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POPULATION LEVELS OF ALCOHOL CONSUMPTION

Report on a WHO Working Group

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ABSTRACT

A working group was convened to consider population levels of alcohol consumption in the Member States of the European Region of WHO and the level of per capita alcohol consumption of lowest risk to physical, psychological and social wellbeing. An understanding of problems at the population level can be established through examining the interaction between individual risk and the distribution of consumption levels within the population. The working group concluded that public health policy within the European Region should continue to stimulate decreases in per capita consumption. The existing data relating alcohol consumption to health originates from countries where the consumption of alcohol is an integral part of the culture. In those countries where there is a cultural or religious tradition of not consuming alcohol, there are no public health grounds for recommending alcohol consumption. Even when taking into account coronary heart disease, at the population level, across all ranges of alcohol consumption found in almost all countries of Europe, a reduction in consumption is linked to better health. Public health policy on alcohol should not, however, be based solely on mortality statistics. All adverse effects of drinking, that is, mortality, morbidity, social and criminal consequences, as well as a deterioration in the quality of life, should be considered.

Keywords

ALCOHOL DRINKING – adverse effects

ALCOHOLISM – prevention and control

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INTRODUCTION

One of the components of the policy formulated in regional health for all target 17 is the reduction of alcohol consumption per capita. If at all possible, this should be supplemented by specific policy guidance on population levels of alcohol consumption that are of lowest risk to physical, psychological and social wellbeing.

A working group composed of experts from research institutions in Europe as well as Canada and New Zealand was convened from 14–16 December 1994 to consider population levels of alcohol consumption. The meeting was chaired by Sally Casswell, and Dr Jürgen Rehm and Dr Robin Room were Co-Rapporteurs. The working papers and the participants are listed in Annexes 1 and 2, respectively.

UNDERSTANDING AGGREGATE RISK THROUGH INDIVIDUAL RISK CURVES AND THE DISTRIBUTION OF CONSUMPTION

Epidemiology has traditionally focused on the health consequences of an exposure or a behaviour at the individual level and then extrapolated from the data to draw conclusions about health consequences at the population level. Such conclusions should respect two crucial points: the possibility of a prevention paradox and the mutual interdependence of behaviour changes within a population.

Depending on the nature of the relationship between different levels of exposure (in this case, consumption of alcohol) and outcome¹ and the distribution of exposure in the population,

¹ In the following the relationship between different levels of alcohol consumption and outcomes will be referred to as "risk curves".

most of the aggregate problems might originate at relatively low levels of exposure. For instance, most of the social and acute alcohol-related problems in industrialized countries originate in groups with moderate consumption because the relevant risk curve is almost linear, and because most people in the population show this kind of drinking behaviour. Thus, a prevention strategy concentrating on the heavy drinkers with the highest risks would not be sufficient. This phenomenon has been labelled the prevention paradox. However, the prevention paradox does not apply to all outcomes of alcohol consumption. For instance, the risk curve between alcohol consumption and liver cirrhosis is exponential, so that heavy drinkers do contribute to the majority of problems in this area, even if they constitute only a small minority of the population.

Changing the consumption behaviour of one subgroup within the population will have effects on the consumption behaviour of other subgroups. Most analyses of changes in alcohol consumption within a population over time showed that they tended to point in the same direction and be proportional to the original consumption level. Exceptions were changes stemming from quite drastic interventionist policies such as prohibition or rationing. In any case, possible consequences concerning the change of alcohol consumption of one subgroup in the population on the drinking behaviour of other subgroups have to be considered.

ALCOHOL CONSUMPTION AT THE INDIVIDUAL LEVEL

Risk curves for alcohol consumption at the individual level

Risk curves describe the relationship between levels of consumption and outcomes. Such curves have been established for a number of adverse outcomes of alcohol consumption

(mortality and morbidity, as well as social and criminal consequences). However, the overwhelming majority of studies have concentrated on different kinds of mortality.

Harmful consequences of alcohol intake include malignancies (cancers of the mouth, pharynx, larynx, oesophagus, liver and breast; with some indication of a dose-response relationship between drinking and colorectal cancer as well), raised blood pressure and haemorrhagic stroke, cardiac conditions other than coronary heart disease (CHD), liver cirrhosis, alcohol-dependence symptoms, criminal delinquency, and social problems. All of these harmful consequences show a monotonic relationship between alcohol consumption and the outcome: the higher the consumption, the higher the risk of the harmful outcome.

Overall, beneficial consequences of moderate alcohol consumption were found with respect to CHD and ischaemic stroke. For ischaemic stroke, there is some indication that the risk curve is curvilinear, so that lighter drinking reduces the risk whereas heavier drinking increases it. For CHD, there is also evidence that heavy drinkers have an increased risk compared to abstainers.

The J-shaped curve between alcohol consumption and all-cause mortality

The summary below is based mostly on epidemiological studies of males aged 40 years and over. More evidence on alcohol consumption and mortality in females has been found recently, but studies on people younger than 40 years of age are still very rare. The current studies are also limited geographically (mostly industrialized countries).

The relationship between alcohol consumption and all-cause mortality is a function of the distribution of the causes of death within the examined population and the risk curves for different causes of mortality. For most industrialized countries,

the overall relationship between alcohol consumption and all-cause mortality is J-shaped for both sexes.

Nonetheless, when taking different age groups into consideration, the relationship between alcohol consumption and all-cause mortality is linear in the younger age groups, both for males and females. The linear risk per unit of alcohol consumption is higher for females than for males. In the older age groups the curvilinear J-shaped relationship can be found. Since the older age groups contribute more to overall mortality, the shape of the function for all ages together is a J.

The reason for this age-specific pattern lies in the different causes of death for the different age groups in most industrialized countries. For example, in the United States the most prevalent causes of death for males aged under 40 are accidents and poisoning (ICD9 800-999), and for females of the same age group neoplasms (ICD9 140-239). For both groups of mortality causes there is a positive linear relationship to alcohol (the higher the consumption, the higher the mortality risk). The mortality pattern changes over the life course and cardiovascular diseases become the major cause of death (for males aged over 40, for females aged over 50), and the epidemiological evidence shows a protective effect of moderate alcohol consumption for cardiovascular diseases, especially for CHD. The protective effect of alcohol on CHD is also supported by biochemical evidence. At present, it is not clear whether the beneficial effect of alcohol consumption is both long-term and short-term or only short-term.²

The exact age when the relationship between alcohol consumption and mortality changes from linear to curvilinear depends on the mortality pattern of the examined region. For countries

² Since case control studies (with measurement of consumption close to outcome) show on the average higher beneficial effects than cohort studies (where the measurement of consumption usually takes place years before the outcome emerges) a short-term effect seems plausible.

where CHD is not a major cause of death, the relationship would be expected to be linear for all age groups. In the United States, the dividing point was found to be about 50 years of age. This dividing point may change in future years with the decline of CHD mortality in the United States, as in other industrialized countries.

The linear relationship between alcohol and mortality for younger age groups, and the curvilinear relationship for older age groups remain stable for different kinds of statistical adjustment: diet, smoking, age,³ education. However, the role of social and psychological factors, e.g. social integration, has not yet been sufficiently explored. The J-shaped relation of drinking and all-cause mortality may partly reflect the protective effect of social integration: moderate drinkers are more socially integrated than either abstainers or heavy drinkers.

If "unhealthy abstainers" are removed from the comparison group (by excluding people with a history of disease and/or by using life-time abstainers as a comparison group rather than "short-time" abstainers), the linear relationship in younger age groups becomes more pronounced, but the beneficial effect of moderate consumption does not disappear.

Epidemiological studies so far have not shown convincing evidence for a differential effect of different types of beverage. (There is some biochemical research showing more protective effects for red wine, but this question is far from being settled.) There is some evidence for the notion that quantity is more influential than frequency with respect to adverse effects; however, the pattern of drinking should be explored in more detail in future studies. Also, some evidence indicates a differential influence of drinking on CHD mortality in regard to smoking

³ When large age groups are analysed (e.g. from 25 to 59 years), where the mortality rate changes significantly within the age group, the variable age has to be adjusted in the statistical analysis.

status: the protective effect seems to be most pronounced in smokers.⁴

In most epidemiological studies on alcohol consumption and mortality, the measurement of consumption is quite poor. Nonetheless, there is some evidence that in measuring consumption the rank order between people is mostly preserved. Since mortality studies are usually based on cohort methodology with many years of follow-up, the influence of recent improvements in measurement will only be seen as the more recent studies are completed.

Conclusions at the individual level

The only beneficial effect found at the individual level concerns subcategories of cardiovascular diseases, in particular CHD. Except for CHD, the conclusions from the epidemiological evidence can be summarized in one sentence: across all ranges of alcohol consumption, the less the consumption, the better with respect to health.

When taking CHD and to a much lesser degree ischaemic stroke into account, the conclusion must be broken down by country. For countries with a low incidence of CHD (largely those outside the European Region) it can be concluded that across all ranges of alcohol consumption, the less the consumption, the better with respect to health.

For countries with a high incidence of CHD, the conclusion must be broken down by age. However, for younger age groups, which have almost no risk of CHD the same conclusion can be made: across all ranges of alcohol consumption, the less the consumption, the better with respect to health. The

⁴ Such an influence cannot fully be modelled by the usual adjustment for smoking (that is, by including one or two dummy variables for smoking status in the equation). As a consequence, almost all of the studies that claim adjustment for smoking did not consider this effect.

exact dividing point between younger and older age groups within a population depends on the mortality pattern of the examined country. With a reduced or declining CHD mortality rate, the dividing point may be more than 50 years of age.

For older age groups that have an increased risk of CHD, cultural and societal patterns of drinking should be taken into account. For alcohol to have a protective effect, one drink every other day appears to be sufficient. Individual advice to drinkers should focus on lower-risk drinking (in the range of up to two drinks a day). For abstainers with a high risk of CHD there are alternative ways of reducing CHD risk such as increasing physical activity, making dietary changes, quitting smoking and taking aspirin.

ALCOHOL CONSUMPTION AT THE AGGREGATE LEVEL

Distribution of consumption

The distribution of alcohol consumption can be characterized as mixed, broken down by those who do not drink and those who intake alcohol at least occasionally. Of course, members of the former group are all concentrated in one consumption level (0 consumption) with no variance.

The distribution of those who drink alcohol at least occasionally usually follows an approximately lognormal shape, with a peak at the lower levels of consumption and a quite long tail to the right side. In no country has a second peak at the right side of high consumption been identified. Such a peak would indicate the overlap of two distinct populations, for instance, non-alcoholics and alcoholics. Instead, the percentages of the population at the right side decrease monotonically with increasing consumption. A lognormal distribution also implies

that the modal value of consumption is smaller than the median, which in turn is smaller than the mean value.

Empirical analyses of shifts and changes in distributions of alcohol consumption indicate that the percentage of heavy drinkers in a population is related to the mean value in a consistent manner. Increases in mean consumption produce increases in the percentages of heavy drinkers and decreases in mean consumption reduce the percentages of heavy drinkers in populations. This pattern is also true cross-culturally: the higher the mean consumption in a country the higher the percentage of heavy drinkers, and the lower the mean consumption, the lower the percentage of heavy drinkers. It should be noted that these relationships are strong empirical tendencies rather than natural laws.

The patterns described are consistent with a social network theory of alcohol consumption, where each drinker influences the drinking of others in the drinking setting, and where each individual's change in consumption, caused by any particular impetus, tends to be proportional to the amount already consumed.

Aggregate level studies about consequences of alcohol consumption

Aggregate level analyses should be conducted to support the results from individual level studies and theoretical conclusions about effects of distribution changes. The cross-confirmation of individual level results is particularly convincing, since the two types of research tend to have different sources of error.

Cross-sectional aggregate studies are of lesser value because they are prone to ecological fallacies. In the past, the outcome of such studies seemed to depend very much on which potential confounder had been added. Time series analysis seems to be a more adequate tool in deciding about possible

benefits and risks of alcohol consumption. However, *a priori* decisions have to be made about the temporal structure of the effects of alcohol consumption. Time series analysis also depends on the availability of comparable data on dependent, independent and confounder variables for longer periods of time (minimally 30 data points).

Compared to individual level studies, there have been very few time series analysis on different outcomes of alcohol consumption. Notable exceptions are studies on alcohol consumption and suicide as well as on alcohol consumption and liver cirrhosis where, in general, positive relations have been found (the higher the consumption, the higher the risks for suicide and cirrhosis). In regions with high per capita consumption, alcohol consumption was also found to be positively related to overall mortality. There is also some evidence from time series analysis that alcohol consumption is related positively to measures of criminal violence, including homicide.

A study on alcohol consumption and CHD mortality in three countries prepared for this Working Group did not disconfirm the protective effect of alcohol consumption. The study modelled acute (= simultaneous) effects of alcohol consumption on CHD.

Public health consequences of the J-shaped curve for all-cause mortality

The lowest level of alcohol consumption in regard to risk of mortality for a population does not correspond to the lowest level with regard to individual mortality. According to the rule of proportionality (that is, in the case of a population's change in consumption, individual changes in consumption are proportional to the previous base individual consumption), the lowest population mortality risk is associated with considerably lower alcohol consumption levels than the lowest individual

mortality risk.⁵ Even when this rule is not strictly true, the lowest population mortality risk is at lower levels of alcohol consumption than the lowest individual mortality risk. The reason for this phenomenon lies in the mutual interdependence of consumption changes in a population.

The presence of uncontrolled confounders in individual studies might add to the difference between lowest mortality risk levels of alcohol consumption for individuals and the population. For instance, combining two subgroups with different absolute risks and different variances of the consumption distribution, but with the same relative J-shaped risk, leads to a combined relative risk for mortality with a lowest risk level at higher consumption levels than in either subgroup. This is illustrated in Table 1.

Table 1. Hypothetical example of a confounding variable that produces bias both in the magnitude of risk reduction and the optimum level in the case of J-shaped risk curves (consumption and risk in arbitrary units)

Consumption level	Risk in group 1	Risk in group 2	Percentage in group 1	Aggregate risk	Aggregate relative risk
0	1.00	2.00	50	1.50	1.00
1	0.90	1.80	60	1.26	0.84
2	0.95	1.90	70	1.24	0.82
3	1.00	2.00	80	1.20	0.80
4	1.20	2.40	70	1.56	1.04
5	2.00	4.00	60	2.80	1.87
6	4.00	8.00	50	6.00	4.00

In Table 1, the assumption is made that the population can be split into two different groups of individuals according to their social ties – weakly and strongly integrated, respectively.

⁵ If, when using a simulation with plausible assumptions, the individual lowest risk level is taken as 20–30 g of pure alcohol per day, or 10–15 litres per year, the lowest population mortality risk level is about one fifth, or 2–3 litres per capita age 15+.

Alcohol is presumed to have a real protective effect, and within both groups the moderate consumers (consumption level 1) have the lowest risk. At higher consumption levels the risk increases, but there are still some protective effects in the next consumption level (i.e. the risk in consumption level 2 is still below the abstainers' risk). At higher consumption levels the risk is assumed to be accelerating. This pattern is the same in both groups, but the weakly integrated group is presumed to have a risk that is twice as high as the strongly integrated group at all consumption levels. The strongly integrated group is assumed to constitute a clear majority in the intermediate consumption categories, while their dominance is less pronounced at both extremes.

It can be seen that the lowest risk is no longer found in the lowest consumption category, but in consumption level 3. Hence, failing to control for social integration would give the impression of protective effects over a much too wide range of consumption levels. Further, while the true reduction in risk is only 10% (from 1% to 0.9% in consumption level 1 and from 2% to 1.8% in level 2), the overall risk function suggests that the protective effect is 20%. Hence, a J-shaped confounder such as social integration may both substantially exaggerate the protective effect and produce a bias towards higher consumption levels. Clearly then, social factors need to be controlled, before any confident conclusions can be drawn on the basis of epidemiological data.

Given that the level with the lowest mortality risk at the population level is known, if population guidelines based on mortality alone were to be developed the described aggregate effects should be taken into account as well as individual variation. The following points in particular should be considered.

- The promotion of alcohol consumption in one subgroup of the population may trigger changes in other parts of the population as well, with the result that the overall risk goes

up. Unintended effects for groups that are already drinking at high-risk levels should in particular be avoided.

- Population level guidelines should take safety margins into account as do security measures and regulations in other areas (e.g. speed limits in traffic, safety standards for toxic chemicals).

Conclusions at the aggregate level

The only beneficial effect discernible at the aggregate level is CHD. Except for CHD, the conclusion from aggregate level analyses can be summarized in one sentence: across all ranges of alcohol consumption, the less the consumption, the better with respect to health.

Taking CHD into account, the conclusion must be broken down by country. For countries with a low incidence of CHD it can be stated that: across all ranges of alcohol consumption, the less the consumption, the better with respect to health. For countries with a high incidence of CHD, public health policy should continue to recommend decreases in per capita consumption. Even when taking only mortality into account, this policy seems appropriate to almost all industrialized countries, since the lowest mortality risk for populations (2-3 litres per capita per year age 15+) is at a lower level of alcohol consumption than the present per capita consumption in these countries. But, public health policy on alcohol consumption should not be based solely on mortality. All adverse outcomes of drinking, that is mortality, morbidity, poor quality of life, and social and criminal consequences, should be considered.

However, since CHD does constitute a major cause of death in most industrialized countries, the consequences of the beneficial effect of alcohol consumption on this outcome should be explored more carefully. From a public health perspective, knowledge about two processes is necessary in order to make additional recommendations.

- Is the beneficial effect mainly an acute effect, or is steady alcohol consumption over an extended period of time necessary to build up the protection against CHD?
- What other types of behaviour can substitute for the beneficial effect of alcohol on CHD? To what extent does other protective behaviour substitute for, rather than add to, the protective effect of drinking? What are the cost-benefit ratios of this substitute behaviour?

PUBLIC DISCOURSE ON THE CONSEQUENCES OF ALCOHOL CONSUMPTION AND ITS IMPACT ON POLICY-MAKERS

The beneficial effect of moderate alcohol consumption on CHD has received more media attention⁶ than other research showing alcohol as a risk factor for different outcomes. It is being used by the alcohol industry and other pressure groups to influence public policies on alcohol as well as to promote more consumption and to reject contrary claims made by people in public health.

From a public health perspective, the beneficial effect of moderate consumption on CHD should be seen together with all other outcomes of alcohol consumption. At present, no convincing argument has been brought forward to modify current public health policies on alcohol, including those on legal drinking ages, random breath testing to enforce the limits for blood alcohol content while driving, or taxation on alcoholic beverages.

⁶ Part of this attention is due to the fact that this message fits well into the ideological climate of the 1980s and 1990s which can be characterized by a shift towards favouring individual freedom over state regulation. This climate has also influenced public discourse on alcohol issues.

Public discourse on the consequences of alcohol consumption should always try to separate the individual from the population level. In certain clinical situations it might well be that a medical doctor advises a 65-year-old overweight smoker with a family history of heart disease to drink moderately in a regular pattern in order to lower chances of a myocardial infarct, especially after having unsuccessfully tried to change other behavioural components. Such clinical cases cannot and should not be generalized to population levels. In particular, there is no sufficient knowledge base to publicly advocate that all abstainers should drink moderately to avoid CHD.

There seems to be some demand for guidelines on low-risk alcohol consumption. Their formulation would, however, be quite difficult. From a scientific point of view such guidelines would inevitably be quite complicated, as they should take into account sex, age and drinking settings. Furthermore, such guidelines must also consider how to influence public discourse on the consequences of alcohol consumption, public health policies and attitudes and behaviour towards alcohol consumption in the general population.

The dissemination of information to the general public through the mass media may have unintended effects and therefore should be phrased differently than advice to individuals.

RESEARCH NEEDS

Certain areas need to be more fully researched in order to guide the formulation of public health policies on alcohol consumption. Three areas in particular are most important.

- Improving our knowledge about individual and aggregate level risk curves for alcohol consumption and outcomes other than mortality.

This knowledge is necessary because public health recommendations should not be based solely on mortality but should integrate all other consequences of alcohol consumption as well.

- Improving our understanding of the lag structure in regard to the beneficial effect of alcohol on CHD through biological, chemical or epidemiological studies, as well as of the substitutability of this effect on CHD through other means (e.g. through changing diet, using certain drugs or taking other behavioural measures).

This research is necessary to improve knowledge about preventive measures for CHD in different age groups.

- Improving our knowledge about the dynamics of shifts in the population distribution of alcohol consumption.

This knowledge is necessary to avoid unintended adverse effects of public health recommendations in certain subgroups of consumers.

In addition to emphasizing these three broad research areas the participants identified the following research needs.

Epidemiological studies at the individual level:

- studies using samples of younger population groups;
- better understanding of the interaction between smoking and alcohol and the effect on mortality;
- studies integrating social and psychological confounders, especially the influence of social integration and social networks;
- exploration of to what extent abstainers and light drinkers differ by self-selection processes at the start of prospective

studies, and what would be the implications of such differences;

- studies integrating other drugs including possible interactions with medical drugs (especially benzodiazepine and contraceptives);
- studies that include different measures of alcohol consumption, especially ones focusing on patterns of consumption (e.g. peak drinking episodes, drinking with meals);
- validation of measures of consumption, including measures of drinking patterns.

Most of these points can be dealt with by reanalysis and/or meta-analysis of existing data sets. Such analyses should be conducted by going into the original data sets, rather than by combining published data. Statistical techniques should be employed that allow the transfer of results from one study with measurements of interesting variables (e.g. on social isolation) to other data from the same population.

Epidemiological studies at the aggregate level:

- exploration of relationships for previously unanalysed outcomes of alcohol consumption (cancer, criminal and social outcomes, different causes of mortality including CHD);
- analysis of the relationship between alcohol consumption and all-cause somatic mortality, especially in cultures with low consumption levels;
- aggregate level studies to examine the effects of "natural experiments" that change the extent or conditions of alcohol consumption.

Incorporation of epidemiological knowledge in the formulation of public health policy and recommendations:

- analysis of the consequences of shifts in the population distribution of alcohol consumption, especially for subgroups;
- development of models for including the consequences of such shifts in population distribution on abstainers;
- evidence from biological, chemical or epidemiological studies of the lag structure with respect to the beneficial effect of alcohol on CHD;
- exploration substituting other means (e.g. change in diet, use of other drugs, other behavioural measures) to achieve the supposed protective effect of alcohol on CHD;
- exploration of the reception to and consequences of guidelines in different population groups (especially with respect to own drinking behaviour) preferably by analysing "natural experiments" or by conducting field experiments;
- studies of the impact of information disseminated by the mass media.

Formulation of public health policy on alcohol:

- exploration of the societal and political effects of public recommendations;
- research into the role of the alcohol industry and other key interest groups in the making of alcohol policy.

*Annex 1***WORKING AND BACKGROUND PAPERS⁷***Working papers*

- EUR/ICP/ALDT 94 02/MT07/6 *Public discourse on benefits of moderation*
S. Casswell
- EUR/ICP/ALDT 94 02/MT07/7 *Alcohol consumption and CHD-mortality*
T. Norström and A. Romelsjö
- EUR/ICP/ALDT 94 02/MT07/8 *Alcohol consumption and all-cause mortality*
J. Rehm et al.
- EUR/ICP/ALDT 94 02/MT07/9 *Public health consequences of the J-curve hypothesis of alcohol problems*
O.J. Skog

Background paper

- Edwards, G. et al. *Alcohol policy and the public good.*
Oxford, Oxford University Press, 1994.

⁷ Copies can be obtained from the ADT unit, WHO Regional Office for Europe, Scherfigsvej 8, DK-2100 Copenhagen Ø, Denmark.

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