Key messages

- Differential survival between smokers and non-smokers with the e4 allele of the apolipoprotein E gene has been proposed to explain the inverse relation between history of cigarette smoking and Alzheimer's disease
- This study shows that the inverse association between smoking history and early onset Alzheimer's disease cannot be explained by a shift in frequency of the apolipoprotein e4 allele
- The inverse relation was significant only in subjects with a family history of dementia who carry the e4 allele
- Our study suggests that clinical trials with nicotine or nicotine derivatives have the greatest chance of success in patients with familial Alzheimer's disease who carry the apolipoprotein e4 allele

protected from Alzheimer's disease by continuing to smoke. The present study has no direct relevance, therefore, for the prevention of disease. Our findings may have implications, however, for the understanding of the pathogenesis and therapeutic strategies in Alzheimer's disease. Our study suggests that clinical trials with nicotine or nicotine derivatives have the greatest chance of success in patients with Alzheimer's disease who have a family history of dementia and carry the apolipoprotein e4 allele.

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Trends in rates and seasonal distribution of sudden infant deaths in England and Wales, 1988-92

E A Gilman, K K Cheng, H R Winter, R Scragg

Department of Public Health and Epidemiology, Institute of Public and Environmental Health, Medical School, University of Birmingham, Birmingham B15 2TT
E A Gilman, *research fellow*
K K Cheng, *senior lecturer*
H R Winter, *senior registrar*

Department of Community Health, School of Medicine, University of Auckland, Private Bag, Auckland, New Zealand
R Scragg, *senior lecturer*

Correspondence to:
Dr Gilman.

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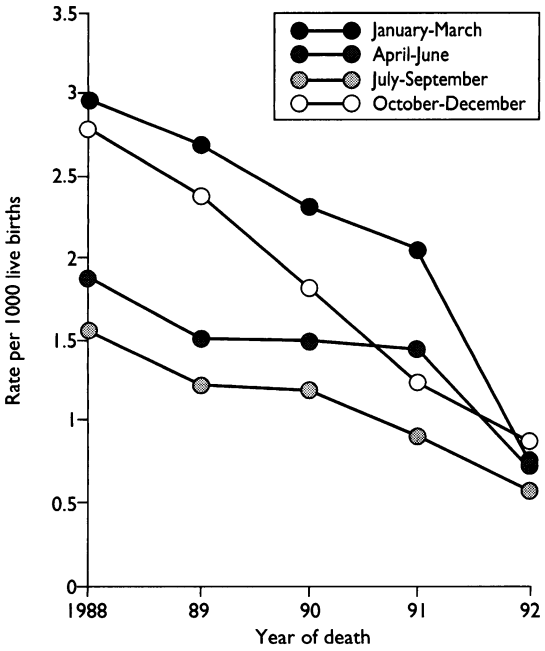
In the United Kingdom around half of all deaths between 1 month and 1 year of age are sudden—that is, cot death, the sudden infant death syndrome, or a similar description is recorded on the death certificate with or without any other cause. Epidemiological features suggest that infections, sleeping prone, exposure to cigarette smoke, and overheating of infants, particularly in the winter, may be associated with sudden infant deaths.[1,2] A campaign launched in October 1991 in the United Kingdom encouraged parents to avoid putting infants to sleep on their fronts, smoking near them, and overheating them. A similar campaign in New Zealand was followed by a fall in sudden infant death rates, and a noticeable decrease in the winter peak.[3] We studied the trends in rates and the seasonal distribution of sudden infant deaths in England and Wales, 1988-92.

Methods and results

We used published statistics.[4] They showed that sudden infant death rates rose more or less continuously from 1971 to a peak of 2·30 deaths per 1000 live births in 1988. Rates then fell steadily to 1·44 in 1991 and abruptly to 0·70 in 1992.

The seasonal distribution is shown in the figure. Linear regression of each quarter's rates for 1988-91 showed that all quarters except the second (April-June) had a significant negative slope (b=-0·13, 95% confidence interval -0·38 to 0·12). For deaths in July-September b=-0·19 (-0·04 to -0·35), particularly



Sudden infant deaths in England and Wales in each quarter from 1988 to 1992

steep slopes being seen in October-December (b=−0·52, −0·39 to −0·65) and January-March (b=−0·31, −0·24 to −0·39). Differences between slopes were significant (P<0·001). Between 1991 and 1992 the rate in April-June fell sharply for the first time since 1989 (by half), while the decline in January-March steepened abruptly. All quarterly rates converged over the period, until in 1992 there was little difference between them.

Comment

Although sudden infant death rates were falling before the campaign, the rates fell dramatically in 1992. Large falls occurred in 1992 in the first and second quarters, those immediately after the campaign's launch, while sudden infant death rates in July-September and October-December were within the range expected on the basis of the slopes during 1988-91.

In Avon the proportion of infants laid to sleep on their backs increased from 5% in September 1991 to 23% in December 1991 and 40% in June 1992.[2] If the campaign had caused national changes in infant care practices which persisted throughout 1992, the larger drop in sudden infant death rates in the first two quarters of 1992 could indicate that the interaction between prone sleeping position and the underlying pathogenic mechanisms that it affects (overheating or respiratory obstruction, possibly exacerbated by infection) is more important in the early part of the year. Alternatively, the lack of a noticeable drop in rates in the last two quarters of 1992 could indicate a waning of the campaign's impact on infant care practices. Less likely, the observed trend might have been dependent on other factors.

Although it is satisfying to see such dramatic reductions, monitoring of the infant care practices featured in the campaign is needed to identify the effect of the campaign on the trend in sudden infant death. Continued monitoring of rates, and the seasonal distribution of sudden infant deaths will also be interesting.

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Neonatal vitamin K prophylaxis in the British Isles: current practice and trends

J S Barton, J H Tripp, A W McNinch

Department of Child Health, Royal Devon and Exeter Hospital, Exeter EX2 5DW
J S Barton, *senior paediatric registrar*
J H Tripp, *senior lecturer in child health*
A W McNinch, *consultant paediatrician*

Correspondence to:
Dr J S Barton.

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In 1992 an unexpected association was reported between intramuscular vitamin K prophylaxis in the neonatal period and later childhood cancer.[1] Although unconfirmed, the report obliged paediatricians to review their prophylaxis policies and led the British Paediatric Association to recommend the routine use of oral vitamin K in all healthy neonates, reserving intramuscular prophylaxis for those at greatest risk of vitamin K deficiency bleeding.[2] We have documented changes in prophylaxis policies so that any consequences can be assessed in terms of either the incidence of vitamin K deficiency bleeding or any possible side effects.

Materials, methods, and results

In October 1993 a questionnaire was sent to every neonatal unit listed by the Neonatal Nurses' Association requesting information on annual delivery rate, details of vitamin K prophylaxis policy, and any policy changes since January 1992. Results were compared with those in a similar survey in 1988.[3] Replies were received from 253 (98%) units, representing 747 000 deliveries a year (90% of births in the British Isles). Trends in practice since 1970 include an increase in the proportion of babies given any prophylaxis (from 23% to 98%) and an increase in the proportion given vitamin K by mouth (from none to 58%). The proportion of babies routinely given intramuscular vitamin K increased from 23% in 1970 to 58% in 1982 and 1988, falling again to 38% in 1993 (table).

No consensus exists regarding the dose or frequency of administration of oral vitamin K, the total dose varying by a factor of 65. The commonest regimens recommend three doses of 0·5-1·0 mg over the first six weeks, but 37 neonatal units (15% of births) did not recommend any doses beyond 7 days of age, despite reports of late failure of single oral dose prophylaxis.[4]

Three quarters (73%) of the neonatal units surveyed had amended their vitamin K policy since January 1992, including 39 units which still recommended parenteral prophylaxis for all but offered oral prophylaxis if parental consent for injection was withheld.

Changes in routine vitamin K prophylaxis in British Isles since 1970. Figures are percentages

Year	Type of study	Intramuscular	Oral	Route not specified	Nil
1970	Prospective cohort of 16 000 babies[5]	23	–	–	77
1982	Retrospective national survey (% of units)[3]	58	5	–	37
1988	Contemporary national survey (% of births)[3]	57	30	–	13
1993	Contemporary national survey (% of births; present report)	38	58	2	2

Note: 1982 data are expressed as percentage of neonatal units surveyed, as retrospective information on annual live birth rate for each unit was not obtained. Prospective data from 1988,[3] however, were similar whether expressed as percentage of births per year or percentage of neonatal units.