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.

I Z MACKENZIE
P J BOWELL
M SELINGER

Oxford Rhesus Therapy Unit,
John Radcliffe Hospital,
Oxford OX3 9DU

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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.

RUTH M HUSSEY

Department of Public Health,
University of Liverpool,
PO Box 147, Liverpool L69 3BX

CYRIL A CLARKE

Department of Genetics and Microbiology,
University of Liverpool

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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.

SYLVIA R LIMERICK

Foundation for the Study of Infant Deaths,
London SW1X 8QB

ANGELA GARDNER

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

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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."

B J SWEETMAN

Department of Rheumatology,
Morriston Hospital,
Swansea SA6 6NL

1 Koes BW, Bouter LM, van Mameren H, Essers AHM, Verstegen GMJR, Hofhuizen DM, *et al*. Randomised clinical trial of manipulative therapy and physiotherapy for persistent back and neck complaints: results of one year follow up. *BMJ* 1992;**304**:601-5. (7 March.)

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-