Sir—Blair and colleagues report 128 (1.10 per 1000 births) sudden unexpected infant deaths, including 101 (0.87) classified as SIDS in 1 year. These rates are very low compared with those of only 5 years earlier. In England and Wales sudden infant death rates peaked at 2.30 per 1000 births in 1988 and then progressively fell to 1.94, 1.70, 1.44, 0.77, and 0.66, respectively, in 1989-93.[1] The largest annual decrease was 0.67 between 1991 and 1992, in response to the Department of Health's Back to Sleep campaign recommending avoidance of the prone sleeping position. However, this campaign was not the only reason for the decrease, which had started more than 2 years earlier after my recommendations that a new mattress is used for every new infant and that an old mattress should be wrapped in polythene.[2] The decrease has continued since 1992 because of changes in cot mattress materials.

My recommendations were prompted by my findings in 1988-89 that cot mattresses become infected by a micro-organism that is usually harmless but which can convert phosphorus, arsenic, and antimony biocides and fire retardants in mattress materials into toxic gaseous phosphines, arsines, and stibines.[3] All infants sleeping on mattresses containing phosphorus, arsenic or antimony compounds are exposed to these gases when natural microbial infection is well developed, but whether babies are unaffected, have headache or illness, or die depends on other factors. The most important of these factors is mattress temperature, which affects the rate of gas generation, such that infants that are hyperthermic through overwrapping or fever are most at risk—especially those sleeping in the prone position who are most severely exposed to accumulations on the mattress surface of heavy stibine (antimony trihydride) gas generated from antimony trioxide fire retardant in PVC. Avoidance of the prone position reduces this risk, but manufacturers have progressively eliminated this component, although organophosphates generating lighter phosphine (phosphorus trihydride) are still in use in some coverings and fillings. The SIDS risk has therefore reduced, but so has the effectiveness of the Back to Sleep campaign, which was specific to the stibine risk.

Unfortunately Blair and colleagues do not take these factors into account in their investigations, attempting to relate SIDS risk to mattress type and ignoring phosphorus, arsenic, and antimony contents. Antimony is rare in the usual infant environment. Exposure to stibines from mattress PVC can be detected by postmortem analyses - high antimony concentrations in hair and liver indicating chronic exposure, but high values in blood and lung indicating acute exposure. Phosphine poisoning cannot be detected in this way because of the natural high phosphorus concentrations in tissue.

It was thus inappropriate for Blair and colleagues to claim that they have investigated my hypothesis. There is obviously a danger that unsubstantiated criticism of my hypothesis will discourage the precautions that have been so effective in reducing the SIDS rate in the British Isles, although all precautions will become irrelevant when the critical elements have been eliminated from cot mattresses. I recommend maximum concentrations of 0.05% for phosphorus and 0.01% for arsenic and antimony in mattresses. If manufacturers wish to include biocides or fire retardants, many suitable compounds exist that do not contain these elements.

B A Richardson
Penarth Research International Limited, Castle Emplacement, PO Box 142, St Peter Port, Guernsey, Channel Islands

1 Office of Population Censuses & Surveys. OPCS Monitor DH3 91/1 (1991) and DH3 94/1. London: HM Stationery Office, 1994.
2 Richardson BA. Cot mattress biodeterioration and SIDS. Lancet 1990; 337: 670.
3 Richardson BA. Sudden infant death syndrome; a possible primary cause. Forensic Sci Soc 1994; 34: 199-204.