

12% of patients in the non-treatment group. In the Lipid Research Clinical Program study of the patients who died, 30% of those receiving treatment died violent deaths – accident, suicide, or murder – as opposed to 14% in the control group.

Lipids comprise about half the dry matter of the brain; the axon, the myelin sheath, and the synaptosomal membrane all having different proportions of lipid constituents; synaptic vesicles have a relatively high content of phospholipids. It is reasonable to assume, therefore, that anything which upsets the balance of cerebral lipid metabolism could have profound effects on the brain function. Several abnormalities of enzymes required for normal lipid metabolism are known to result in severe mental subnormality, e.g. deficiency of sphingomyelinase results in Niemann Pick disease. Disruption of the structure of the synaptic membrane or synaptic vesicles could disrupt normal function.

It is tempting to speculate how lipid-lowering drugs could affect the brain: cerebral lipids are synthesised in the brain from water-soluble precursors. Any alteration in peripheral lipid ratios are unlikely therefore to affect brain lipid metabolism. A direct effect of the drug itself in the brain could be postulated; however, cholestyramine is not absorbed from the gut. Gemfibrozil is absorbed, although its ability to cross the blood-brain barrier has not been established.

In both studies these results were interpreted as being a chance finding. We find these results most interesting and raise the question as to whether alteration of the ratio of HDL:LDL could be important psychiatrically.

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- FRICK, M. H., ELO, O., HAAPA, K., *et al* (1987) Helsinki Heart Study: primary prevention trial with gemfibrozil in middle-aged men with dyslipidemia. *New England Journal of Medicine*, **317**, 1237–1245.
- LIPID RESEARCH CLINICAL PROGRAM (1984) Lipid Research Clinics Coronary prevention trial results. 1. Reduction in the incidence of coronary heart disease. *Journal of the American Medical Association*, **251**, 331–364.

Tea and Antipsychotics

SIR: Silverstone *et al* (*Journal*, August 1988, **153**, 214–217) suggest that obesity in patients on long-term depot antipsychotics is most likely due to an increase in food intake brought about as a result of a drug-induced stimulant effect on appetite. There may be a possible alternative explanation for this weight gain, the clues to which lie in the patient's consumption of drinks. All antipsychotic medication has quite marked anticholinergic effects and, in particular, produces dry mouth. This leads to an increased consumption of drinks and, in particular, of the favourite British beverage of tea which is often taken with sugar. Simply drinking five more cups of tea per day would lead to an increased intake of 150 calories per day if milk and one sugar were taken. This net increase in calories would lead to an intake of 1050 calories per week, which could explain some of the weight gain. It only requires 7700 calories intake in excess of a balanced state in order to gain 1 kg in weight. On these calculations, 1 kg in weight would be gained every eight weeks. In addition, this effect would be accelerated because of the decreased activity which is one of the (often desired) effects of antipsychotic medication.

I have certainly observed this phenomenon of increased tea consumption in patients on lithium, and wonder whether this may be the underlying mechanism of weight gain with antipsychotics.

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A HUNDRED YEARS AGO

Inebriate Criminal Responsibility

A lecture on Inebriate Criminal Responsibility was delivered to the Society for the Study of Inebriety, at the rooms of the Medical Society of London, on Tuesday last, by the President, Dr Norman Kerr.

The lecturer reviewed the varying criminal procedure of different countries in criminal cases

complicated with inebriety. Germany, Italy, and Switzerland recognised a culpable and inculpable intoxication; America, England, and France did not. Yet in America the confirmed was practically dealt with as a diseased drunkard, and capital punishment was averted by a verdict of "murder of the second

degree". In England, Plowden in the sixteenth century, and Mansfield in the eighteenth century, laid down that drunkenness was no excuse; while Coke went further, and held it to be an aggravation. Since then the rulings had been somewhat contradictory. All our previous criminal proceedings had been founded on our former ignorance of any pathological state preceding intoxication. But the comparatively recent revelation of modern scientific research, that drunkenness was often the effect of disease, had not been without influence on the administration of justice. There had been a number of acquittals in charges of murder during an attack of delirium tremens, and there was a growing disposition in both judge and jury to accept a delusion of this disease as a valid plea for irresponsibility. A general ruling to this effect would be a desideratum. Many drinkers could

not limit their potations. Some were born with a predisposition to intoxication, others with a defective inhibitory power. Such should not be judged by the same standard as those thoroughly sound in body and brain. When drunkenness was but a symptom of insanity, or when permanent insanity was caused by drink, all admitted irresponsibility. But when there was short-lived insanity from alcohol, why should there be responsibility any more than in temporary insanity from any other cause? Dr Kerr suggested a mixed commission of medical and legal experts, and held that in criminal cases complicated with inebriety there should be an investigation of the health history and heredity of the accused.

Reference

The Lancet, 16 March 1889, 541.

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey