LETTER TO THE EDITOR

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Moderate drinking, psychological factors, and cardiovascular protection

Two recent EHJ articles1,2 add to the evidence that moderate alcohol consumption, compared to heavy or non-drinking, has beneficial effect in reducing mortality from ischaemic heart disease. Interesting information is that the effect is independent and cannot be compensated by physical activity.2 The authors1,2 and commentators3 discussed possible explanations including alcohol’s effects on lipoprotein and glucose metabolism, ischaemic myocardium, endothelial function, inflammatory and coagulatory factors, smooth muscle proliferation, and methodologically, ‘the sick quitter bias’.

It has been well-established that negative psychosocial and psychological factors, in particular depression and anxiety syndromes, contribute to the pathogenesis of ischaemic heart disease.5,7 Their effect is comparable with abdominal obesity and hypertension, and proposed pathophysiological mechanisms involve sympathetic nervous system predominance, arrhythmia susceptibility, relative hypercoagulability, and adverse influence on endothelial function and inflammation. Psychological factors can also trigger acute coronary syndromes and cardiac arrhythmias, and have a dose-dependent relationship with the risk of sudden cardiac death.2,5 In that line, we reported a protective effect of anxiety against ventricular arrhythmias.6

Alcohol is an old cure for relieving of emotional distress and fear, often effective in coping with social situations and anxiety.7,9 Besides metabolic and pathophysiologic mechanisms affecting cardiovascular system,1–3 alcohol may have favourable effect on autonomic nervous system and neurocardiac regulation due to its anxiolytic-like and calming properties. Thereby, positive psychological impact of moderate drinking may lead to a relative slowing of progression of atherosclerosis, and the lower rate of fatal coronary events may result from coupled beneficial effect of sympathetic autonomic modulation during acute coronary events, particularly regarding arrhythmias. A support for the latter may be found in the increased heart rate variability observed in wine consumers.10,11 Nevertheless, presently it is not clear whether this is confined only to wine (not to other alcoholic drinks),10,11 women,12 or perhaps is confounded by fish-derived consumption of polyunsaturated fatty acids or other factors.11,13

Epidemiological evidence of the risk lowering effect of moderate alcohol consumption is compelling, but cannot be fully explained by currently proposed biological mechanisms. The hypothesis of possible positive psychological, anxiolytic, or autonomic effects on coronary disease and cardiac death should be considered in future research. However, experience from prior studies, including the two most recent,1,2 suggests that such an evaluation will be difficult because of multiple confounding and methodological issues. In addition to aforementioned factors, the individuality of emotional and pathophysiological response to alcohol (for example associated with personality types) and drinking pattern (total amount, regularity, and binge) also requires further examination. Only additional characterization and stratification of coronary patients according to possible health risks and benefits associated with alcohol consumption will have practical value for clinicians.

References


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