The incidence of rhesus D immunisation has been relatively static since 1973; as yet, there has been no demonstrable effect from the introduction in part of the region in 1986 of routine antenatal prophylaxis. We thus fear that the trend of the past 18 years is likely to continue.

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AUTHORS' REPLY,—MacKenzie and colleagues' presumption about our figures is correct. We thought our method of ascertainment was made clear at the start of the "methods and results" section of our paper[1]; a similar procedure has been used for all our reports on deaths from rhesus haemolytic disease since 1977.[2]

We have always appreciated that deaths before 28 weeks were underreported because the Office of Population Censuses and Surveys registers deaths and stillbirths from 28 weeks only. However, the underreporting of these deaths will have gone back many years, long before the introduction of anti-D prophylaxis, and is a constant feature; our figures relate to neonatal deaths and stillbirths from haemolytic disease of the newborn after postnatal anti-D was introduced about 1970.

We were aware of the problem of immunisation during pregnancy rather than at delivery (not necessarily resulting in a dead baby), but Bowman, Bowman and Pollock, and Tovey *et al* suppressed most of these cases.[3-5] However, Dovey, whom we quote in our letter[6] (not Tovey as MacKenzie *et al* seem to suppose) found a static immunisation rate in Yorkshire, as have the workers in Oxford. Admittedly Bowman and Pollock[4] give a much bigger dose than that used in Yorkshire, but we cannot understand why the Tovey regimen, which we believe is being followed in Oxford, is not as successful there as it was in Yorkshire. MacKenzie *et al* show a histogram giving the incidence of sensitisation for the whole of the Oxford region. Could we know the findings in those districts of the Oxford region where the antenatal trial is actually taking place?

A further point in MacKenzie *et al*'s letter needs clarification. Since D antibodies do not cause intrauterine death before about 18 weeks at the earliest, at what stage of pregnancy did the 23 therapeutic abortions performed in Oxford in 1988-90 "because of rhesus disease" occur? Do MacKenzie *et al* mean that the patient or her obstetrician did not want to run the risk of having another seriously affected baby?

We are grateful to Professor P L Mollison and Dr D Lee for advice on some aspects of this reply.

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What counts as cot death?

SIR,—Recent comment on the incidence of cot death makes it essential to agree what counts as a cot death.[1] The term, first used in 1954 by Barratt—"an apparently healthy infant is unexpectedly found dead in its sleeping quarters"—originally included deaths later explained at postmortem examination.[2]

In 1965 Carpenter and Shaddick narrowed the definition to "those cases in which the information available does not reveal the cause or causes of death."[3] This corresponded closely with the definition of the term sudden infant death syndrome proposed by Beckwith in 1969: "The sudden death of any infant or young child, which is unexpected by history, and in which a thorough post mortem examination fails to demonstrate an adequate cause of death."[4] This diagnosis is reached by exclusion of explained deaths.

The unsatisfactory situation of the 1950s and '60s, when many unexplained infant deaths were attributed to a respiratory cause,[3] was recognised by the inclusion in the eighth revision of the ICD (in 1968) of a category for sudden death (cause unknown)—code 795.

Since 1971 the registrar general and the Coroners' Society of England and Wales have accepted sudden or unexpected death in infancy syndrome as a natural, registrable cause of death, and the Office of Population Censuses and Surveys (OPCS) has identified sudden infant death when there is any mention of sudden or unexpected death in infancy, cot death, or such a term in the death certificate. These figures are published every two years in *OPCS Monitor DH3*, usually incorrectly headed sudden infant death syndrome.

In 1979 the ninth revision of the ICD included sudden infant death syndrome (code 798.0). In some districts, however, coroners or pathologists rarely use this as a cause of death but follow Emery and Weatherall's recommendation that a specific cause should be given,[5] mentioning also "unexpected" when this is clinically appropriate. Such deaths are counted by OPCS as sudden infant deaths. The figures for the sudden infant death syndrome (code 798.0), published annually in *OPCS Monitor DH2*, however, comprise deaths in which the syndrome or sudden infant death or cot death is the sole cause given in the death certificate and are therefore an underestimate. VS3 mortality statistics for regions and districts, which R R Gordon used in his table,[6] give figures for category XVI—symptoms, signs, and ill defined conditions (ICD numbers 780-799).

For monitoring purposes in England and Wales two statistics may be used. Firstly, any mention of sudden infant death may be used: the numbers (and rates/1000 live births) from birth to 1 year are 1593(2.3), 1326(1.9), and 1193(1.7) for 1988, 1989, and 1990 respectively. Secondly, the sudden infant death syndrome (ICD 798.0) given as the sole cause of death may be used; since 1986 only postneonatal figures have been published, whereas about 5% of cases occur in the first month of life. The numbers (and rate/1000 live births) for the syndrome are 1419(2.1), 1190(1.7), and 1079(1.5) for 1988, 1989, and 1990 respectively.

We recommend that doctors, pathologists, and coroners should mention in the death certificate if an infant death was clinically unexpected, whatever the cause, and that the figures for any mention of sudden infant death should be used for monitoring cot deaths. The OPCS should publish these figures annually by region and district for England and Wales.

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Treatment of back and neck complaints

SIR,—Manipulation usually takes only a moment or two and yet the median duration of therapy in the "manipulative" treatment group in the study by Bart W Koes and colleagues was 40 minutes.[1] This would seem to imply that much of the time was in fact taken up with the mobilisation procedures.

That the therapists were permitted to modify the regimens makes it even more difficult to be sure what can be extrapolated from such group comparisons.

It is interesting, however, that after the experience of conducting this large study the authors should in effect conclude that the most pressing research requirement is to find ways of reliably distinguishing "specific" sorts of low back pain from among the numerous patients who present with "non-specific low back pain."

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Ozone depletion and skin cancer

SIR,—The report on the alarming levels of chlorofluorocarbons (CFCs) over North America and Europe in recent months[1] included reference to the prediction from the United Nations environment programme[2] that depletion of stratospheric ozone by chemical reactions involving the degradation products of CFCs will lead to a rise in the incidence of skin cancers as a consequence of increased levels of solar ultraviolet radiation at the earth's surface. Implicit in these estimates is that behaviour and time spent outdoors remain unchanged in populations at risk.

Unlike agricultural and marine ecosystems, which are also at risk from the potential effects of increased ultraviolet radiation,[3] humans have the opportunity to modify their behaviour and so their exposure. By combining a behavioural model of human exposure to solar ultraviolet radiation with total ozone trends for United Kingdom latitudes obtained from satellite data[4] and the expected increase in terrestrial ultraviolet radiation conse-

quent on ozone depletion,[5] I estimate that the dose received by British people over the next 20 years will be about 6% higher than if stratospheric ozone was to remain at current levels. However, simple sun protection measures can readily modify these estimates. For example, staying indoors for one hour around midday during the period May to August can more than offset the expected increase in ambient solar ultraviolet radiation. Wearing a wide brimmed hat every day of a two week summer holiday, with no other changes to behaviour, can achieve the same reduction in exposure. Greater changes in behaviour, such as wearing a hat whenever outdoors at weekends during the summer, can lead to substantial reductions in cumulative exposure to the face and consequently in the risk of developing skin cancer.

These calculations show that by encouraging people to adopt minor changes to behaviour it should be possible to maintain, or even reduce, exposure of body sites to those levels currently encountered despite the expected increase in ambient solar ultraviolet radiation. It is to be hoped that public awareness about potential environmental and health effects of ozone depletion will achieve these changes, which could lead to a reduction—rather than the anticipated increase—in skin cancer incidence.

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Facilitating prevention in primary care

SIR,—I share David Mant's disappointment that primary care practitioners can be persuaded to adopt preventive interventions in the absence of convincing evidence of efficacy, acceptability, and cost effectiveness.[1] His view that it is as easy to facilitate the uptake of unproved interventions as interventions of proved preventive value, however, is largely unsupported by published reports.

I recently completed a structured review to analyse the impact of strategies directed at practitioners that had been designed to promote screening for cervical cancer, a worthwhile preventive test. I included only studies of postgraduates in outpatient clinics or general practice and excluded studies without a concurrent control group. I analysed reported changes in the rate of Papanicolaou testing after the intervention compared with before.

Of 14 studies that met the criteria for the review, only four showed a significant positive effect on screening for cervical cancer. Effective interventions included computer generated prompts attached to medical records, the introduction of a practice nurse, and attaching partially completed request forms for smear tests to medical records. Seven studies reported no significant change in the rate of screening after interventions such as giving reading lists, audit with feedback, and, again, attaching reminders to the medical record with or without concurrent reminders directed at patients. Unexpectedly, three studies showed a significant negative effect on screening. The interventions concerned were participation in a quality assurance programme[2]; introducing computer generated prompts attached to medical records, particularly when combined with a monthly audit providing negative feedback about screening rates[3]; and introducing prompts attached to medical records (but without sending reminders to patients concurrently).[4] A recently published study adds another non-significant result to this review.[5]

Given the conflicting and disappointing nature of these findings, I and a colleague are now examining the extent to which they are explained by inattention to adult learning theory, inadequate statistical power, failure to overcome inadequate undergraduate education in prevention, or a combination of these.

Like Mant, I despair of the limited resources available to evaluate the effectiveness of preventive interventions before they are widely advocated. My review suggests, however, that showing that a screening test is effective is insufficient to guarantee its uptake by clinicians. We must continue to develop and evaluate multifaceted, sustainable, and individualised strategies to disseminate preventive interventions that are worth doing. Only then will we have a rationale to underpin our efforts to improve preventive care through professional education and reform of health services.

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SIR,—As the trio responsible for the development and evaluation of the Oxford "facilitator" model,[1,2] we read the findings of Allen J Dietrich and colleagues in the United States and Jill Cockburn and colleagues in Australia, as well as the accompanying editorial by David Mant, with special interest.[3-5]

An important feature of our model is the provision of continuing assistance to the practice team by the facilitator. This may partly explain the difference in outcome in the two studies reported. The facilitator in the American study, in which preventive services were improved, was closely analogous to ours, visiting the practice three times over three months and "providing additional assistance as needed." In the Australian study, on the other hand, the educational facilitator visited the practices twice only to instruct the practice in the use of the quit smoking intervention kit, with no offer or provision of additional assistance. As we reported, we regard the provision of continuing advice and support as an important feature of successful facilitation.[2]

We agree with David Mant about the need to confine facilitation to interventions of proved effectiveness. But, as pointed out by Nick Black in the same issue, uncertainty about the effectiveness of interventions applies to most medical practice.[6] The facilitator approach offers an opportunity to steer general practice in the direction of scientifically validated activities rather than, as his analogy with the runaway train may seem to suggest, to encourage behaviour that causes disaster. Maintaining his analogy, we see the facilitator, rather than releasing the brake on the runaway train, as shifting the points on the track to enable validated clinical practice to proceed in a systematic way; without facilitation, clinical practice sometimes resembles Brownian movement. We join Mant and Black in urging critical evaluation of many general practice activities and urge the development and funding of the research base which is necessary for this.

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Care of asthma in general practice

SIR,—The stated aim of Cedrick R Martys's paper was to ascertain whether the asthma clinic improved care for his patients with asthma.[1] This question is vitally important to the future of the care of asthma in general practice, but caution should be exercised before the author's conclusion that "objective improvement in patients' asthma could not be detected" is accepted.

The conventional clinical audit cycle starts with measuring data against predefined standards. These data are then analysed and followed by appropriate intervention and re-audit or completion of the first cycle. The first audit established that only 15% of patients had had a measurement of peak flow recorded in the previous year (24 of 61 who had been "clinically reviewed"). Although there was strong evidence for an increase in the proportion of patients who had had at least one measurement of peak flow recorded during the past year, the 60% achieved post-clinic fell far short of the agreed standard of 100%. Analysis of the first audit before the clinic protocol was devised might have helped.

We are not told who ran the clinic. Was it a trained asthma nurse?

Aspects of the protocol are open to question: we should take advantage of the availability of prescribable peak flow meters to enable patients to monitor their acute attacks and therefore decide when their steroids are no longer required.[2-4]

The absence of a definition of asthma and the inadequate description of the methodology make the study difficult for other general practitioners to duplicate. How were the 161 known asthmatic patients diagnosed in 1989? Does the increased proportion of asthmatic patients entered in the computer problem list (from 58% to 98%) signify a true increase in the prevalence of asthma in this practice? Alternatively, did the asthma clinic result in 77 new or previously undiagnosed asthmatic patients being recognised (238 minus 161)?

Labelled asthmatic patients are known to be appropriately treated,[5-7] yet the audit did not attempt to identify the proportion of patients prescribed prophylactic treatment before and after the introduction of the clinic.

Finally, the outcome measures used in auditing