

Chapter 12

Conclusions and Recommendations

Conclusions

12.1 In this report we have considered in detail the toxic gas hypothesis. We have concentrated on PVC and other plastic cot mattress covers widely used in the UK and have not investigated natural materials such as sheepskin. Figure 12.1 summarises the key steps in our investigations and our findings. These are outlined in more detail below.

Step 1

12.2 We investigated the fungal infestation of the covers and fillings of cot mattresses. Mould contamination was found on almost all mattresses but the nature and extent of contamination on mattress covers from SIDS infants was similar to that on control covers. *Scopulariopsis brevicaulis*, however, was not found on a single mattress from a SIDS infant but was found on a small proportion of control covers.

12.3 Richardson's experiments were repeated in an independent laboratory with his cooperation and his presence at key stages. He agreed that the organisms grown from the mattress samples were identical in colonial appearance to those he had observed (when examined by eye). When observed under a microscope, however, these organisms were found to be bacteria. They were formally identified as a mixed population of *Bacillus* spp. commonly found in the domestic environment.

12.4 We conclude, therefore, that cot mattress contamination with the fungus is rare and no more common in SIDS mattresses than in other used mattresses, and that, in his experiments, Richardson had mistaken bacteria for the fungus.

Step 2

12.5 Richardson considered the test paper colour changes he had observed as evidence of production of phosphine, arsine, stibine, and their lower alkyl derivatives. The repeat experiments went further in that the test papers were chemically analysed. The colour changes occurred in the

presence of bacterial growth on the medium, whether or not there was any mattress material present. Chemical analysis of the test papers showed clearly that the colour changes were not due to deposits of phosphorus, arsenic or antimony but appeared to have been caused by sulphur-containing gases. Subsequent investigations have identified these sulphur-containing gases as dimethylsulphide and dimethyldisulphide resulting from the action of bacteria on sulphur-containing compounds in the medium.

12.6 Richardson's response to the results obtained from the replication of his studies was to claim that the culture medium used contained too much nitrogen and was contaminated with excessive sulphur. The culture medium employed in the repeat tests, however, was that which Richardson himself had recommended and used in his own experiments and the sulphur content of the soya flour used in the media was identical.

12.7 We were aware that trimethylarsenic could be biogenerated from arsenic compounds, and that, under anaerobic conditions, methylated antimony compounds and phosphine may be generated by unidentified microorganisms in environmental samples such as sewage sludge and river sediments. Small amounts of phosphine may also be formed in the human gastrointestinal tract. Our own replication of Richardson's experiments did not substantiate his claims. We, nonetheless, considered it essential to undertake further research to establish whether there were any other laboratory conditions in which biovolatilisation of antimony and phosphorus compounds could be achieved, and, if so, to identify the gases and consider whether the conditions were relevant to an infant's cot.

12.8 A number of independent research groups found no evidence of volatile phosphorus, arsenic or antimony compounds being produced when mattress samples were incubated with *S. brevicaulis* under temperature and aerobic conditions relevant to the cot. New research has identified certain experimental conditions when antimony from added antimony compounds (but not from antimony incorporated in mattress material) can be biovolatilised by added *S. brevicaulis*. This produced trimethylantimony but not stibine. One research group has shown that if the antimony incorporated in PVC was first extracted from a sufficient amount of PVC mattress cover by heating at very high temperatures (conditions which would destroy most microorganisms including any naturally occurring *S. brevicaulis* present on PVC mattresses), very small amounts of antimony were biovolatilised by the added *S. brevicaulis*.

12.9 No research group has found evidence for biovolatilised phosphine or trimethylphosphine by *S. brevicaulis* under any of the laboratory conditions tested.

12.10 We conclude, therefore, that there is no evidence for biovolatilisation of phosphorus, arsenic and antimony from PVC cot mattress samples by *S. brevicaulis*, under conditions relevant to an infant's cot. We have, however, identified laboratory conditions, wholly unlike those that could occur in an infant's cot, in which antimony is biovolatilised to trimethylantimony, not stibine.

Step 3

12.11 A critical component of the hypothesis is that death from SIDS is due to anticholinesterase activity of phosphine, arsine and stibine and their alkyl derivatives. Although we have found no evidence to support the claim that these gases are released from mattresses in infants' cots, we reviewed the toxicity data for these compounds to see, in particular, if they showed anticholinesterase activity. We examined also the postmortem findings in SIDS infants to see if they were compatible with the known features of poisoning by any of these gases.

12.12 While it is agreed that phosphine, arsine and stibine are highly toxic, their modes of action are not identical. Phosphine poisoning is characterised by gross pulmonary oedema, whilst haemolysis is the hallmark of poisoning by arsine and stibine. Anticholinesterase activity is not a feature commonly associated with any of these gases even at toxic concentrations. In vitro experiments have shown that stibine at toxic concentrations does not inhibit cholinesterase activity. Although phosphine can inhibit human red blood cell acetylcholinesterase in vitro, there is no evidence that this is a significant contributor to human poisoning with this gas. The available data indicate that the trimethyl derivatives are considerably less toxic than the parent compounds.

12.13 Postmortem examination of SIDS infants indicates that the features commonly found are not compatible with those of acute poisoning with anticholinesterases such as organophosphate or carbamate pesticides. There is no evidence of reduction in the mean brain acetylcholinesterase activity in the brains of SIDS infants. In addition, SIDS babies have not shown evidence of significant haemolysis, characteristic of poisoning by arsine and stibine, or of pulmonary oedema of an intensity found following poisoning by phosphine. A detailed review of infant developmental physiology, the pathophysiology of SIDS and possible mechanisms for such deaths finds many features which cannot be explained by the hypothesis.

12.14 We conclude, therefore, that there is no evidence that SIDS is due to poisoning by phosphine, arsine and stibine or their methylated derivatives.

Step 4

12.15 The detection of antimony in tissues from SIDS infants has been used as key evidence in support of the toxic gas hypothesis.

12.16 Research undertaken by several independent groups has shown that low amounts of antimony can be detected in samples from the majority of live infants and that the concentrations in SIDS infants are not exceptional. The antimony tissue distribution in infants is typical of the pattern seen with other non-essential trace elements. Its concentration varies in individual infants and in different types of sample such as lungs, liver, blood, urine and hair. Studies have not found the antimony concentrations in liver and lung tissue taken at postmortem from SIDS infants to be significantly different from those found in infants who have died of known causes. Antimony concentrations in liver and lung were not higher in older infants. Serum antimony concentrations in SIDS cannot be reliably interpreted.

12.17 Antimony is present in the diet and is ubiquitous in the domestic environment. It is not surprising that low concentrations of antimony are found in the blood, serum and urine of healthy infants. The measurable amounts of antimony in fetal liver, lung and umbilical cord tissue indicates maternal transfer of antimony during pregnancy. Sources after birth could include the diet or physical transfer from fabrics or household dust. Antimony was experimentally transferred physically from child care articles to hair. Pre- and post-natal accumulation could explain why hair antimony is higher in infants than in their mothers.

12.18 The presence of antimony in infants is not related temporally to the presence of fire retardants in mattresses. Antimony was reported in infants' hair and in fetal liver even before the introduction of antimony-based fire retardants. There is no correlation between antimony concentrations in infants' tissues and hair and their mattresses. SIDS infants have died on mattresses containing negligible antimony.

12.19 We conclude that antimony concentrations in the tissues of SIDS infants are not excessive and that there are a number of sources other than fire retardants in cot mattress materials to account for the presence of antimony.

Step 5

12.20 The hypothesis proposes that changes in SIDS rates correspond with the introduction and removal of antimony- and phosphorus- containing fire retardants and plasticisers used in PVC cot mattresses. In particular, the dramatic fall following the ‘Back to Sleep’ campaign has been claimed to be a consequence of Richardson’s advice and publicity concerning the toxic gas hypothesis.

12.21 The epidemiological features of cot death can, however, be explained without invoking the toxic gas hypothesis and some aspects of the epidemiology are at variance with it. The higher cot death rate among babies of higher birth order can be explained by reference to the epidemiology of infectious disease in infants. Similarly, the association with socioeconomic status has other possible explanations. The observation that premature babies die later than others is not explained by the theory. The suggestion that higher cot death rates are found amongst service families because service mattresses contained an arsenical preservative is incorrect.

12.22 We have also found no evidence that changing rates of sudden infant death correspond to the introduction and removal of fire retardants. Antimony trioxide was introduced to domestic cot mattresses in about 1988 and was used in many until 1994. During this period when median antimony levels were high the fall in the SIDS rate was the most rapid. Furthermore, similar falls in the cot death rates have been seen in other countries, some of which never had any publicity regarding the toxic gas hypothesis and, reportedly, did not use antimony fire retardants in cot mattress materials.

12.23 The ‘Reduce the Risk of Cot Death’/‘Back to Sleep’ campaign to place infants supine was launched in Autumn 1991 and was associated with the most rapid decline in the cot death rate. The risk of prone sleeping had begun to be recognised at least two years earlier which probably contributed to the small reduction from 1989-1991.

12.24 We conclude, therefore, that although some features of the epidemiology of SIDS fit the hypothesis, a detailed review of the data does not support it.

Overall Finding

12.25 We have presented the key steps and our findings diagrammatically as a flow diagram (see Figure 12.1). We conclude that the toxic gas hypothesis is unsubstantiated. There is no evidence to suggest that antimony- and phosphorus-containing compounds used as fire retardants in PVC and other cot mattress materials are a cause of SIDS deaths.

12.26 In addition we considered the wider question of whether chemicals used as fire retardants in cot mattresses pose any risk to infants. We have reviewed the toxicity of the chemicals used as fire retardants in PVC and other cot mattress material, and have found no reason to believe that they present any risk to the health of infants.

Recommendations

Any recommendations regarding changes in child care practices should be based on clear scientific evidence. During our deliberations we found no evidence either of toxic gases being generated from fire retardant chemicals encapsulated in cot mattress materials or that such gases are a cause of SIDS. Nor is there any evidence that the wrapping of cot mattresses with polyethylene made any difference to the incidence of SIDS. Also, infants have died on wrapped mattresses. We therefore see no reason to recommend that antimony- and phosphate-based fire retardants should be removed, and consider PVC covered mattresses as safe as any other mattresses. Mattress covers should, of course, be kept clean to minimise contamination with any microorganisms.

We make the following recommendations:-

- (i) The chemical composition of cot mattress materials, which we found some difficulty in obtaining, should be recorded by manufacturers of the final product. Data on production levels, changes in composition and year of manufacture by batch number should also be recorded. We invite the Department of Trade Industry (DTI) to consider how to make this information more readily available to interested parties.

- (ii) The introduction of any new fire retardant should be subject to scrutiny by the manufacturers and the DTI before use.
- (iii) In view of the difficulty of acquiring suitable tissue to measure antimony levels, the feasibility of a national tissue bank for tissue samples from sudden unexpected deaths in infancy and appropriate controls should be explored. The relevant government departments should consider devising a national strategy to facilitate obtaining parental permission before autopsy for the collection and future use of tissue samples for research.
- (iv) In all cases of sudden unexpected infant death, a full clinical history should be taken to include information on child care practices and conditions in the home to complement a thorough postmortem examination. The relevant government departments should consider how to take this forward.
- (v) The Office for National Statistics, the General Register Office for Scotland and the Northern Ireland Statistics and Research Agency, should publish annual tabulations of SID (or SIDS) more speedily to allow more timely analysis of trends.
- (vi) We endorse the current Foundation for the Study of Infant Deaths/ Department of Health advice in the leaflet 'Reduce the risk of cot death' since, following the issue of such advice, there has been a dramatic fall in the number of sudden infant deaths. Professional organisations with responsibility for infant care should, however, review regularly the advice they give in the light of new evidence.

Figure 12.1 Key Steps in Our Investigation of the Toxic Gas Hypothesis

