Authors' reply

EDITOR,—D M Tappin has three criticisms of our article: that it is impossible to separate the effects of breast feeding and socioeconomic status, that the control babies were inappropriately chosen, and that the sample was an inadequate size.

Tappin states correctly that socioeconomic status is associated with breast feeding, but it is not perfectly correlated. In Britain in 1990, 86% of women categorised as social class I breast fed compared with 48% of women categorised as "unclassified" (registrar general's classification of husband's or partner's occupation).¹ It is therefore possible to analyse the effect of breast feeding on sudden infant death having allowed for socioeconomic status. Whether one should adjust for the socioeconomic status is a matter of judgment. This is why we presented the odds ratios for being bottle fed before and after adjustment for employment status (the proxy we used for socioeconomic status).

Secondly, we are puzzled by Tappin's claim that the selection of control babies was biased as the health visitor's caseload would have included all infants in a geographical area. The use of controls matched by neighbourhood would not have underestimated the effect of being bottle fed as the matching was taken into account in the analysis.

Tappin's final point was discussed in the paper. We stated that our results were consistent with an odds ratio for sudden death as high as the upper limit of the confidence interval and that larger studies might be able to distinguish a small, independent effect of being bottle fed. It would have been irresponsible not to publish such findings solely because they were not significant at the 5% level as this would have led to publication bias.²

In answer to J A Walker-Smith, the type of feed was categorised as breast, bottle, or mixed, according to age. No details were collected on the type of milk. This information, however, will soon be available from the confidential inquiry into sudden death in infancy, which is in progress in three regions.

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Sudden infant death in France

EDITOR,—Claudia Court and Alexander Dorozynski both state that the incidence of sudden infant death is still rising in France.^{1,2} They seem to be unaware of the data we obtain every year at the National Institute of Health and Medical Research.

After being steady from 1987 to 1990 the rate of the sudden infant death syndrome fell from 1.93/1000 in 1991 to 1.74/1000 in 1992. The provisional figure for 1993 is 1.6/1000. For the past 20 years all other causes of infant death have decreased every year, except in 1988 (figure).

Trends in the sudden infant death syndrome and other causes of infant death, France, 1974-93 (data for 1993 are provisional)

Secondly, in France no studies are done about ethnic groups since civil registration data do not include ethnic origin. So nobody could say that "the incidence of cot death is about the same among all... ethnic groups,"² or the reverse.

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Cot mattresses and the sudden infant death syndrome

EDITOR,—P J Fleming and colleagues' editorial on the possibility of a relation between the fire retardants, biocides, and plasticisers in cot mattresses and sudden infant death should help medical professionals understand how the sudden infant death syndrome may be caused by gases generated from mattresses by micro-organisms¹; arsine from wallpaper similarly caused deaths of infants a century ago.²

Scopulariopsis brevicaulis develops through warmth and perspiration on cot mattresses and can convert phosphorus, arsenic, and antimony compounds into toxic trihydride gases, but activity is low for several months; the risk is lowest for firstborn infants, who are most likely to have new mattresses, and with reused mattresses the risk becomes important only after several weeks of fungal reactivation.^{2,3} Poisoning first causes headache and irritation, with dislodgement of bedding by stronger infants, so that risk decreases with age; hyperthermia from fever or overwrapping increases the generation of gas, and analgesics suppress warning headaches.

Emphasis on antimony in the editorial and in the television programmes to which the editorial refers is unfortunate as arsenic and phosphorus are also hazardous; arsenic is rarely found, but phosphorus is common. The normal low antimony concentration in tissue permits chemical detection of stibine poisoning, but phosphine poisoning is not detectable. The editorial criticises the analysis of antimony in hair for the television programmes, but many infants had high antimony concentrations while their mothers had low concentrations, confirming exposure in the infants' environment alone. Antimony is usually found in homes only as a fire retardant in polyvinyl chloride and can transfer to infants only as a gas.

Statistics published by the Office of Population

Censuses and Surveys for England and Wales show a reduction in the rate of the sudden infant death syndrome of about 40% after the "back to sleep" campaign in November 1991—the same decrease that occurred after similar interventions in other countries—but the rate had already decreased by about 30% over several years before the campaign started.⁴ The decrease followed recommendations for changes in the manufacture and care of cot mattresses.

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Sampling for analysing blood gas pressures

Arterial samples are the best

EDITOR,—I cannot agree with Khavar Dar and colleagues' conclusion that capillary sampling should be used for analysing blood gas pressures in routine clinical practice.¹ Tissue stabs yield a mixture of true capillary, arteriolar, and venular blood: the similarity of the sample to arterial blood depends almost entirely on the degree of "arterialisation" achieved by warming or rubbing the sample site. At worst the sample is similar to venous blood, and this is particularly so with vasoconstriction.²

Although the authors found that for the partial pressure of oxygen the mean capillary value was within 0.09 kPa of the arterial value, they do not report what the actual arterial values and differences were for each subject or whether their subjects were breathing added oxygen. These are important omissions since in the presence of arterial hypoxaemia the difference between arterial and venous values of partial pressure of oxygen is less (thus making capillary or venous sampling inherently more accurate) than it is when the arterial partial pressure of oxygen is high. The reason for this is the sigmoid shape of the haemoglobin dissociation curve. Fifty four of the 55 patients described in Dar and colleagues' study were admitted urgently to hospital with conditions associated with severe hypoxaemia; it might be predicted that as these patients' conditions improved and they became less hypoxic the difference between capillary and arterial values of partial pressure of oxygen increased.

I am also uncertain whether the authors' method lends itself readily to the repeated sampling essential in managing asthma and controlled oxygen treatment in chronic airways disease. The study provides no evidence that repeated stabs to the ear lobe are less painful than repeated arterial puncture. Repeated stabs would almost certainly be more painful than insertion of an arterial cannula under local anaesthesia on a single occasion, from which repeated samples may painlessly be taken. Interestingly, as many patients expressed no preference between arterial puncture under local anaesthesia and ear lobe sampling as expressed a preference for ear lobe sampling.¹ Repeated "arterialised" venous samples may be obtained from a cannula in a dorsal hand vein: for partial pressure of carbon dioxide the agreement with arterial values is generally within 0.13-0.26 kPa.³ I have found, however, that even in the presence of a