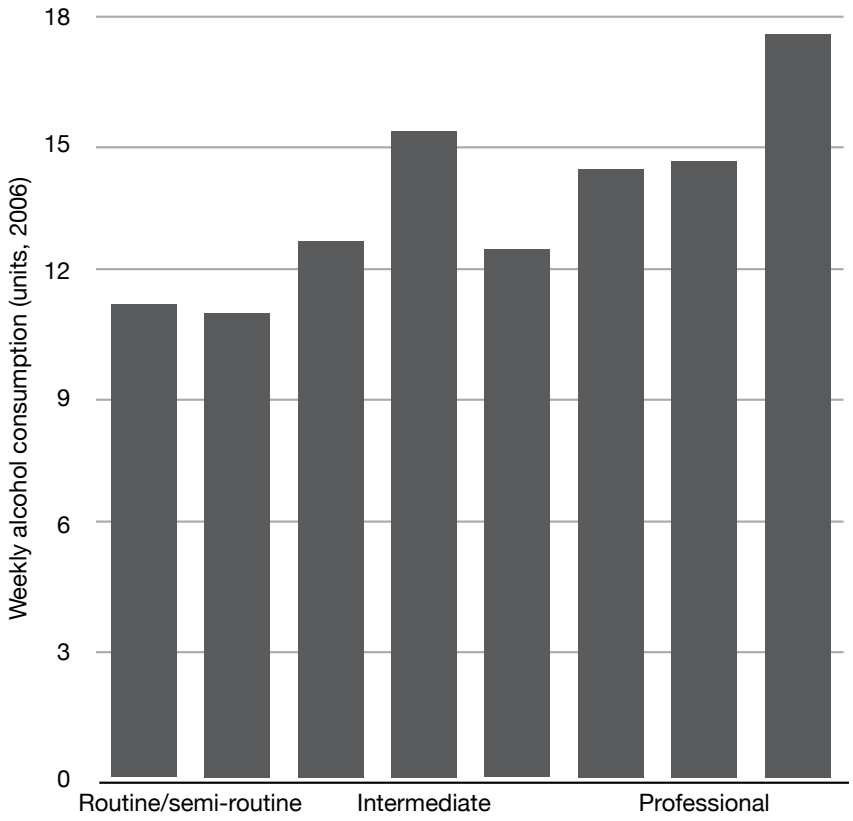
A relevant question here is whether there is a time lag between consumption and alcohol-related mortality which might explain why falling consumption in the UK has not led to a commensurate fall in alcohol-related mortality. The answer is that there *is* a time lag for chronic diseases such as liver cirrhosis, but that it is much shorter than might be supposed. As Holmes et al. (2012: 8) note, 'despite the anticipated long-term effect, much of the impact on cirrhosis mortality rates occurs in the first year following a change in consumption'. Studies show that 60 per cent of the total decline is seen within twelve months (ibid.: 10). Although counterintuitive, this can be explained by the fact that those who have liver cirrhosis can often prevent their death by ceasing, or dramatically reducing, their drinking. This is supported by evidence from the two world wars when rationing had a rapid impact on alcohol-related deaths in several countries; notwithstanding the fact that many young men were overseas (Norström, 1987: 69). But aside from liver cirrhosis,

most forms of alcohol-related mortality (eg. acute alcohol poisoning, drink driving, drownings and suicide) have no lag at all. If Ledermann were correct, we should have started seeing an inevitable and significant decline in both hospital admissions and alcohol-related mortality across the whole of Britain ten years ago. This has not occurred.

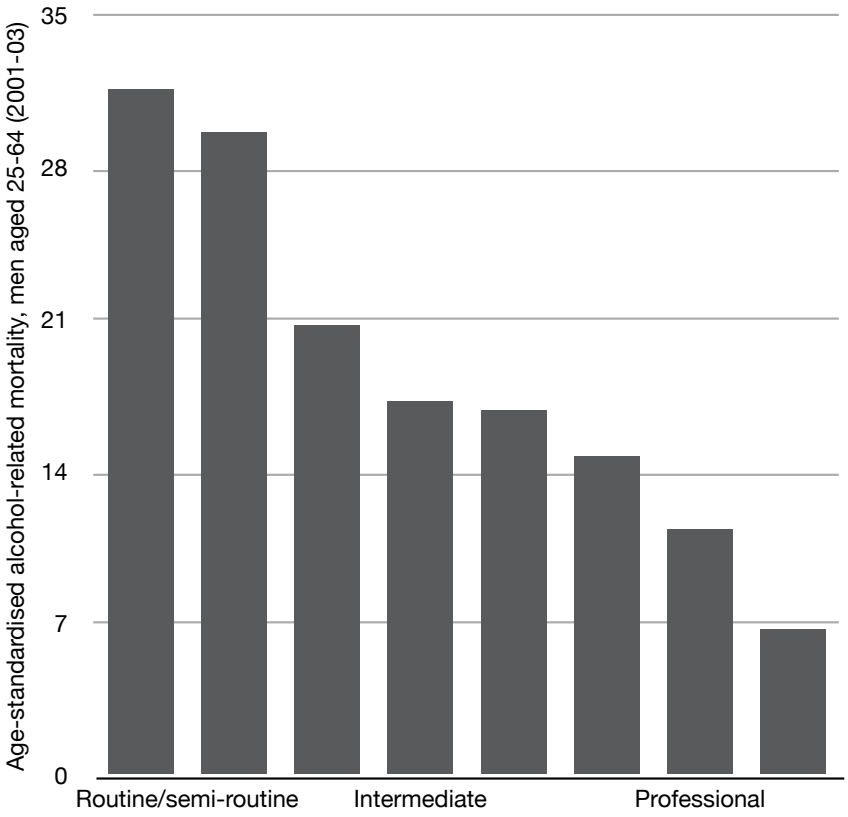




Another striking paradox can be seen if we divide England and Wales into subpopulations based on socio-economic status. The graphs below show alcohol consumption and alcohol-related deaths amongst different occupational groups (ONS, 2008). Although alcohol consumption is highest amongst the wealthiest groups and lowest amongst the poorest groups, the relationship is completely reversed when it comes to alcohol-related mortality (Siegler et al., 2011: 20). The socio-economic groups that drink the least have far higher rates of alcohol-related disease and death than those that drink the most.



It is certainly likely that Britain's high tax regime contributes to the lower rates of alcohol consumption amongst those who can least afford to drink, but this clearly does not translate into better health outcomes. According to the Office for National Statistics, men and women working in routine jobs are 3.5 and 5.7 times (respectively) more likely to die from an alcohol-related disease than those working in higher professional jobs, despite being in socio-economic groups that drink the least alcohol (ONS, 2011). The most plausible (partial) explanation for the 'alcohol harm paradox', as it is known, is that lower alcohol consumption in the poorest groups disguises the presence of a minority *within the group* who are drinking vastly more than the group average. Once again, overall consumption is a poor predictor of individual risk and averages are misleading.



As we write this, Alcohol Research UK are supporting research into this issue with a grant to academic researchers to support a project on *Understanding the 'Alcohol Harm Paradox'*. Perhaps one consequence of the research will be to identify the factors (social or individual) which eliminate or reduce the potential impact of consumption, but we fear that it will instead be another rescue attempt for the primacy of average consumption, using ad hoc arguments to 'explain' why although it doesn't work, it 'really' does. As Popper wrote, 'Some genuinely testable theories when found to be false, are still upheld by their admirers - for example by introducing *ad hoc* some auxiliary assumption, or by re-interpreting the theory *ad hoc* in such a way that it escapes refutation. Such a procedure is always possible, but it rescues the theory from refutation only at the price of destroying or at least lowering its scientific status' (Popper, 1963). The alcohol research and advocacy community frequently make predictions which are shown later to be wrong, and are assiduous in attempting *ad hoc* rescues. As long ago as 1980, when results indicated that the liberalisation of previously fairly draconian Scottish licensing laws did not lead to the predicted dire consequences, researchers tried to maintain that this was because Scotland experienced a particularly severe recession. It didn't (Duffy, 1992). It is extraordinary and dispiriting to see academics whose predictions fail time after time, who misrepresent the situation on the ground (such as claiming that alcohol consumption in the UK is increasing dangerously when it is not), and who persist in offering only one 'solution' (restrictions) being taken seriously.

There are too many counter examples of alcohol-related harm rising while consumption falls - and vice versa - for them to be dismissed at 'paradoxes'. As has become increasingly obvious since the 1970s, the many exceptions disprove the rule.

The persistent appeal of the model – and why

The Ledermann hypothesis no longer enjoys the academic support it enjoyed in the 1970s (see, for example, Parker and Harman, 1978; de Burgh, 1983; Skog, 2006; Roche, 1997, and Nelson, 2013). One of the key shifts in thinking about alcohol policy in recent decades, notes Roche (1997: 621), is the 'acceptance that there is no single distribution theory that adequately accounts for alcohol consumption'. Moreover, it is increasingly recognised that drinking patterns are more important than overall consumption and that 'trying to shift the population average may require a specific, targeted focus on smaller populations that generate the bulk of the problem' (Harper, 2009: 1744).

Nevertheless, many campaign groups and some government institutions cling to the Total Consumption Model and urge the state to implement policies aimed at the majority in the hope of reaching the minority. The European Association for the Study of the Liver (2013), for example, insists that: 'Any evidence based policy in Europe needs to implement preventive measures aimed at reducing alcohol consumption at the population level.' In the USA, the Centers for Disease Control was advised by the Community Preventative Services Task Force that there is 'extensive evidence' for the Total Consumption Model and yet cited only one reference to support this claim (Butterworth, 2013).

In the UK, citing the theories of Geoffrey Rose, the National Institute of Clinical Excellence (NICE, 2010: 28) states that 'the number of people who drink a heavy or excessive amount in a given population is related to how much the whole population drinks on average. Thus, reducing the average drinking level, via population interventions, is likely to reduce the number of people with severe problems due to alcohol.' Similarly, the state-funded pressure group Alcohol Focus Scotland says that they 'aim to reduce harm by bringing about a significant reduction in alcohol consumption across the population.'[6] Both organisations aim to reduce per capita consumption by targeting the Three A's - advertising, affordability and availability.

Although the traditional objectives of limiting advertising, availability and affordability are simple and politically appealing, there are good reasons to doubt their efficacy. There is a substantial economic literature showing that advertising does not increase primary demand for alcohol (or most other established products) - see, for example, Duffy (1995) and Nelson (2006, 2010). With regards to availability, in the run-up to licensing liberalisation ten years ago Professor Roger Williams - then famous for providing medical care to the late George Best - simply repeated this availability-consumption mantra. 'I don't think there is any evidence that lengthening the periods of drinking in this country will lead to less alcohol consumption. It will lead to more'. In fact, consumption began falling immediately after the Licensing Act came into force and has since fallen by 18 per cent - the largest sustained decline in alcohol consumption since the Second World War (BBPA, 2014).

As for affordability, basic economics suggests that price and consumption typically have an inverse association and yet there has been a decades-long decline in alcohol consumption in most European countries despite alcohol becoming more affordable. In the UK, campaign groups such as the Institute of Alcohol Studies frequently state that alcohol 'is 45% more affordable than it was in 1980'[7] without acknowledging that per capita consumption is at

6 http://www.alcohol-focus-scotland.org.uk/what-we-do (accessed 8 April 2014)
7 http://www.alcoholconcern.org.uk/campaign/statistics-on-alcohol (accessed 8 April 2014)

almost exactly the same level today as it was in 1980 (BBPA, 2012: 100). In any case, it is well established that heavy and dependent drinkers - the ostensible target of policy interventions - are much less price sensitive than moderate drinkers (Wagenaar et al., 2009; Nelson, 2013b). Recent research has concluded that 'reducing alcohol consumption through price and tax increases will be less effective or more costly than previously suggested or claimed' (Nelson, 2013: 9).

It is not surprising that such a simple idea as population-based alcohol policy remains attractive despite its scientific shortcomings. From the outset, Ledermann's theory had 'connotations of temperance and morality rather than science' (Berridge, 2002: 146). The Total Consumption Model gives anti-alcohol campaigners a justification for returning to the policies of the nineteenth century. These policies are fundamentally political in nature, targeting the general population, rather than being fundamentally about healthcare for high-risk groups (see table). As with the original temperance movement, they offer no target to aim for - the goal is always incrementally lower consumption through sales restrictions and ever-higher taxes.

Generalised interventions	Targeted interventions
	Rehabilitation services
	Educational campaigns
Advertising bans	Anti-alcoholism drugs
Tax rises	Enforcement of laws regulating sales
Minimum pricing	Helplines
Licensing restrictions	Licensing codes (eg. Challenge 21, Pubwatch)
State alcohol monopoly	Primary care interventions (eg. brief advice)
Prohibition	Harm reduction (eg. safer bars, designated driver schemes)

Nothing in this paper will seem new or controversial to those who have followed this academic debate in the last forty years. We have written it for lay readers who are likely to have been misinformed by campaign groups that remain wedded to the theory despite extensive empirical evidence to the contrary. Medawar (1996) described certain types of explanations in science as 'analgesic pills that dull the aches of incomprehension without going to their causes'. It is not always easy to reach problem drinkers and even those who seek help may find it difficult to tackle their alcohol problems. No wonder, then, that a population-level response has wide appeal as a means of altering the behaviour of alcoholics indirectly. The Total Consumption Model has an attractive simplicity that has allowed it to withstand the battering it has received from real world evidence.

'Who benefits from the domination of the total consumption model?' asks the Finnish alcohol researcher Kari Poikolainen in a recent book. 'Governments that tax alcoholic beverages, countries that have state alcohol monopolies, bureaucrats that monitor and control the rules and the profession of alcohol researchers. And who suffer? Moderate drinkers. They are mostly in good health but they are a large group and thus tempting for the taxman' (Poikolainen, 2014: 133). We argue that moderate drinkers are not the only ones to suffer. Those who experience alcohol problems have long been neglected by a public health lobby that favours ineffective, broad brush population interventions over specialist alcohol treatment.

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