

Alcohol and Heart Disease

Lecture given at the SMAB conference, Oslo in October 2000, by Hans Olav Fekjær.

During the last two decades, several studies have shown a correlation between a low or moderate alcohol consumption and a reduced mortality from coronary heart disease and stroke. Most have been cohort studies, a limited number have been case-control. Over time, the studies have been refined, taking a larger number of potential confounders into account. Most studies now not only control for age, blood pressure, smoking and body mass index, but also for pre-existing diseases, cholesterol,

And in approximately 80% of the studies, the statistical correlation between a low alcohol consumption and the reduced mortality has been demonstrated. In addition, possible mechanisms to mediate a beneficial effects have been established, although, admittedly, mechanisms in the opposite direction have also been demonstrated.

Has, then, a causal relationship been established once and for all, and do attempts to cast doubt on the existence of a causal relationship qualify for membership in the Flat Earth Society?

A Norwegian expert committee studied the literature rather thoroughly and delivered a report nearly two years ago. The members were 5 university professors, four in medicine plus our leading alcohol researcher, in addition to myself. We concluded that at the present stage of research, the evidence makes it probable that alcohol is a causal factor in establishing the reduced morbidity and mortality among light drinkers. We also stated that further research may strengthen or weaken the interpretation that alcohol plays a causal role.

Why, then, is there room for doubt and too early to draw a final conclusion?

Coronary heart disease and the related disease, stroke induced by thrombosis, is a multi-factorial life style disease. Several life-style factors have been demonstrated rather convincingly to play a role. Still, our knowledge is very incomplete. This is shown that in screening populations, we can identify many individuals who are at risk. We identify them by the known risk factors like cholesterol, smoking etcera. Still, when these cohorts are observed over the next years, the established risk factors only enables us to identify one half or less of those who actually are hit by coronary artery disease. Thus, the shortcomings of our present stage of knowledge are obvious. I would like to quote my hero Frank Sinatra: "How little we know - how much to discover".

The main obstacle for making a definitive conclusion is, in my opinion, not the handful of studies which do not show any J curve for coronary heart disease and neither the somewhat higher number of studies which do not show any J curve for total mortality. A little more problematic is another inconsistency, the fact that levels of consumption

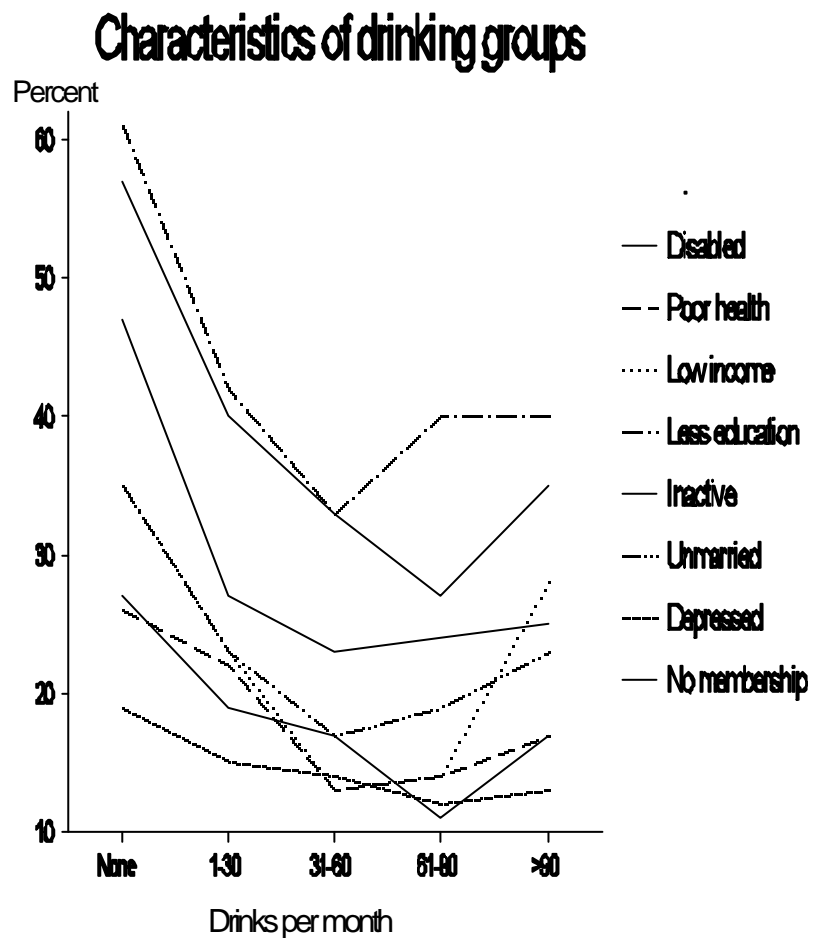
which is at the bottom om the J in some studies, that is, levels which apparently prevent disease in some studies, are on the ascending part of the J, that is, apparently increases the risk in other studies.

The main problem arises from the fact that we have been and probably will remain unable to perform prospective studies based on randomised groups, combined with the fact that the non-drinkers as a group are definitively not recruited by random from the general population, but are deviant in several ways.

One obvious fact is that the non-drinkers are not complying with the prevailing norm. The prevailing norm in the mainly urban areas in the industrialised countries which have been subject to studies, is that normal, socially functioning individuals are expected to drink alcohol and to do so in moderation.

One study has explored the non-drinker group more in detail:

Among our great-grandparents, some were teetotallers based on an ideology or a conviction. Many, perhaps most, prominent and famous Norwegians a hundred years ago were belonged to this kind of non-drinkers. This study demonstrates a non-drinker group which is deviant and less well functioning. They are, on the average, more depressed, more socially isolated, poorer and less educated, more often unmarried and less active. Knowing this, it seems premature to conclude that among their numerous characteristics, it is their abstention from alcohol which alone may explain their extra morbidity or mortality.



Source: Camacho 1987.

Very few of the studies of alcohol's relationship to coronary heart disease or total mortality analyse the non-drinker group in this way. There is, however, a very large body of epidemiological research from the Collaborative Alcohol-Related Longitudinal Project which has been analysed by Kaye Fillmore and her co-workers from several countries. They conclude:

"Across studies, adult male former drinkers are consistently more likely to be heavier smokers, depressed, unemployed, lower SES and to have used marijuana than long-term abstainers. Adult female former drinkers are consistently more likely to be heavier smokers, in poorer health, not religious, and unmarried than long-term abstainers. Both types of abstainers tend to be of lower SES than light drinkers and report poorer health (not consistent). Female abstainers are more likely to be of normal or overweight than light drinkers."

The groups main conclusion was, I quote:

"Characteristics of two groups of abstainers, other than their non-use of alcohol, may confound the associations found between drinking and mortality risk."

In general, we may say that the studies demonstrating a J-shaped relationship have controlled for several confounders of bodily nature, but have largely ignored the psychosocial factors. Does it matter? Coronary heart disease is, after all, a bodily disease, isn't it?

It is certainly a bodily disease. Still, psychosocial factors seem to be independent risk factors. An example is the golden ring on the finger which seems to be made of a magic preventive metal. Being married seems to be an independent risk factor for premature mortality in men. That is, when controlling for all the known risk factors, being married still seems to have a significant independent effect. We do not know which mechanisms are involved, which apparently transfer a psychosocial factor into an effect upon this bodily effect, so again, it is a demonstration of my quote "How little we know - how much to discover". My guess for an explanation is that as we know that marginal social groups have less favourable ratings at the risk factors we know today, they will also probably be less favourable at the risk factors which we have not yet been revealed.

This also applies to other psychosocial factors like depression, social isolation, socio-economic status and education. Socio-economic status and education have been controlled for in a minority of studies, other psychosocial factors in practically none. An exception is the study by Mertens and co-workers, in which the J-shaped curve largely disappeared when correcting for life stressors, social support, social activities og coping responses.

Other factors which are seldom taken into account are diet and physical activity. We might guess that marginal group also are less favourable on these variables.

In my opinion, the studies by Morten Grønbaek and other colleagues in the Copenhagen heart study also should be seen as pointing to the importance of factors related to the

drinker and not to the drink. According to their studies, moderate wine consumption apparently prevent not only heart disease and stroke, but also lung cancer, cancer in the upper digestive tract and fractures. Beer has apparently a mild preventive effect, while spirits largely seem to have a negative effect.

The idea that wine prevents coronary disease is, of course, preferable to teetotallers, as the non-alcoholic ingredients of wine may, of course, be ingested without alcohol. But the literature as a whole does not support the idea that wine has any advantage over beer and spirits. This was stated not only in the review by Rimm, Klatsky and Grobbee in 1996, but also by the review by Cleophas in 1999.

But when Grønbaeks group in Copenhagen have demonstrated J-shaped curves for wine's relationship to diseases for which we have no good explanations for any causal relationship, and demonstrate that wine is apparently beneficial while spirits is apparently harmful, their research may as well be taken as an argument against alcohol's possible healthy effects, indicating that other characteristics of the drinker groups' are more important. And indeed, their study published in the American Journal of Clinical Nutrition i February 1999 demonstrates that wine drinkers have a much healthier diet.

Now, I have described why I still consider the jury to be out, and I agree with, people like the leader of NIAAA and the French minister of health who conclude that it has not yet been **proven** that moderate drinking prevents coronary heart disease. But medical doctors are also human. So when confronted with a large body of research, moderate drinkers, like others, will easily conclude that their own life-style is healthy. And all groups will easily conclude that their opponents are suffering from a personal bias, while I myself observe the reality with an open mind.

That much about the factors which led the Norwegian expert group to formulate a preliminary, not a final conclusion. In the following, I will ignore the reservations and discuss the consequences of considering moderate drinking as a causal preventive factor.

When the report from the Norwegian expert committee was published, our leader had to send corrections to our two largest newspapers, as both of them wrote rather unconditionally that the group recommended alcohol for preventing heart disease. In the same week, the Journal of the American Association published a meta-analysis of acetylsalicylic acid in preventing coronary heart disease, concluding that acetylsalicylic acid reduces the mortality by 32%. This conclusion is, of course, based on better research, as it is based on randomised groups, differing only in the use of acetylsalicylic acid. But while several decades of headline have declared alcohol or wine to be healthy, the better founded research on acetylsalicylic acid has been completely ignored. From a public health point of view, this is, of course, a large problem. And if the group of nice wine-loving Scandinavian doctors who arrange this conference primarily are interested i health, why do they ignore more firmly established preventive measures than alcohol?

Studies which conclude that moderate drinking get a wide publicity. The emphasis on moderation is often ignored, so that headline state that "alcohol prevents heart disease" or "wine is healthy". And the age factor is always ignored.

Age is a prime factor. In 1998, I found that in studies demonstrating a J-shaped curve, the average age during the time of observation varied between 49 years and more than 80 years. In the only, but respectable study of alcohol and mortality between 20 and 40 years of age, the Swedish study of former conscripts, there was a linear relationship between alcohol and mortality. The less alcohol, the lower mortality, and vice versa.

For the age group between 30 and 40 years of age, the mortality from all cardiovascular diseases is (in Norway) 0,15%, that is, neglectable. Between 40 and 50 years of age, the mortality is less than a half percent.

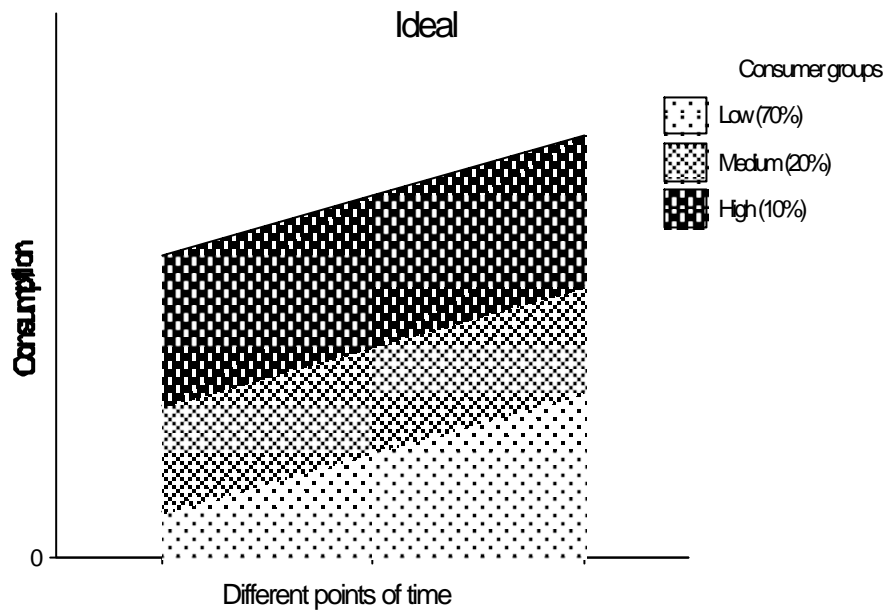
For young adults, alcohol is only a risk factor for health. If moderate drinking is among the several factors which prevents heart disease, at what age should the prevention begin? Early stages of atherosclerosis have been demonstrated in fairly young people. There is, however, evidence that the life-style in the years preceding a possible disease is very decisive. Ex-smokers reduce their risk quite fast after quitting smoking.

If someone of average risk want to use alcohol for preventing heart disease, they should probably wait untill they are 40 or 50 years of age. And alcohol should, of course, not be the first or second or third choice for prevention.

But, at least in Norway, most of the alcohol is consumed by people below 40 years of age. And almost untill 50 years of age, the most important cause of death is sudden unnatural death (accidents, suicide and homicide), for which alcohol is one of the most important risk factors.

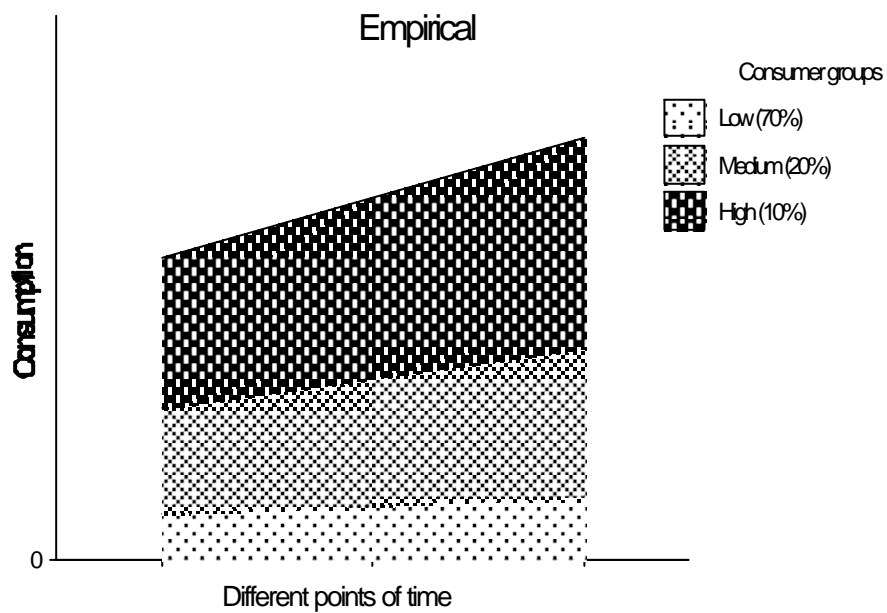
The so-called SMAB wants an increase of moderate drinking. The dream of the alcohol industry, which has paid most of the expenses for this seminar, has always been to selectively increase the consumption among low consumption and low risk groups. I will illustrate this by the following graph:

When consumption is changing....



We know that approximately 10% of the population consume a half of the total consumption. This graph illustrates the dream of changing this distribution of alcohol in the population, with an increased consumption only among low consumption and low risk groups.

When consumption is changing....



But there is a large body of epidemiological research demonstrating that when the total consumption changes, the distribution between consumer groups remain the same. Giving moderation a more prominent position has always been the aim of alcohol education and prevention. And it has never succeeded. The distribution remains the same as it is for almost all consumer goods.

The reason is probably that alcohol is consumed together with other people. In Norway, 9 out of 10 glasses of alcohol are consumed together with others, and studies have demonstrated that a huge mutual influence takes place. Olaf Aasland, a member of the Norwegian group, has even formulated that the proportion of harmful drinkers depends on how much the other drinkers consume. And from an epidemiological point of view, he is right. The idea to foster moderation is very old and the vigorous attempts have always been futile.

And what happens when consumption does increase in a population? Europe's largest epidemiological study, the European Comparative Alcohol Study, is in its final phase. By time series studies, the relationship between total consumption and mortality in the EU countries and Norway has been scrutinized. Most parts of the study have been presented at conferences and the main conclusions will be published in the journal *Addiction* in a couple of months. Some of Europe's most recognized alcohol researchers are responsible for the study. It concludes that an increase in total alcohol consumption increases mortality from accidents, suicide, homicide, liver cirrhosis and - most important - the total mortality. And it does not show that the total alcohol consumption has no influence upon the mortality from cardiovascular disease. Therefore, ladies and gentlemen, I think we should be careful not to jump to conclusion and not over-sell alcohol as a preventive agent.