von Kries *et al* (p 199) and Ansell *et al* (p 204), meet that need and are therefore of great importance. Von Kries *et al* performed a fully designed case-control study that included 272 children with leukaemia or cancer and 334 controls matched for age and sex.¹³ Records of vitamin K administration in the neonatal period were critically assessed and showed conclusively that there was no association between the intramuscular administration of vitamin K and subsequent development of childhood cancer.

How can the difference between the findings of Golding *et al* and von Kries *et al* be explained? Von Kries *et al* avoided some of the design flaws in the Golding study: instead of unmatched controls, von Kries *et al* matched for age and sex; instead of using only two hospitals (and over 90% of cases and controls from one hospital), von Kries *et al*'s study was population based. Also, von Kries *et al* consider that they had more accurate and complete data on whether vitamin K had been given or not.

In the study by Ansell *et al*, cases were born at three hospitals in England and included 109 infants with leukaemia and 218 controls matched for age, sex, and hospital of birth.¹⁴ The authors found no association between childhood leukaemia and intramuscular injection of vitamin K at birth. They note that several studies are weakened by the fact that they infer information on vitamin K administration at birth from hospital policies.^{4 10 12}

The case for using oral vitamin K to prevent early haemorrhagic disease of the newborn is sound. However, it has been shown to be ineffective in preventing late disease. Although giving breast fed infants repeated oral doses of vitamin K in the first two months of life may prevent late disease, compliance for such a regimen has been shown to be poor, leaving infants at risk.⁹

Because haemorrhagic disease of the newborn can be life threatening but is preventable, the studies by von Kries *et al*

and Ansell *et al* should allay our fears and doubts about the dangers of administering intramuscular vitamin K immediately after birth. It seems that haemorrhagic disease of the newborn can be completely eradicated without the threat of leukaemia and childhood cancer as a side effect.

ALVIN ZIPURSKY
Emeritus professor

Department of Pediatrics,
University of Toronto,
Toronto, Ontario,
Canada M5G 1X8

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Sudden infant death syndrome: after the "back to sleep" campaign

Further declines may come from reducing maternal smoking

See pp 191, 195

The incidence of sudden infant death syndrome in England and Wales fell by two thirds between 1989 and 1993. The decline in deaths followed the "back to sleep" health education campaign (started in 1991), which advised parents to place babies on their back or side to sleep, to avoid overheating and smoky environments, and to contact a doctor if their baby was unwell.¹ Similar campaigns were conducted in Australia, New Zealand, and several other countries, with a decrease in the incidence of sudden infant death syndrome of 50% or more.² In Tasmania a within-cohort analysis of more than 30 factors that might have contributed to the decline in incidence found that 70% of the decline could be attributed to the reduction in the proportion of infants who usually slept prone.³ Much of the research effort is now focused on understanding the mechanisms through which the prone position exerts its effect and on identifying the causal pathway for sudden infant death in infants who do not sleep in the prone position.

Two papers in this week's *BMJ* present the findings from a two year case-control study conducted in England after the "back to sleep" campaign was launched.^{4 5} The study used a similar design to that used in the 1990 Avon and Somerset study, which identified the prone sleeping position and overheating as risk factors for sudden infant death.⁶ Its strengths include a standardised and detailed system for case ascertainment over a large population, a short interval between death and interview, high response rates, and an emphasis on data collection for potentially amenable risk factors. With the rapid decline in the incidence of sudden infant death syndrome in most countries, this will be one of the largest

studies of sudden infant death in a post-intervention population. However, recall bias must be considered in a case-control study on a population that was well informed about the risk factors for sudden infant death syndrome.

The first paper by Fleming *et al* (p 191) makes the important observation that the risk of sudden infant death syndrome was higher in infants placed to sleep on their side than those placed on their back.⁴ The highest risk was for infants reportedly placed on their side and found prone. The New Zealand case-control study also found the risk to be higher in babies placed on their side rather than on their back.⁷ While neither study was able to demonstrate conclusively whether this was due to differences in reporting or in infant care practices, the likelihood that infants sleeping on their side are at greater risk is supported by evidence that the side position is unstable, with a proportion of infants changing to the prone position during sleep.⁸ As the effect of sleeping position on the risk of sudden infant death syndrome depends on features of the infant and the infant's environment such as swaddling,⁹ Fleming *et al* could usefully analyse their data on sleeping position with regard to effect modification as well as confounding.

Bed sharing has been a controversial area, particularly since the New Zealand case-control study found that bed sharing was a risk factor among infants whose mothers smoked.¹⁰ Infants who shared their parents' bed all night during the last sleep were four times more likely to suffer sudden infant death after adjusting for confounding factors, and the risk was potentiated if the mother smoked.⁴ Even among the infants of non-smoking mothers, bed sharing doubled the risk of sudden

infant death syndrome. This increase in risk was not significant, but this may have been due to a type II statistical error in view of the small numbers in the subgroup.

With only a small proportion of infants sleeping prone after the "back to sleep" campaign, Blair *et al* in the second paper (p 195) found parental smoking to be the major contributing factor to sudden infant death syndrome, accounting for 62% of cases.⁵ This estimate is based on an unusual composite measure which records a positive exposure if the mother smoked during pregnancy or the father smokes in the household. Previous work supports the view that prenatal and postnatal smoking by the mother and probably postnatal smoking by others increase the risk of sudden infant death syndrome.¹¹⁻¹³ The findings of relative risks greater than three and a dose-response relation in these studies support the inference that this is causal. However, lingering doubts remain that the association may be due, in part, to the association of parental smoking with other infant care practices, which might vary by socioeconomic status.

Blair *et al* have not helped resolve to this issue, and the separate contributions to risk of prenatal and postnatal exposure to tobacco smoke from various sources are difficult to ascertain from their analysis. Importantly, the paper does not report estimates of risk due to maternal postnatal smoking after adjustment for maternal smoking during pregnancy. The dose-response patterns for postnatal exposure (table 4) are reported without adjustment for socioeconomic status, and the multivariate models used are not described in detail. Further analysis could provide an estimate of the population risk attributable to smoking, which could be more readily

evaluated and may contribute more to the already strong case for avoiding cigarette smoking during pregnancy or in the vicinity of babies.

TERENCE DWYER

Director

ANNE-LOUISE PONSONBY

Research fellow

Menzies Centre for Population Health Research,
University of Tasmania,
Hobart 7001,
Australia

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Inappropriate treatment for dysentery

See p 205

Perverse incentives and inadequate information may accelerate antibiotic resistance

Keeping abreast of changing patterns of microbial resistance presents a formidable challenge to all those prescribing antibiotics. When national standards are developed for treating infectious diseases, recommendations must be transmitted to local practitioners and drug sellers. Even if there is an effective communications network this process takes time. Given that many recommended treatments are based on outdated texts or "common practices," it is perhaps not surprising that practice changes slowly, if at all. Common practices can often be traced to the influence of one or two influential physicians whose ideas have had a disproportionate effect on the literature. For example, the overuse of metronidazole and furazolidone to treat shigella infections in Bangladesh (and many other countries) almost certainly reflects overdiagnosis of amoebiasis that 25 years ago was mistakenly considered to be the commonest cause of bloody diarrhoea.

The short report by Ronsmans *et al* in this week's *BMJ* (p 205) reveals the extent of inappropriate treatment of dysentery in Bangladesh and raises issues that are relevant to both developing and developed countries.¹ The authors interviewed four groups of medical practitioners in Dhaka to assess how much their treatment of dysentery differed from the World Health Organisation's guidelines and in what way their knowledge was influenced by their training. Correct treatment for dysentery in children was recommended by about 10% of drug dispensers and 45% of doctors (with or without postgraduate training) and medical students. The most common treatment errors were in the use of multiple and inappropriate antimicrobial drugs and in the failure to recommend use of oral rehydration solution.

Incorrect use of antibiotics has consequences beyond the wellbeing of the patient. Inappropriate drugs cost more because adequate treatment can often be achieved with fewer and less expensive drugs. These extra costs are typically borne by the patient's family, because few people in Bangladesh have health insurance. In addition, inappropriate use of antibiotics may accelerate the development of drug resistant strains of shigella wherever it occurs. For example, southern Africa is in the midst of an outbreak of *Shigella dysenteriae* type 1 that is sensitive only to nalidixic acid and other quinolones.

The authors and others refer to incorrect treatment as irrational rather than inappropriate. The term irrational is unfortunate because practitioners' choice of treatment may be rational given their knowledge of the subject or incentives to prescribe. The most up to date information available to health providers comes from drug company representatives, whose main aim is to sell their products. Textbooks are uncommon and usually outdated, and peer reviewed journals are rarely available. Obtaining correct information is not easy. Practitioners who are trying to increase their income may resort to polypharmacy or prescribe drugs that maximise profits. Physicians have suggested that they are only trying to maintain their customer base by responding to customer demands when they overprescribe or use the latest, most expensive drugs.

What can be done to change this behaviour? Education programmes are necessary, but how the information is presented and reinforced is critical. In an attempt to change prescribing patterns for treating diarrhoea in a government hospital in Mexico, Gutierrez *et al* used two techniques.² Firstly, they