

the first phase of their national immunisation days by March 1996.⁷ John *et al* set up mass polio vaccination in Vellore, India, in the 1980s, using pulse or cluster immunisation, in which oral polio vaccine is administered simultaneously on a single day to all children in the specific age group; it greatly decreased the incidence within the town.⁹ A similar programme was conducted successfully around Delhi in 1994. Now it has been taken up at national level, and the first round of a three year programme of a pulse (mass) vaccination programme across India took place in December 1995 and January 1996, successfully covering 75 million children, and it will continue for the next two years.

Oral poliovirus vaccine is used not only for economic reasons but because of its immunological effects: it mimics natural infection and so confers both humoral and intestinal immunity much faster than the inactivated polio vaccine. Moreover, it establishes itself in the alimentary tract, blocking infection with wild virus. This breaks the cycle of infectivity and reduces the incidence of poliovirus infection. An additional advantage is that the vaccine virus spreads by the faeco-oral route to children who have not been vaccinated, extending immunity to the wider population without additional expenditure. Rates of seroconversion are higher when oral polio vaccine is administered during mass campaigns¹⁰—possibly because of person to person spread of vaccine virus—during the cool and dry season.

China and India are implementing modified immunisation strategies that limit the mass campaigns to children under 4 and 3 years of age respectively, and large reductions in vaccine requirement would result if these approaches proved successful.¹¹ Schedules combining oral and inactivated polio vaccine have been advocated, but most cases of polio in endemic countries occur among unimmunised children, and eradication must be achieved primarily by improving vaccine delivery systems rather than by altering immunisation schedules.¹² Such combined schedules may, nevertheless, be especially helpful at the terminal phase of eradication in areas of high prevalence.

The success of mass polio vaccination programmes in tropical countries warrants further effort towards maintaining the cold chain to improve vaccine delivery. Moreover, adequate laboratory infrastructure is required, including the ability to

confirm that cases of acute flaccid paralysis are due to poliovirus so that other causes can be ruled out. An ideal surveillance strategy would detect even a single case.

The recent certification of the interruption of wild poliovirus transmission in the Americas is an important achievement in the effort to eradicate the disease. Together with the worldwide decline in the incidence of poliomyelitis, it makes the goal of global eradication by the end of this century realistic. Nevertheless, concerted efforts, including mass campaigns, will be required, particularly in the developing countries of South East Asia and sub-Saharan Africa. Along with the eradication of smallpox, the eradication of poliomyelitis will represent an appropriate gift from the 20th to the 21st century.² It will also be a noble tribute to the developers of the oral and inactivated polio vaccines, Albert Sabin and Jonas Salk, who both died only in the past three years.

JAGDISH CHANDER
Reader and head

SUBHA SUBRAHMANYAN
Demonstrator

Department of Microbiology,
Government Medical College Hospital,
Chandigarh 160036,
India

- 1 Sabin AB. Oral poliovirus vaccine: history of its development and use and current challenge to eliminate poliomyelitis from the world. *J Infect Dis* 1985; 51:420-36.
- 2 Cockburn WC. The work of the WHO Consultative Group on poliomyelitis vaccines. *Bull WHO* 1988;66:143-54.
- 3 Robertson SE, Chan C, Kim-Farley R, Ward N. Worldwide status of poliomyelitis in 1986, 1987 and 1988 and plans for its global eradication by the year 2000. *World Health Stat Q* 1990;43:80-90.
- 4 Centers for Diseases Control and Prevention. Progress toward global poliomyelitis eradication 1985-1994. *MMWR* 1995;44:273-81.
- 5 Kumar R, Kumar V. Poliomyelitis control by annual immunisation campaigns with oral poliovirus vaccine in a rural area of India. *Trop Geogr Med* 1991;43:215-9.
- 6 Biellik RJ, Bueno H, Olive JM, de Quadros CA. Poliomyelitis case confirmation: characteristics for use by national eradication programmes. *Bull WHO* 1992;70:79-84.
- 7 Centers for Diseases Control and Prevention. Progress toward poliomyelitis eradication—South East Asia Region, 1988-1994. *MMWR* 1995;44:791-801.
- 8 De Quadros CA, Andrus JK, Olive JM, de Macedo CG, Henderson DA. Polio eradication from the Western Hemisphere. *Annu Rev Public Health* 1992;13:239-52.
- 9 John TJ, Pandian R, Gadomaski A, Steinhoff M, John M, Ray M. Control of poliomyelitis by pulse immunisation in Vellore, India. *BMJ* 1983;286:31-2.
- 10 Richardson G, Linkins R, Earnes M, *et al*. Immunogenicity of oral poliovirus vaccine (OPV) given in mass campaigns versus routine immunisation programs. In: *Proceedings of the 33rd Interscience conference on Antimicrobial Agents and Chemotherapy*. Washington, DC: American Society for Microbiology, 1993.
- 11 Hull HF, Ward NA, Hull BP, Miltien JB, de Quadros CA. Paralytic poliomyelitis: seasoned strategies, disappearing disease. *Lancet* 1994;343:1331-7.
- 12 Patriarca PA, Foegle WH, Swartz TA. Progress in polio eradication. *Lancet* 1993;342:1461-4.

The cardioprotective effects of moderate alcohol consumption

No real evidence exists that wine is better than beer or spirits

See p 1200

The recent British government report *Sensible Drinking*¹ followed a scientific consensus in recognising the cardioprotective effect of moderate alcohol consumption in men aged over 40 and postmenopausal women. Two points of controversy are the government's consequent effective increase in the sensible drinking levels² and whether specific alcoholic beverages carry more benefit than others.

Developed countries with higher wine consumption tend to have lower mortality from coronary heart disease^{3,4}; the association is weaker or absent for beer and spirits. This contrast has led to the hypothesis that wine has a special cardioprotective effect. The finding, however, may be an artefact resulting from higher total alcohol consumption in wine drinking countries than in those where beer and spirits are drunk more often. Comparisons between countries are often misleading—they also suggest that the effect of alcohol consumption on male mortality from coronary heart disease is three times that of smoking³—and should be viewed with great caution.

The recent paper by Rimm *et al* provides an overview of 10 cohort studies which related coronary heart disease in individuals to their consumptions of particular beverages.⁵ The results are remarkably contradictory. For example, consumption of spirits was associated with significantly increased mortality from coronary heart disease in the Copenhagen city heart study and a significantly decreased incidence of coronary heart disease in the health professionals follow up study. This evidence does not point to a greater cardioprotective effect of any specific beverage. Rather, the consistency of the cardioprotective effect of alcohol consumption across populations with different beverage preferences⁶ suggests that alcohol per se is the major factor. The different socioeconomic or behavioural characteristics of wine, beer, and spirits drinkers in different countries may explain the diversity of these results. An alternative explanation lies in the cultural drinking patterns associated with different beverages—for example, spirits might be consumed more in “binges” in some European countries and more regularly in the United States.⁷

The French paradox refers to the finding that French consumption of animal fat is near to the average of other developed countries while French mortality from coronary heart disease is only one third of the average. Comparison between countries seemed to show that the high French consumption of wine could be an explanation,⁴ but the results of Rimm *et al* now contradict this. Nevertheless, alcohol consumption per se is an implausible explanation of a national threefold risk reduction since in individuals its effect is only a 25-50% reduction in mortality from coronary heart disease.⁶

The recent paper by Hein *et al* proposed an alternative explanation for the French paradox.⁷ In Copenhagen the incidence of coronary heart disease was inversely related to alcohol consumption for men in the top fifth of the low density lipoprotein cholesterol distribution (above 5.25 mmol/l) but was only weakly related in others. Thus the incidence of coronary heart disease was about three times higher in non-drinking men with high low density lipoprotein cholesterol concentrations than in all other men. This result can, however, explain only a small part of the lower French mortality from coronary heart disease, since non-drinkers are in general a small minority (10% in the United Kingdom). It may also be a chance finding; many other studies with data on low density lipoprotein cholesterol have related coronary heart disease to alcohol consumption. None of these studies has reported differences in this relation between low density lipoprotein subgroups and this may mean that there were no differences. These studies should be reanalysed to confirm or refute the findings of Hein *et al*.

Alcohol consumption is believed to reduce the incidence of coronary heart disease partly by increasing concentrations of high density lipoprotein cholesterol. The study by Paunio *et al* in this issue (p 1200) further investigates the inverse relation between mortality from coronary heart disease and high density lipoprotein cholesterol concentrations in male Finnish smokers and supports a previous finding of an upturn in the highest group.⁸ Put differently, men with high density lipoprotein cholesterol concentrations of 1.62-1.75 mmol/l have one third the mortality from coronary heart disease of men in adjoining high density lipoprotein groups, but on the basis of

only four deaths. It would be unwise to place much credence on this result without assessments of significance⁹ in this and other studies.

The report *Sensible Drinking* based its recommendations for both men and women on the statement, "The evidence we have studied for men of all ages identifies a band of minimal mortality associated with a weekly consumption of between about 7 and 28 units a week."¹¹ This summary of the scientific evidence may disproportionately reflect two studies^{10,11} published in 1994. Other studies,¹² a preliminary quantitative overview,¹³ and a more extensive overview currently in progress suggest that the minimum mortality occurs below 21 units per week for men. This casts serious doubt on the appropriateness of the upwards revision of the sensible limits. Reliable conclusions about alcohol epidemiology require pooling of comparable analyses from all available cohort studies.

IAN R WHITE

Lecturer in medical statistics

Medical Statistics Unit,
London School of Hygiene and Tropical Medicine,
London WC1E 7HT

- 1 Inter-Departmental Working Group. *Sensible drinking*. London: Department of Health, 1995.
- 2 Edwards G. Sensible drinking. *BMJ* 1996;312:1.
- 3 St Leger AS, Cochrane AL, Moore F. Factors associated with cardiac mortality in developed countries with particular reference to the consumption of wine. *Lancet* 1979;i:1017-20.
- 4 Criqui MH, Ringel BL. Does diet or alcohol explain the French paradox? *Lancet* 1994;344:1719-23.
- 5 Rimm EB, Klatsky A, Grobbee D, Stampfer MJ. Review of moderate alcohol consumption and reduced risk of coronary heart disease: is the effect due to beer, wine, or spirits? *BMJ* 1996;312:731-6.
- 6 Marmot M, Brunner E. Alcohol and cardiovascular disease: the status of the U shaped curve. *BMJ* 1991;303:565-8.
- 7 Hein HO, Suadicani P, Gyntelberg F. Alcohol consumption, serum low density lipoprotein cholesterol concentration, and risk of ischaemic heart disease: six year follow up in the Copenhagen male study. *BMJ* 1996;312:736-41.
- 8 Paunio M, Virtamo J, Gref CG, Heinson OP. Serum high density lipoprotein cholesterol, alcohol, and coronary mortality in male smokers. *BMJ* 1996;312:1200-3.
- 9 Goetghebuer EJT, Pocock SJ. Detection and estimation of J-shaped risk-response relationships. *J R Statist Soc A* 1995;158:107-21.
- 10 Gronbaek M, Deis A, Sorensen TIA, Becker U, Johnsen KB, Muller C, *et al*. Influence of sex, age, body mass index, and smoking on alcohol intake and mortality. *BMJ* 1994;308:302-6.
- 11 Doll R, Peto R, Hall E, Wheatley K, Gray R. Mortality in relation to consumption of alcohol: 13 years of observations on male British doctors. *BMJ* 1994;309:911-8.
- 12 Boffetta P, Garfinkel L. Alcohol drinking and mortality among men enrolled in an American cancer society prospective study. *Epidemiology* 1990;1:342-8.
- 13 White IR. Alcohol consumption and all-cause mortality. *Int J Epidemiol* 1996;25:217-8.

Seriously deficient professional performance

Needs defining as that which puts the public at risk

The Medical (Professional Performance) Act, which became law last November, has given powers to the General Medical Council (GMC) to take action when it seems that a doctor's professional performance may be seriously deficient. The council has been concerned for some time about its inability to intervene when consistently poor care seems to result not from misconduct but from a doctor's inadequate knowledge and skills, and it sees the new powers as essential if it is to fulfil its duty to protect the public. There has also been external pressure for a means of addressing poor performance among doctors, and if the task had not been given to the GMC an alternative mechanism would almost certainly have been demanded.

Consultations with medical organisations, patients' representatives, and politicians before enactment of the legislation showed wide support for the proposed measure, but doctors are clearly worried that the resulting "performance procedures" could prove inquisitorial and oppressive.¹ The GMC now has the task of devising assessment procedures that are reliable, fair, and enjoy public confidence but avoid being cumbersome or onerous. This is a complex and difficult task, given the wide variation in types of

medical work and the very different skills which doctors practising in different disciplines may need.

An important principle which the GMC should establish at the outset is that the new procedures will deal only with *seriously* deficient performance. Although concerned to promote high standards of medical practice, the GMC's fundamental task is to maintain a register of doctors in whom the public may reasonably have confidence. The performance procedures must thus operate only when there are grounds for believing that a doctor's professional performance has fallen below a standard justifying such confidence, bringing into question his or her continued unrestricted registration.

The council has not, however, reached agreement on a definition of seriously deficient professional performance, and there are diverging views over the amount of detail which such a definition should contain. A detailed definition might offer clarity and objectivity, as well as some measure of protection against GMC decisions being challenged in the courts, but it would be restrictive. No definition could specify every form or category of seriously deficient performance. The adoption of one which attempted to do so might impose an unhealthy