Alcohol consumption, stroke and public health - an overlooked relation?

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THE PUBLIC HEALTH IMPORTANCE OF STROKE

Each year, approximately 15 million people world-wide suffer a stroke, most of whom are over 40 years of age [1]. Of these strokes, the majority are ischaemic strokes, accounting for more than 80% of the total. Stroke is a disease with relatively high case fatality and is associated with high levels of disability. For instance, in Canada, among those hospitalized for stroke, approximately 20% die and 25% require ongoing care. Less than 50% of stroke patients are living independently in the year following the stroke, and many of them have physical, mental or behavioural changes as a result [2]. Globally, stroke is a leading cause of death and a top 10 contributor to total global burden of disease [3,4]. Alcohol, paradoxically, has been identified as both a risk and a protective factor for this burden, accounting for a net detrimental effect on haemorrhagic stroke (10% of global burden of this disease category) and a net beneficial effect on ischaemic stroke (1.3%) [5,6].

ALCOHOL AS AN OVERLOOKED CONTRIBUTOR TO STROKE

Although alcohol consumption, especially heavy drinking, has been identified as a potential risk factor for stroke for several decades [7], it is often notably absent from discussions on how to reduce the public health burden of stroke (e.g. [8]). Several reasons may account for this phenomenon. First, surely, the seemingly inconsistent results of many epidemiological studies contribute to the absence of alcohol use in these debates [9]. Secondly, alcohol is not generally present in discussions of chronic disease and public health [10–12], more specifically in discussions with respect to cardiovascular disease [13]. To give but one example: the World Health Organization (WHO) fact sheet to the review on Preventing Chronic Diseases [10] explicitly lists only tobacco, unhealthy diet and physical inactivity as risk factors (http://www.who.int/chp/chronic_disease_report/media/information/en/index.html) and dismisses harmful alcohol use with a vague remark, suggesting that the relationships are complex. This is indeed surprising, especially as the WHO Comparative Risk Assessment found that alcohol is one of the five most important risk factors for global burden of disease and that almost 60% of the net global burden of disease attributable to alcohol was due to chronic disease categories such as cancer, neuropsychiatric disease, diabetes, cardiovascular disease and liver cirrhosis [5,6].

FUTURE STEPS

What can be done to improve the current situation, and to paint a clearer picture of alcohol consumption and stroke and of the relationship between alcohol and chronic disease in general? First, we should always differentiate between stroke categories, minimally between ischaemic and haemorrhagic stroke. The most comprehensive meta-analysis on average volume of alcohol consumption and stroke clearly showed different dose–response relationships between the stroke subtypes: for haemorrhagic stroke there was an almost linear dose–response relationship (the more alcohol, the higher the risk of stroke) with no significant protective effect at low levels of consumption, whereas for ischaemic stroke there was a *J*-shaped relationship with a significant stroke-protective effect seen for consumption of up to 24 g/day of alcohol, the equivalent of two drinks/day [14]. However, differentiation into these larger subcategories may not be enough, and there are indications that subtypes such as embolic stroke for ischaemic stroke [15], and subarachnoid versus intracerebral distinction for haemorrhagic stroke [16], may have different risk curves with respect to alcohol. Even subcategories of stroke will be associated with comparatively more burden compared to many other diseases; so looking for causal influencing factors is certainly of public health relevance.

Secondly, we should include patterns of drinking in future analyses on alcohol use and stroke [17], especially irregular occasional heavy drinking occasions. This may seem an old proposal, given that many studies show up in a search of PubMed or other databases with the keywords ‘drinking’, ‘patterns’ and ‘stroke’. However, closer inspection shows that very few of these articles include indicators of irregular heavy drinking. What has become fashionable of late are analyses or re-analyses of studies with volume as derived from usual quantity and usual frequency indicators in a way that both indicators are analysed separately, as well as by beverage type. Both types of analysis, usually carried out exploratorily with no biological underpinning or specific hypothesis, are often labelled as ‘pattern analysis’.

What are the reasons for the suggestion to concentrate on irregular heavy drinking? One good reason is that a major pathway from alcohol to stroke, especially

haemorrhagic stroke, is high blood pressure, and there is some evidence that high blood pressure is affected by irregular heavy drinking occasions [18–20]. Another reason can be seen in the ecological correlations between irregular heavy drinking patterns and stroke rates [21], again especially highly pronounced for haemorrhagic stroke. A large part of the higher alcohol-attributable stroke mortality in Central and Eastern European countries could well be due to (a) the relatively higher prevalence of haemorrhagic stroke in these countries compared to western Europe or North America and (b) to the drinking patterns of irregular heavy drinking [5,22–24]. Finally, some of the effects between irregular heavy drinking occasions and ischaemic heart disease [20,22,23] may well generalize to ischaemic stroke. The risk curves [25] between average volume of drinking or ischaemic heart disease and ischaemic stroke, respectively, certainly appear to be very similar, and both disease categories share risk factors and biological pathways. Similarly, the cardioprotective and the ischaemic stroke-protective effects of regular moderate consumption may have the same underlying mechanisms.

While we certainly admit that the above reasons are not conclusive and that we need better-designed prospective studies on different patterns of drinking, with different subtypes of stroke as a measurable end-point, we hope that our arguments will lead to the initiation of exactly these kinds of studies. From a public health perspective, given the global exposure of alcohol consumption, especially of irregular heavy drinking occasions [5,24], the generation of better knowledge on the relationships between different levels and patterns of drinking as causal factors for different types of stroke seems a priority.

WHAT CAN BE DONE NOW?

In a wider context, it is suggested that the role of alcohol not only in stroke, but in chronic disease in general, should be better acknowledged. While there are always important avenues for new research, there is currently a wealth of information on the causal relationships between alcohol and different chronic disease categories (e.g. [5,18]), which makes it imperative that this research is used to form policy and recommendations in the public arena. Simply citing that the relationships between alcohol and chronic disease are in part complex, and are multi-dimensional for some disease categories including stroke [10,11], is no argument against using the relevant research as an evidence base.

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References

1. Mackay J., Mensah G. A. *The Atlas of Heart Disease and Stroke.* Geneva: World Health Organization; 2004.
2. Hankey G. J., Warlow C. P. Treatment and secondary prevention of stroke: evidence, costs, and effects on individuals and populations. *Lancet* 1999; **354**: 1457–63.
3. Mathers C., Bernard C., Moesgaard Iburg K., Inoue M., Ma Fat D., Shibuya K. *et al. Global Burden of Disease 2002: Data, Sources, and Methods*, revised version. Geneva: World Health Organization; 2004.
4. Lopez A. D., Mathers C. D., Ezzati M., Jamison D. T., Murray D. J. L. *Global Burden of Disease and Risk Factors.* New York/Washington: Oxford University Press and the World Bank; 2006.
5. Rehm J., Room R., Monteiro M., Gmel G., Graham K., Rehn N. *et al.* Alcohol use. In: Ezzati M, Lopez A, Rodgers A, Murray C., editors. *Comparative Quantification of Health Risks. Global and Regional Burden of Disease Attributable to Selected Major Risk Factors*, vol. 1. Geneva: World Health Organization; 2004, p. 959–1109.
6. Rehm J., Room R., Monteiro M., Gmel G., Graham K., Rehn N., *et al.* Alcohol as a risk factor for global burden of disease. *Eur Addict Res* 2003; **9**: 157–64.
7. Gill J., Zezulka A., Shipley M., Gill S., Beevers D. Stroke and alcohol consumption. *N Engl J Med* 1986; **315**: 1041–6.
8. Ding E., Mozaffarian D. Optimal dietary habits for the prevention of stroke. *Semin Neurol* 2006; **26**: 11–23.
9. Mazzaglia G., Britton R., Altmann D. R., Chenet L. Exploring the relationship between alcohol consumption and non-fatal or fatal stroke: a systematic review. *Addiction* 2001; **96**: 1743–56.
10. World Health Organization (WHO). *Preventing Chronic Diseases: a Vital Investment.* WHO Global Report. Geneva: WHO; 2005. Available at: http://www.who.int/chp/chronic_disease_report/media/information/en/index.html (accessed 31 March 2006).
11. Strong K., Mathers C., Leeder S., Beaglehole R. Preventing chronic diseases: how many lives can we save? *Lancet* 2005; **366**: 1578–82.
12. Haydon E., Roerecke M., Giesbrecht N., Rehm J., Kobus-Matthews M. *Chronic Disease in Ontario and Canada: Determinants, Risk Factors, and Prevention Priorities*. Research Report no. 205. Toronto: Centre for Addiction and Mental Health; 2005.
13. Tolonen H., Mahonen M., Asplund K., Rastenyte D., Kuulasmaa K., Vanuzzo D. *et al.* Do trends in population levels of blood pressure and other cardiovascular risk factors explain trends in stroke event rates? Comparisons of 15 populations in 9 countries within the WHO MONICA Stroke Project. *Stroke* 2002; **33**: 2367–75.
14. Reynolds K., Lewis B., Nolen J. D., Kinney G. L., Sathya B., He J. Alcohol consumption and risk of stroke: a meta-analysis. *JAMA* 2003; **289**: 579–88.
15. Mukamal K., Ascherio A., Mittleman M., Conigrave K.,

Camargo C., Kawachi I. *et al.* Alcohol and risk for ischemic stroke in men: the role of drinking patterns and usual beverage. *Ann Intern Med* 2005; **142**: 11–19.
16. Leppala J., Paunio M., Virtamo J., Fogelholm R., Albanes D., Taylor P. *et al.* Alcohol consumption and stroke incidence in male smokers. *Circulation* 1999; **100**: 1214.
17. Hillbom M. Pattern of alcohol drinking and stroke: a comment on Mazzaglia *et al. Addiction* 2002; **97**: 102.
18. Rehm J., Room R., Graham K., Monteiro M., Gmel G., Sempos C. T. The relationship of average volume of alcohol consumption and patterns of drinking to burden of disease—an overview. *Addiction* 2003; **98**: 1209–28.
19. Seppa K., Sillanaukee P. Binge drinking and ambulatory blood pressure. *Hypertension* 1999; **33**: 79–82.
20. Puddey I. B., Rakic V., Dimmitt S. B., Beilin L. J. Influence of pattern of drinking on cardiovascular disease and cardiovascular risk factors—a review. *Addiction* 1999; **94**: 649–63.
21. Sarti C., Rastenyte D., Cepaitis Z., Tuomilehto J. International trends in mortality from stroke, 1968–94. *Stroke* 2000; **31**: 1588–601.
22. McKee M., Britton A. The positive relationship between alcohol and heart disease in eastern Europe: potential physiological mechanisms. *J R Soc Med* 1998; **91**: 402–7.
23. Rehm J., Sempos C. T., Trevisan M. Average volume of alcohol consumption, patterns of drinking and risk of coronary heart disease—a review. *J Cardiovasc Risk* 2003; **10**: 15–20.
24. Rehm J., Rehn N., Room R., Monteiro M., Gmel G., Jernigan D. *et al.* The global distribution of average volume of alcohol consumption and patterns of drinking. *Eur Addict Res* 2003; **9**: 147–56.
25. Rehm J., Greenfield T., Kerr W. Patterns of drinking and mortality from different diseases—an overview. *Contemp Drug Probl* 2006, in press.