Animal studies have shown that, in experimentally induced renal impairment, progression to end stage renal failure is accompanied by increased glomerulosclerosis, seemingly precipitated by an increased haemodynamic load on the glomeruli. This in turn stimulates cytokine release from mesangial cells and consequent collagen production.

If a low protein diet is given the cycle is interrupted. Afferent arteriolar dilatation and glomerular haemodynamic stress is much less, and progressive glomerulosclerosis is delayed or absent. Adequate control of blood pressure complements these benefits. As Narins and Cortez point out, the findings of the modified diet in renal disease study do not necessarily conflict with this suggested mechanism.[5] Possibly the requirements of entry into a long term clinical study, which demand informed and disciplined attention to dietary intake, select out patients already using advice on diet and blood pressure to the limit so that they have already accrued any possible benefit. The slower than predicted fall in glomerular filtration rate in control patients in the modified diet in renal disease study is consistent with such a suggestion. Nevertheless, had there been a decisive benefit from protein restriction a disciplined study such as this would have shown it.

In animal studies benefit has been obtained consistently when dietary intervention occurred immediately after the desired degree of renal impairment had been achieved experimentally. Such early intervention seems impractical in humans. Renal failure occurs relatively silently in many patients, so the public health measures that would be necessary to detect all such patients early enough in their condition to allow months to years of dietary modification are daunting.

For doctors the complex series of variables imposed by underlying renal disease, metabolic and other complications of uraemia, comorbid conditions, and the problems of compliance mean that intervention has to be tailored to each patient. Furthermore, across the world and certainly in both the United States and Britain many patients are first seen by nephrologists when they are at or close to the point of needing dialysis and so are not seen early enough in the course of their disease for dietary intervention to have whatever benefit is possible. Thus the effects of strict dietary control on the number of patients entering renal replacement programmes is likely to be small.

If protein restriction is undertaken then nitrogen and energy intake must be balanced to ensure that nutrition is maintained. Adequate energy intake is at least as important as any other aspect of the diet; modest protein restriction (0·6 g of protein/kg body weight/day) minimises the overall load of protein products that the kidney must excrete and is a feasible goal. At least one third of the protein should be high in essential amino acids. Once the glomerular filtration rate has reached some 20 ml/min stricter protein restriction does not seem to confer the benefits in terms of preserving the glomerular filtration rate that animal studies have found; currently the emphasis should be on maintaining nutritional balance and minimising the amount of protein waste product that is produced. Furthermore, modestly reducing the proportion of protein low in essential amino acids seems prudent.

As protein intake is reduced so is the intake of phosphates; these are excreted mainly through the kidney, so the rise in plasma phosphate concentration is minimised. This has benefits in terms of ensuring that calcium phosphate is not laid down in crystallised form in body tissues and may also reduce secretion of parathyroid hormone.

Because some lipid subfractions may damage the glomerulus directly, dietary control of lipid intake may turn out to be important. (This is currently being researched.) If so, dietary intervention may yet prove to be a valuable tool in changing the rate at which renal function falls in progressive disease; its place in halting such a fall is much less certain.

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1 Klahr S, Schreiner G, Ichikawa I. The progression of renal disease. *N Engl J Med* 1988;318:1657-66.
2 Brenner BM. Nephron adaptation to renal injury or ablation. *Am J Physiol* 1985;249:F324-37.
3 Fouque D, Laville M, Boissel JP, Chifflet R, Labeeuw M, Zech PY. Controlled low protein diets in chronic renal insufficiency: meta-analysis. *BMJ* 1992;304:216-20.
4 Klhar S, Andrew S, Levey MD, Beck GJ, Caggiula W, Hunsicker L, *et al.* The effects of dietary protein restriction and blood pressure control on the progression of chronic renal disease. *N Engl J Med* 1994;330:877-84.
5 Narins RG, Cortes P. The role of dietary protein restriction in progressive azotemia. *N Engl J Med* 1994;330:929-30.

The new measles campaign

Immunisation should prevent an epidemic predicted by modelling

See also letters on p 1161

The national measles and rubella campaign that begins next month is one of the most ambitious vaccination initiatives that Britain has undertaken. The aim is to vaccinate 95% of the seven million schoolchildren aged between 5 and 16 within one month and so to prevent an epidemic of measles that would otherwise be likely to occur early next year.[1 2] The novel feature of this campaign is that it is meant to prevent an epidemic: the more usual response is to wait for an outbreak to occur before doing anything. This preventive action is likely to be far more cost effective than attempts to curtail spread once the chains of transmission are established. Experience in North America has shown that, despite the adoption of prompt and aggressive measures to control outbreaks, transmission is rarely interrupted.[3 4]

The campaign is based on comprehensive epidemiological surveillance data including serological studies, number of cases notified and confirmed, rates of complications and deaths, and immunisation coverage.[2 5 6] These data have been used in two independent mathematical models: both have predicted a high probability of a major resurgence of measles, with the greatest burden of cases in children in secondary schools and a considerable number in children in primary schools.[1]

The Department of Health is convinced that the cost of preventing such an epidemic through a mass campaign will be considerably less than the direct and indirect costs of the predicted epidemic. By including rubella vaccine the campaign is expected to hasten the elimination of the congenital rubella syndrome by reducing the pool of

susceptibility in adolescent boys, in whom the virus continues to circulate.[7]

Surveillance has shown that increasing numbers of cases of measles are already occurring in schools, confirming the probability of an epidemic if action is not taken promptly.[2] Late last year and early this year several health boards in the west of Scotland saw a large increase in measles—mainly in secondary schoolchildren—which resulted in 138 admissions to one infectious disease unit alone (P Christie, personal communication). If applied to England and Wales the incidence in the Scottish outbreak would give rise to the 100 000-200 000 cases predicted by the mathematical models. The five deaths among the 10 000 cases that occurred during a recent outbreak in schoolchildren in Quebec were further evidence of the severity of measles in older age groups and confirmed the mortality predictions of the modellers.[8]

The resurgence of measles in Britain is largely the result of poor vaccine coverage in the past, but over a tenth of cases occur in children who have been vaccinated.[2] Vaccine failure is known to sustain transmission in populations with high vaccine coverage,[9] and this is the rationale for including all schoolchildren in the campaign irrespective of whether they have been vaccinated against measles previously. The next logical step will be a recommendation of a two dose schedule—a strategy being adopted by an increasing number of countries which like Britain are seeking to eliminate measles.[9] Several countries that have achieved high coverage with a single dose have experienced epidemics after "honeymoon" periods of low incidence.[9] Such epidemics are attributable to an accumulation of susceptible subjects, both unvaccinated children and those in whom vaccination has failed, and are triggered when their number reaches a critical threshold. There is therefore a great epidemiological advantage in introducing a second dose in the wake of a campaign to maintain the number of susceptible subjects well below this threshold.[1] The simplest strategy would be to give a second dose at school entry—when boosters of the diphtheria-tetanus and oral polio vaccines are given—but whether this strategy would be successful in reaching the 7-8% of children who miss the first dose is unknown.[10] Periodic mass campaigns might be a more effective way of reaching this group, but relying on them without a guarantee that resources would be available when required would be risky. Speculation on whether this approach is a serious option in Britain must await the outcome of the present campaign.

The campaign approach for delivering measles vaccine has not been tried before in an industrialised country but has been used successfully in the Caribbean and Central and South America, where measles is now at an all time low.[11] In Cuba transmission of measles seems to have been interrupted after a massive campaign in 1989 that achieved over 95% coverage of children aged 1-14.[11] It will be interesting to see whether the immunisation services in Britain can match this performance. Whatever the operational outcome, the comprehensive surveillance systems in place (which now include routine salivary diagnosis in all suspected cases[2]) will allow the epidemiological consequences of the campaign to be assessed accurately.

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1 Babad HR, Nokes DJ, Gay NJ, Miller E, Morgan-Capner P, Anderson RM. Predicting the impact of measles vaccination in England and Wales: model validation and analysis of policy options. *Epidemiol Infect* (in press).
2 Ramsay M, Gay N, Miller E, Rush M, White J, Morgan-Capner P, *et al.* The epidemiology of measles in England and Wales: rationale for the national vaccination campaign. *CDR Review* (in press).
3 Wassilak SGF, Orenstein WA, Strickland PL, Butler CA, Bart KJ. Continuing measles transmission in students despite a school-based outbreak control program. *Am J Epidemiol* 1985;122:208-17.
4 Davis RM, Whitman ED, Orenstein WA, Preblud SR, Markowitz LE, Hinman AR. A persistent outbreak of measles despite appropriate prevention and control measures. *Am J Epidemiol* 1987;126:438-49.
5 Brown D, Ramsay M, Richards A, Miller E. Salivary diagnosis of measles: a study of notified cases in the United Kingdom, 1991-93. *BMJ* 1994;308:1015-7.
6 Morgan-Capner P, Wright J, Miller CL, Miller E. Surveillance of antibody to measles, and rubella by age. *BMJ* 1988;297:770-2.
7 Miller E, Tookey PA, Morgan-Capner P, Hesketh L, Brown DWG, Waight PA, *et al.* Rubella surveillance to June 1994: third joint report from the PHLS and the national congenital rubella surveillance programme. *CDR Review* (in press).
8 McLean ME, Carter AO. Measles in Canada - 1989. *Canada Diseases Weekly Report* 1990; 16-42:213-8.
9 Cutts F, Markowitz L. Successes and failures in measles control. *J Infect Dis* (in press).
10 White J, Leon S, Ramsay MEB. COVER (cover of vaccination evaluated rapidly) 31 *CDR Review* 1994;4:R129-30.
11 Measles elimination in the Americas. *Bulletin Pan Am Health Organisation* 1992;26:271-4.

The eradication of gonorrhoea

Can be achieved

The continued global survival of *Neisseria gonorrhoeae* is inevitable because of its immunobiology,[1] the seeming lack of natural or acquired immunity, human sexual behaviour, and the frequency of asymptomatic infection. Nevertheless, endemic gonorrhoea could be eradicated from Britain in the 1990s. Sweden has achieved this public health goal, and many other developed countries report dramatic reductions in incidence.[2] In Britain reported cases of gonorrhoea have declined every year since 1977, with a particularly big decrease in 1987. Many health districts have already achieved the nationally set target for 1995 of fewer than 49 cases per 100 000 population aged 15-64.[3] Geographical variation in the incidence of gonorrhoea in England is, however, considerable, with some urban areas still substantially above this target.[4]

Has any one factor been decisive in recent successes in controlling gonorrhoea? Certainly, radical innovations in treatment cannot take the credit. Effective drugs have been available throughout the rise and fall in the incidence of gonococcal infection during the 1960s, '70s, and '80s. Effective treatment is, however, a key component in control and has required some major changes in the past 30 years. Even before the emergence of penicillinase-producing *N gonorrhoeae* in 1976, increasing resistance to penicillin had been widely reported. The reduced sensitivity to penicillin was associated with failure of treatment but did not prove a major problem, as was once feared.[5] Higher doses of penicillin coupled with probenecid re-established high rates of clinical cure.

The appearance and subsequent rapid spread of penicillinase-producing *N gonorrhoeae* demanded an alternative strategy. In sharp contrast to the reported high prevalence of this strain in some parts of the world (notably Africa and South East Asia), its incidence in Britain has remained