INSTITUTE OF ALCOHOL STUDIES

IAS Response to the Strategy Unit's National Alcohol Harm Reduction Strategy Interim Analytical Report

1 Introduction

1.1 As you know we greatly welcome the development of the National Alcohol Harm Reduction Strategy. We would like to congratulate the Strategy Unit on producing the Interim Analysis which we believe has many positive aspects and which we think makes a substantial contribution to the field. However, we are concerned by certain negative features of the Analysis's coverage and use of the available evidence, which in our view will limit the value and impact of the National Alcohol Harm Reduction Strategy, when it finally appears.

1.2 The main problem with the Analysis is that it sometimes appears to downplay the negative consequences of alcohol consumption while being noticeably willing to recognise any alleged benefits. The Analysis, described on page 2 as focusing on the harms associated with alcohol, actually begins with a section summarising its supposedly large positive contribution to the longevity and quality of life of the population and its key contribution to the national economy. This section noticeably lacks the critical edge evident in later sections of the report concerned with the issues of price and availability.

1.3 There appears to be a double standard whereby a higher standard of proof is required in regard to supposed negative consequences of alcohol, and also to policies designed to prevent alcohol problems, than in regard to the supposed benefits of alcohol. For example, we are told (page 37) that heavy drinking may adversely affect mental well-being but that the relationships are complex and not necessarily directly causal. No such qualifications are entered in regard to the assertion (page 71) that alcohol plays a positive role in workplaces as a perceived antidote to pressure and as a way to `network' with colleagues.

1.4 There are some notable omissions and some basic confusions. For example, it is strange that a report which gives considerable emphasis to the problems of youthful binge drinking ignores evidence suggesting that bingeing in adolescence may cause brain damage, a finding which may be considered to have major implications for policy.

1.5 On page 99, headed `Levels of excessive drinking vary across occupational groups, but in rather complex ways", the data presented to do not relate to occupational groups but to socio-economic status. The relationship between drinking and occupational groups such as publicans and journalists is in fact rather clear and straightforward. There is no mention of workplace alcohol policies in this section.

1.6 The way that information is presented is sometimes potentially misleading. For example, the graph on page 93 resembles, and could easily be mistaken for, a pie-chart, which it is not. The equal size of the segments of the chart could be taken to imply that each causal factor so indicated is of the same weight and significance, and that the market is less important for the generation of harm than individual factors, or that drinking culture is of relatively little significance compared with the other influences indicated. There is of course nothing in the text to justify such inferences.

1.7 The whole section on the causes of problem drinking seems particularly prone to confusion. For example, on page 133, the issue is confused by the bracketing in the diagram of `frequency/volume of consumption' after `alcohol'. Where a triangular diagram of risk factors is employed, the `alcohol' factor is more usually considered to be the drug itself i.e ethyl alcohol, with all its various pharmacological properties. Frequency and volume of consumption are a product of the behaviour of the individual in a given environment. It would have been more helpful to have summarised the ways in which individual and environmental factors interact to modulate risk at the three levels of consumption, problems and dependence.

1.8 The diagram on page 134 is fairly helpful, but it also resembles a pie chart, which it is not. The apparent implication that `low price', for example, is equivalent to `early parental divorce' or `living in north' is not justified by the text.

1.9 Our other main concerns relate to the Analysis's treatment of the following issues:

- The relationship between alcohol consumption and harm at the population level
- The relationships between the availability of alcohol and problems of crime and disorder at the local level
- The economic costs and benefits of alcohol
- Alcohol and health

We will comment on these in turn. Dr Marsha Morgan has drafted comments specifically on alcohol and health and these are attached.

2 Consumption and Harm at the Population Level

2.1 The analysis of the relationship between overall consumption and harm is weak. The report acknowledges that as a nation we are drinking more but it avoids conceding directly that the overall level of consumption is the principal driver of the amount of harm, with all that that implies for prevention. In particular, the report omits any discussion of the fact that a relatively small proportion of the population – the heaviest drinkers, and those most likely to experience harm - account for most of the total amount of alcohol consumed. This is why, as the Royal College of Psychiatrists pointed out in its submission to the Strategy Unit, the alcohol industry has to be considered a competing interest in alcohol policy. This omission allows the report to sidestep completely the ethical and policy dilemmas arising from the inherent tension between health and social well-being on the one side, and the profitability of sections of the alcohol industry on the other.

3 Availability, Crime and Disorder

3.1 We have already raised the question of the, in our view, unhelpful and unwarranted changes to the text of an earlier draft of the Interim Analysis.

3.2 Extracts from the Draft analysis of August 2003 read as follows:

Extract 1

"There are two main supply side levers, price and availability, which can be used to influence alcohol use and misuse….Availability is governed by a number of factors: number and density of outlets; opening hours; regulation on who buys" (p. 150 –now page 152 of final report).

Extract 2

"As with price, restrictions on availability reduce general consumption and therefore general levels of harm. Conversely relaxing availability increases general harm whether through more outlets (Finland), denser outlets (California), longer hours (Western Australia) or reducing minimum age (New Zealand) where measures are not taken to pre-empt the consequences…"
(p. 152, now p.154)

Extract 3

"Supply and Pricing: Key Findings….Availability: Number and density of outlets and opening hours: where there are too many outlets, too densely packed, harm results. Communities need power to choose, and to respond where there is clear harm" (p. 154, now p.156).

3.3 In the final version of the report as released for public consumption on 19 September 2003 the analysis is now quite different:

- All mention of the control of outlet numbers/density and opening hours has been removed;
- The research findings from Finland, California and Western Australia in this regard have disappeared;

3.4 We are now told that, as levers of harm reduction, price and availability:

"…act in the context of a complex range of other factors that influence consumption (culture, advertising, setting and market innovation described earlier in the analysis). This means that changes in price and availability alone will not always affect behaviour, and that changes in behaviour may come about for other reasons (p.152 final version)

…In New Zealand a reduction in the minimum drinking age led to a perceived increase in anti-social behaviour by young people. But again the evidence suggests that the issue is more complex (p.154 final)

…So the issue is more complex than simply restricting price and availability for the whole population…The impact of policies involving price and availability will depend upon the range of different factors that influence consumption…there are limitations in using the evidence base to predict the response to specific policy measures on actual consumption. Whilst there is a clear association between price, availability and consumption overall, there is less sound evidence for the impact of introducing specific policies in a particular social and economic context and determining the right level. Other factors have to be taken into account too: Targeting-restricting price and availability would affect all drinkers, not just those experiencing problems. Limiting choice to reflect the needs of a minority who do experience problems may raise questions about fairness and acceptability….All of this suggests that price and availability, whilst important, are not the only levers and that they interact with other factors in ways which can have unintended consequences" (p.155 final).

"Supply and demand: key findings- Price and availability are important levers on overall consumption: there is clear evidence of links between price and availability and overall consumption, and hence harm. However, the evidence is less able to demonstrate the likely impact of specific measures: in some cases measures which should have reduced consumption have failed to do so; and in other cases consumption has fallen independent of policy measures. The interplay with other factors is crucial in determining overall behaviour. This means that policies can have unintended consequences…policies on price and availability have to be seen in a wider economic and social context" (p.156, final).

3.5 We are, of course, fully aware of the Minister's statement that changes to the text of earlier drafts were only to be expected, but this neither explains nor justifies these specific changes. The statement in the report that availability (in relation to, for example, number/density of outlets and trading hours) interacts with a range of other contributory factors to determine alcohol-related harms is a platitude. The 'this is no panacea' caveat must surely apply generally across the analysis, not only to issues of availability.

3.6 Issues of outlet density and trading hours go to the heart of the harm reduction debate. Empirical evidence in relation to such issues is exactly what practitioners and many other readers of the Analysis will be seeking. It is difficult to avoid the conclusion that an important agenda in research and practice has been ignored because its findings make uncomfortable reading for politicians and the drinks industry. The contents of the deleted 'extract 2' (above), for example, do of course stand in direct opposition to the Government arguments in support of the Licensing Act 2003.

4 Economic Costs and Benefits

4.1 In her notes on alcohol health, Dr Morgan points out that the estimated costs of alcohol problems to the health service given in the Analysis are surprisingly low in comparison with those given in the recent report from the Royal College of Physicians `Alcohol – can the NHS afford it?'.

4.2 However, perhaps the most surprising feature of the Analysis is the treatment of the supposed economic benefits of alcohol. Page 8 of the Analysis summarises these benefits in regard to tax revenue, employment etc. This section, however, sits rather uneasily with the discussion in the accompanying document `Alcohol Misuse: How much does it cost?'. In this, the claim that the income and employment generated by the industry should be represented as being benefits the community receives from the production of alcohol is rejected as fallacious. It is concluded that `*..the output, income and employment generated by the alcohol industry are not measures of social benefits attributable to alcohol.'* (page 14)

4.1 It is also worthy of note that whereas the chart on page 31 of the Interim Analysis gives costs totalling £15.4bn, the accompanying document gives costs totalling £18.5bn - £20bn.

A McNeill
14 January 2004

National Alcohol Harm Reduction Strategy Interim Report:

Critique of the Aspects Relating to Physical Harm

Marsha Y Morgan

Introduction

In preparing this critique I have reviewed the following documents:

- The Strategy Unit's Document ' Alcohol misuse: How much does it cost? September2003
- The National Harm Reduction Strategy Interim Analysis: Executive Summary: September 2003
- The Strategy Unit Alcohol Harm Reduction Project Interim Analytical Report: September 2003
- Alcohol-can the NHS afford it? Recommendations for a coherent strategy for hospitals by the Royal College of Physicians: February 2001

Alcohol-related Physical Harm

General Comments

1. For reasons that are not entirely clear, the definitions of alcohol consumption are unconventional and incomplete as is the ascription of harm in relation to the amount consumed. The definitions used in the report are alleged to be based on those used in official surveys *viz*:

    - *Binge*: x 2 the daily guidelines: _ ≥ 6units: _ ≥ 8units.
    - *Low to moderate*: within the weekly guidelines: _ ≥ 14units: _ ≥ 21units.
    - *Moderate to heavy*: _ ≥ 14-35 units: _ ≥ 21-50 units.
    - *Very heavy*: _ ≥ 35 units: _ ≥ 50 units
    - *Chronic*: sustained drinking, which is causing or likely to cause harm.

In addition *two* harmful patterns of drinking are recognised *viz*:

- Chronic heavy drinking - high frequency/high volume consumption; and
- Heavy single occasion or 'binge' drinking –low frequency/ high volume consumption

Thus there is no mention of the fact that a considerable amount of harm can arise from regular *moderate to heavy* drinking and no acknowledgment that *regular* binge drinking is commonly encountered in older individuals.

The term '*alcohol misuse*' is used in the report and is defined as 'alcohol-related disturbance of behaviour, disease or other consequences that are likely to cause an individual his/her family or society, harm now or in the future.' This is NOT a recognised definition (see below).

Similarly the term '*hazardous drinking*' is defined as 'an established pattern of drinking, which brings the risk of physical or psychological harm now or in the future'. It is stated that hazardous drinking can be rated by use of the AUDIT questionnaire and that it is also described as very heavy and binge drinking. This is clearly not an accepted definition (see below).

2. There is no mention of the fact that the risk for developing alcohol-related harm begins to increase at levels of consumption that would generally be considered moderate.

:

Low risk	
Intake unlikely to be associated with	_ ≤21 units/week
the development of alcohol-related	_ ≤14 units/week
harm if taken over 7 days	
Hazardous drinking	
Intake likely to increase the risk of	_ 22-50 units/week
developing alcohol-related harm	_ 15-35 units/week
Harmful drinking	
(synonym: **alcohol misuse**)	
A pattern of drinking associated with	_ >50 units/week
the development of alcohol-related harm	_ >35 units/week

3. No mention is made of the fact that the risk for alcohol-related injury increases with any level of consumption.
4. No mention is made of recent published work indicating that the levels of consumption associated with harm may be appreciably lower in adolescents and young adults.

5. No attempt has been made to systematically delineate the physical harm that alcohol can cause or to differentiate the effects of acute and chronic misuse. There appears to be no appreciation that while acutely intoxicated individuals may injure themselves, intentionally or otherwise, they may also develop gastrointestinal bleeding, cardiac arrhythmias, cerebrovascular accidents and respiratory depression, any of which may prove fatal. Equally, while it is acknowledged that individuals who chronically misuse alcohol may develop, and indeed die from cirrhosis of the liver, the fact that they may develop damage to every organ system in the body and that they are just as likely, if not more likely, to die as a result of suicide or injury, is not made clear. Indeed few of the recognised physical effects of alcohol are mentioned; those that are, are indicated in the lists below:

Effects of Acute Alcohol Misuse

Accidents and injury	√	Pancreatitis	
Acute alcohol poisoning	√	Cardiac arrhythmias	
Aspiration pneumonia		Cerebrovascular accidents	
Oesophagitis		Neuropraxia	
Mallory-Weiss syndrome		Myopathy/rhabdomyolysis	
Gastritis		Hypoglycaemia	

Effects of Chronic Alcohol Misuse

Accidents and injury		Brain damage:	
Oesophagitis	√	• dementia	
Gastritis	√	• Wernicke-Korsakoff syndrome	
Malabsorption		• cerebellar degeneration	
Malnutrition		• Marchiafava-Bignami syndrome	
Pancreatitis	√	• central pontine myelinolysis	
Liver damage:		Peripheral neuropathy	
• fatty change		Myopathy	
• hepatitis		Osteoporosis	

• cirrhosis	√	Skin disorders	
Raised blood pressure		Malignancies	√
Cardiomyopathy		Sexual dysfunction	
Coronary heart disease	√	Infertility	
Cerebrovascular accidents	√	Foetal damage	√

Mention is made in the report of 'neuropsychiatric' conditions but no specifics provided. There is no mention of alcohol-related brain damage; this is a serious omission

6. The section is confusingly and illogically written with no attempt made to separate: (i) the effects of acute and chronic alcohol misuse; (ii) the physical and mental effects of alcohol misuse; and (iii) alcohol intoxication and dependence. Thus, in sequence this section covers:
    - Hospital admissions and deaths
    - The disease burden—excluding the beneficial effects!
    - Liver disease
    - Blood pressure/ haemorrhagic stroke
    - Mental health
    - Alcohol dependence syndrome
    - Cancer
    - Foetal damage
    - Accidents
    - Alcohol intoxication
    - Suicides
    - Total deaths-excluding lives saved
    - Potential years of life lost
    - Costs

7. The data on the protective effects of moderate consumption on coronary heart disease are used selectively, incorrectly and repeatedly throughout the document. The specifics will be addressed in the next section.

Specific Comments

A number of statements are made which are incorrect or where evidence has been over interpreted:

1. The risks of some harms, such as liver cirrhosis, is disproportionately higher when larger amounts are drunk (Page 26).

   This statement is incorrect. The propensity to develop cirrhosis is individually determined by a combination of genetic, constitutional and environmental factors. In susceptible individuals the risk for developing cirrhosis increases significantly once consumption regularly exceeds a threshold level variously identified as 40-60 g/day in men and 20-40g/day in women. Above this threshold the risk increases but rapidly reaches a plateau at levels of intake that would still be considered relatively modest. Thus, in susceptible individuals, the intake required to increase the risk of developing cirrhosis is modest and risk does not increase substantially if the intake is increased. Conversely, individuals who are not susceptible will not develop cirrhosis no matter how much they drink nor for how long.

2. Liver cirrhosis is found in one in five heavy drinkers, *but the risks are higher in Mediterranean rather than non-Mediterranean peoples, perhaps due to different patterns of drinki*ng (Page 35).

   There is no evidence to support this contention of which I am aware. The development of cirrhosis is associated with *regular daily drinking*. It therefore follows that the cirrhotic death rates will be higher in countries adopting this pattern of consumption e.g. UK, France, Italy and Spain. It also follows that cirrhotic death rates will be lower in countries such as Sweden Norway and Finland where binge drinking is more common. Thus the tenor of this sentence is incorrect. It should state that ' Cirrhosis develops is approximately 20% of men whose alcohol consumption regularly exceeds 40-60g/day and a similar percentage of women regularly consuming in excess of 20-40g/day'.

3. Age –specific rates of mortality from liver disease explicitly attributed to alcohol have risen by about 90 % over the past decade. *Interaction of alcohol misuse with hepatitis C virus probably contributes to this trend* (Page35).

There is absolutely NO evidence to support this contention. The number of alcohol misusers infected with HCV in the UK is probably less than 5%. It is possible that *susceptible* individuals infected with HCV who also abuse alcohol may develop cirrhosis more quickly than their uninfected counterparts but infection with HCV does not itself confer susceptibility. Individuals infected with HCV who misuse alcohol are less likely to respond to treatment with anti-viral agents and this is the most important aspect of the relationship between these two hepatotoxic agents.

4. A high alcohol intake can also have adverse effects on other parts of the digestive system, including gastritis, *stomach ulcers*, oesophagitis, *oesophageal varices*, and pancreatitis (Page 35).

Alcohol misuse is NOT associated with an increased prevalence of peptic ulceration; indeed, alcohol misusers tend to have a lower prevalence of infection with *Helicobacter pylori* than non-habitual drinkers; indeed it has been suggested that alcohol might have an important anti-*Helicobacter pylori* effect. Alcohol does not cause oesophageal varices. These develop as a complication of the portal hypertension that develops in patients with cirrhosis.

5. In assessing the disease burden of alcohol misuse comment is made that high blood pressure is a higher risk factor (Page 34). However, the fact that alcohol is a causal factor for hypertension is not mentioned although this is commented upon later (Page 36).

6. Chronic high alcohol intake raises the risk of both haemorrhagic and ischaemic stroke (Page36)

Acute alcohol misuse is also associated with an increased risk of stroke, which is a particular risk for young drinkers. Alcohol taken acutely or more regularly, even in moderate amounts, is a risk factor for subarachnoid haemorrhage.

7. Drinking alcohol increases the risk of breast cancer in women, though the magnitude of the risk is small and the mechanism unclear. The results of new research are being considered by the Committee on Carcinogenicity which will help to improve the knowledge base in this area (Page 40).

The results of this meta-analysis are now available and confirm that there is a significant increase in the risk of developing breast cancer associated with the consumption of as little as two drinks a day.

8. The whole section on drinking in pregnancy was poorly done (Page 40).

There is no mention of the fact that pregnant woman are advised not to drink during the first three months of pregnancy and to limit their drinking to 1 to 2 units of alcohol once or twice a week for he remainder of their pregnancy.

The Fetal Alcohol Syndrome (FAS) is described but nothing is said about the spectrum of Fetal Alcohol Effects (FAE). The more severe forms of foetal damage are observed in women who misuse alcohol throughout their pregnancy but even these women may produce infants that are seemingly unharmed. However, over time, these apparently unaffected infants may develop both cognitive and behavioural abnormalities; they tend to perform poorly at school and up to two-thirds show hyperkinetic, emotional, eating and speech disorders. The most vulnerable period for the foetus is from 4 to 10 weeks gestation but alcohol-related damage may occur throughout the pregnancy. Less severe but still recognisable foetal effects are also observed in the offspring of women who drink regularly throughout pregnancy but below misuse levels

A number of the statements made in this section do not make sense *viz*:

- There is no consensus on whether light drinking affects risk of FAS.
- FAS is also related to socio-economic status, maternal nutrition, obstetric history, maternal age, and possibly other confounding factors.

9. The cardioprotective effect of alcohol is mentioned repeatedly throughout the report but little detail is provided. The cardioprotective effect is said to apply across the age range, but is seen mainly at ages over 40, where the incidence of coronary heart disease is highest (Page 7).

This is not the way in which these data are normally interpreted. The cardioprotective effect conferred by consumption of 2 units of wine daily is seen in middle-aged men and possibly post-menopausal women. The effect is not conferred by consumption of alcohol during earlier life. Individuals who do not drink alcohol are NOT advised to start drinking in middle-life, as the subsequent risk of injuries and hypertension would offset the cardioprotective effect.

10. The deaths attributable to alcohol are offset by the lives saved (Page 44)

These data are flawed. The figures for alcohol-related deaths are clearly an underestimate and the data on lives saved are theoretical.

Health –related Costs

1. One-year data from one large hospital (Pirmohamed et al QJM 2000: 93: 291-5):
   - 11,520 alcohol-related attendances at A&E
   - 3,226 alcohol-related admissions via A&E
   - 12,000 additional alcohol-related outpatient referrals from A&E

The figure used in the cost-analysis for alcohol-related A&E attendances in the Interim Report was 4.8 million. This number would have been generated by 400 large hospitals alone.

2. Each year there are approximately 1,500 admissions to the Royal Free Hospital, London for alcohol dependence. The figure quoted the Interim Report for the entire country was 30,000.

3. According to the Royal College of Physicians Report (2001), alcohol-related NHS *inpatient costs* account for 2-12% of total NHS expenditure of £23.4 billion, that is £0.5 to £2.8 billion. The higher figure exceed the *total* health care costs provided in the Interim Report of £1.4 to 1.7 billion.

4. Finally there is the question of the missing £40,000,000 allocated for treatment services. The NHS is said to be the largest funder of alcohol services but provides only 30% of the services itself. The voluntary sector is the biggest provider of services. Alcohol Concern provided data in 2002 on the costs of specialist alcohol treatment services, estimating that the total costs were of the order of £50.2 million per annum. This figure has been extrapolated to £96.2 million in the Interim Report though no explanation is provided.

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