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EDITORIAL

Trends in the epidemiology of alcoholism[1]

A Symposium of the epidemiology of alcoholism was held in London in January 1977 – at a time when theories about the aetiology and prevalence of alcoholism had reached a crossroads.

The epidemiological approach has continually been at odds with the mainstream of theories about alcoholism. The traditional disease theory perceived alcoholics to have certain psychological or physiological idiosyncrasies which rendered them unable to control their drinking. The most popular explanations were that either some personality defect predisposed them to become alcoholics, or else some biochemical abnormality made them physiologically allergic to alcohol. As Davies (1977) noted at the Symposium 'there is little or no evidence to support any of these constitutional views' and, for some years, the epidemiological study of alcoholism has been suggesting an alternative perspective to these aetiological theories of psychological or physiological predisposition. Epidemiology has linked the prevalence of alcoholism with *per capita* consumption, i.e. the total amount of alcohol consumed in a society divided by the adult population. Whenever *per capita* consumption rises, so do all the rates of alcohol-related problems such as alcoholic mortality, liver cirrhosis mortality, arrests for drunkenness and for drunken driving, and hospital admissions of persons diagnosed as alcoholics. The strength of this relationship between the level of consumption and the level of alcohol-related problems can be traced in the national statistics of England and Wales, for example, as far back as World War I (Wilson, 1940). The years in which alcohol consumption increased the most were invariably the same years in which the prevalence of alcohol-related problems also increased the most. The consistency of this relationship implied that the number of people diagnosed as suffering from alcoholism – conceptualized by the disease theory as a permanent affliction – actually rose and fell with the amount of alcohol consumed by the total population. This suggested that the size of the problem should not therefore be conceived in terms of the number of people who were in some way pathologically inclined to become alcoholics. Whereas the disease theory presumed the causes of alcoholism to lie in traits internal to the drinker, the epidemiology of alcoholism was suggesting the causes lay not in the drinker, but in the drink.

Ledermann (1964) incorporated the evidence of this relationship into a theory which claimed that the level of *per capita* consumption actually determined the prevalence of alcoholism. He asserted that the frequency distribution of drinkers according to their individual consumption will always be highly skewed, with a majority of the population drinking relatively small amounts and successively smaller proportions drinking increasingly heavier amounts. In mathematical terms this distribution would always conform to the characteristics of a logarithmic normal curve. He further asserted that the end point of the upper distribution of individual consumption must be fixed at a point of one litre of absolute alcohol per day, on the grounds that drinking any more than this would be fatal. Usually a lognormal curve has 2 parameters – the standard deviation and the mean. By fixing the upper limit, the 'Ledermann distribution' allows only 1 parameter – the mean. Therefore, as this mean, i.e. *per capita* consumption, rises, the frequency of drinkers will always be smoothly and continuously redistributed so that predictable proportions of drinkers move into higher consumption categories. According to Ledermann, any increase in *per capita* consumption must represent a general move towards heavier drinking throughout the population. Since he also showed that societies with a higher *per capita* consumption also had the highest rates of alcohol-related problems such as cirrhosis of the liver, then a movement of a society to a higher overall consumption must inevitably lead to a higher rate of alcohol-related problems within that society. At the Symposium, De Lint (1977) concurred that the distribution curve is typically continuous, unimodular and positively skewed, and

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he believed 'the gradual transition from moderate to excessive consumption agrees very well with the failure thus far to discover real differences in the personality and physiology of the so-called social drinker and the so-called "alcoholic"'. His work over the years with Schmidt has led them to conclude that 'the prevalence of alcoholism is invariably determined by the overall level of consumption in the population' (De Lint & Schmidt, 1971). They have also been influential in demonstrating that *per capita* consumption was in turn related to the price and availability of alcohol. This phenomenon had been observed for many years (Wilson, 1940), but the Ledermann-based theory seemed to go further by explaining why reducing alcohol availability would reduce *per capita* consumption and thus also reduce the risk of people becoming alcoholics. A similar view was propounded in 1975 by a group of international experts headed by Bruun (1975), who propounded that alcohol-related health problems were concentrated among heavier drinkers and that the greater the average consumption the greater the number of heavy drinkers. They too suggested that the number of heavy drinkers, and consequently the number of alcohol-related problems, could both be reduced by restricting the availability of alcohol.

Prior to the Symposium, this consensus view had been somewhat disturbed by Miller & Agnew (1974) who queried Ledermann's manipulation of the lognormal distribution curve. They maintained that the fixed end point of one litre of absolute alcohol was just an arbitrary choice, and they cast doubts on the validity of the characteristics of a lognormal curve which had such a parameter artificially fixed. These criticisms were endorsed by statisticians at the Symposium, and Duffy (1977) added that surveys of the consumption of general population samples have produced distributions which did not correspond to a Ledermann distribution, or even a straightforward lognormal distribution. He therefore concluded that, despite the evidence of a strong relationship between consumption and problems, the explanations of this relationship reached by Brunn *et al.* 'are unjustified by their data', and that 'to reproduce the distribution of alcohol consumption on the basis of mean consumption alone' is 'illogical and contradicted by empirical evidence'.

Such a critique undermines many popular assumptions about the epidemiology of alcoholism. Ledermann's theory suggested that when *per capita* consumption rises the population moves towards generally heavier drinking; a much simpler explanation might be that there has just been a reduction in the number of abstainers. It has been assumed that consumption has increased because its real price has fallen, yet little data have been produced to show that groups whose disposable income has increased the most have also increased their alcohol consumption the most. Neither is there much good evidence that groups such as young people and women who have recently figured particularly in increased rates of alcohol problems were also the same groups who had most increased their consumption. In short, once the theories explaining the relationship between consumption and prevalence were exposed as equivocal, there was clearly very little data available to suggest any alternative mechanisms which might explain this relationship. The most blatant deficiency was the lack of data on relationships over time between consumption and problems within particular groups and individuals.

Three papers presented at the Symposium attempted to remedy this situation. Cartwright *et al.* (1977), Sulkunen (1977), and Makela (1977) all considered the potential importance of the stability of drinking patterns in determining how increased consumption was distributed. In 1974 Cartwright *et al.* replicated some measures of consumption in a London suburb previously made in 1965 (Edwards *et al.* 1972). Over the nine years *per capita* consumption increased 47 % and, as the Ledermann theory predicted, proportions of individual drinkers were smoothly redistributed into higher levels of consumption. Cartwright *et al.* felt that Ledermann's predictions had held pragmatically because there had been relatively little overall change in drinking patterns. The percentage of abstainers remained constant at 11 % and people did not drink on any more days of the week than previously. Rather, the major change was that on a drinking day in 1974, 'average drinkers' consumed 56 % more alcohol than they would have done in 1965. This fitted in with the theoretical framework suggested in papers by Sulkunen and Makela which hypothesized that increases in *per capita* consumption did not usually create new drinking patterns but were usually superimposed on existing patterns.

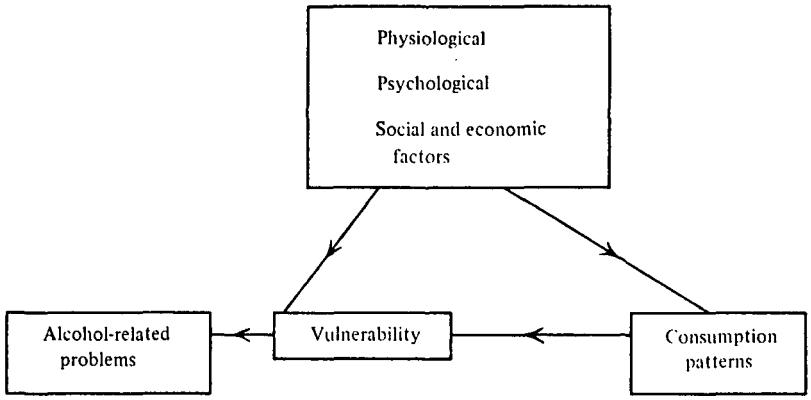

FIG. 1.

Makela's 'addition hypothesis' implies that new ways of drinking do not displace old ones but rather add new elements to them. Makela supported this conclusion with examples from Finland. Sulkunen stated that similar observations could be made in other countries. He pointed out that these additions to the existing drinking patterns comprised new consumer groups, new drinking situations, and new ways of drinking.

Cartwright *et al.* also replicated measures of some alcohol-related problems of individual drinkers. While there was a consistent correlation between consumption and problems in the population in 1965 and in 1974, there was considerable variation between different groups of drinkers within these populations. Groups reporting similar consumption levels reported different numbers of alcohol-related problems. The relationship between consumption and problems seemed to be mediated by two major factors – the drinking pattern and personal characteristics.

When two groups drank the same amount, the group who consumed more *per drinking day* reported more problems. Although this suggests that spreading consumption over more days of the week may carry less risk of experiencing some problems, other studies have shown that high-frequency moderate consumption is more likely than occasional heavy drinking to cause cirrhosis of the liver (Rankin *et al.* 1975). The data, however, show that there are probably relationships between types of drinking patterns and the development of specific alcohol-related problems.

The second intermediary factor between the level of consumption and the prevalence of problems appeared to be personal characteristics which made some people more vulnerable to experiencing problems than other people whose consumption and drinking pattern were the same. Physical, social and psychological factors must explain why one person is more likely than another to experience more problems from the same level of consumption consumed in the same way. For example, young males were more likely than any other demographic group to get into fights after drinking, but this would have to be explained as much in terms of the behaviour of young males as by their alcohol consumption.

This interpretation would comply with other well-known socio-cultural factors which seem to protect certain groups against developing alcohol-related problems while making others vulnerable. For example, Davies (1977) quoted one paper (Glad, 1948) which contrasted the rates of arrest and conviction for inebriety in one American city as 7876 per 100000 people of Irish ethnicity compared with only 27 per 100000 of Jewish ethnicity.

Nevertheless, the intermediary role of drinking patterns and vulnerability factors does not obviate the conclusion that an overall increase in consumption in a population is still likely to lead to more problems in that society. Although we cannot say that if any individual drinks a certain amount a day he will definitely suffer specific problems, we can say that if an individual increases his consumption, he is at a greater risk of experiencing alcohol-related problems. We can also say that his risk

is probably greater if he drinks in certain patterns or if he appears particularly vulnerable to developing problems because of his physical, psychological or social characteristics. The reconceptualized view of the aetiology of alcohol-related problems could be represented diagrammatically as in Fig. 1.

Although the epidemiology of alcoholism is now heading in this more sophisticated direction, the current data still reaffirm the basic principle of alcohol control policy that reducing alcohol availability is likely to decrease consumption, and hence reduce the prevalence of alcohol-related problems. However, the epidemiological advances towards a new theory of the aetiology of alcohol problems also have implications for treatment, as recognized by Davies (1977) in his concluding remarks on the Symposium. He pointed out that dealing with people in terms of the risk associated with their particular drinking pattern and their personal vulnerability would be a much more rational approach to individual treatment. The question would then become not 'is this man an alcoholic or not?', but rather 'what factors operated in his case which made him drink so much and therefore what can I do to help him in the future to deal with those particular factors?'.

Thus the epidemiological challenge to traditional concepts of alcoholism is developing into a rationale for alternative approaches to conceptualization and treatment. To realize the potential of this new direction, there is a need for much closer investigation of the relationships between an individual's consumption pattern, his vulnerability and the development of alcohol-related problems.

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