Low cholesterol and violent death

Sir,—
We were most interested in the editorial on low cholesterol and violent death (1a) in which the relationship of triglycerides with hostility was also discussed. There is a positive correlation of apolipoprotein A-IV and triglyceride in human serum (1b) and, although we did not study triglycerides, we found high levels of apoprotein A-IV in the high density lipoproteins of violent offenders in prison when compared with non-institutionalised controls who had similar total cholesterol and similar HDL cholesterol concentrations (2).

We had embarked on the study of lipoproteins and apolipoproteins in plasma in violent offenders because of the finding of Virkkunen (3) of low serum cholesterol concentrations in antisocial personality disorder subjects and because of the excess of violent deaths observed in trials of cholesterol lowering to prevent coronary artery disease (4,5,6). Horrobin (5) made the important point that drugs which increase violent deaths are also likely to be giving rise to lower grade and more general aggression, with consequent unhappiness not only to the person himself, but to those around him also.

Our two groups were well matched for age, height, weight and smoking histories but as there were differences in alcohol consumption and differences in general lifestyle it is important that the two groups did not differ in plasma concentrations of total cholesterol, HDL-C, or VLDL-C. Apoprotein A-IV, like apoprotein E, may be involved in removal of cholesterol from tissues (7) but a CNS function has yet to be defined. By contrast, apoprotein E, which was also raised in the HDL of our violent offenders, is secreted by CNS glial cells (8) and the astrocytes possess receptors capable of recognising apo E. Pitas et al (9) suggested that apo E-containing lipoproteins may be involved in the regulation of cholesterol homeostasis within the brain. Apo E may also be involved in the restoration of the function of damaged neurones (10,11). A major recent advance in the genetics of Alzheimer's disease is the finding that the apolipoprotein E type 4 allele was strongly associated with the late onset form of the disease (12). Study of apolipoprotein E gene polymorphism may therefore be important not only in susceptibility to coronary artery disease (13), but also in Alzheimer's disease and in predisposition to violence.

While our findings can be criticised on the grounds that our controls were not institutionalised and therefore had access to alcohol, access to offenders is difficult to obtain and the ethical problems are considerable. We hope that our preliminary findings, which suggest the potential role of the apolipoproteins in mediating, or marking, some of the ill effects of cholesterol reduction, will not be ignored by researchers with the necessary facilities, and that study of apoprotein changes in primary prevention trials of coronary artery disease will clarify the links between lipid-lowering and violence.

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References