In an earlier Letter¹ I stated that the report by the 'Expert Group to Investigate Cot Death Theories' (the Limerick Report²) was incorrect in concluding that there was no possibility that toxic gases could be produced from cot mattress materials. My comments were in response to statements in the Report that "PVC mattress samples incubated with the fungus did not produce volatile phosphorus, arsenic or antimony compounds" and that "the toxic gas hypothesis is unsubstantiated".

The Limerick Report claims that there is no evidence for the production of trimethylstibine from antimony encapsulated in PVC. However, the Report clearly documents that, in certain conditions, trimethylstibine is readily generated by the action of common fungi on inorganic forms of antimony. To show that there is no possibility that trimethylstibine might be produced from PVC surely requires considerable discussion of the mechanisms by which, and conditions under which, trimethylstibine may or may not be formed. The Report is completely deficient in this regard. To simply dabble in a laboratory hoping that the experimental conditions are right and then adopt the approach that 'we didn't see it - so it wasn't there' strikes me as being rather second-rate.

Also, the Report does not adequately investigate the evolution of 'volatile arsenic' from PVC mattress samples. The mechanism for the biomethylation of inorganic arsenic is well established and S brevicaulis has a great propensity to perform this conversion under a range of conditions.³ I see no evidence in. the Report which demonstrates why PVC might be resistant to the action of S brevicaulis. How, then, can the Expert Group be so certain the S brevicaulis cannot produce trimethylarsine from PVC? Futhermore, the toxic gas hypothesis espoused by Richardson' and Sprott⁵ includes the evolution of trimethylarsine from cot mattress materials other than PVC. However, the Report does not even consider this issue. Therefore the Report cannot validly conclude that the toxic gas hypothesis is unsubstantiated.

I also noted that the Expert Group has

considered the wider issue of general risk to infants from exposure to chemicals used as fire retardants. The Group concluded that these pose no risk to infant health and that there is no reason to recommend that the use of antimony-based fire retardants should be discontinued. Given the known toxicity of antimony (and of arsenic which is almost invariably present in commercial antimony), I find this hard to fathom. The statement that 'antimony is ubiquitous in the domestic environment' is also disturbing, as is the Expert Group's failure to appreciate the gravity of this fact.

In fact, antimony exposure should not be taken lightly, since both acute and chronic toxicity can result from exposure to even small amounts of compounds of this element. The Report itself provides evidence of overexposure to antimony by UK infants when it cites that the antimony concentration in domestic dust is 13 mg/kg. This high level of antimony found in homes is no doubt due in part to the use of this element in fire retardants in household items. Based on standard default exposure factors 6 a young child may be exposed to up to 2.8 ηg/day of antimony. This level of exposure exceeds the USEPA Oral Reference Dose (RfD) of 0.4 \(\eta g/kg/day\). Moreover, the International Agency for Research on Cancer (IRAC) lists antimony trioxide as a Category 2B (possible human) carcinogen - all the more reason why exposure to this compound should be avoided.

Arsenic is a common impurity in antimony trioxide and infants' mattress materials may contain up to 430 mg/kg of arsenic. The acute toxicity of arsenic is well known but inorganic forms of arsenic are also carcinogenic and this has prompted various international agencies to establish maximum acceptable exposure levels. For example, the California EPA has set the 'no significant risk level' for arsenic at 10 ng/day for 70 kg adults8 (whether or not the arsenic is in a 'soluble' or other form). Therefore ingestion by an infant of only 25 mg of a PVC mattress cover could result in exposure to arsenic in excess of this adult exposure level.

It is important to minimise exposure to antimony and arsenic, especially in the case of infants. The Expert Group may not have a problem with the amounts of antimony and arsenic in mattress materials to which infants may be exposed but I do. I also find it difficult to understand why any legislation would permit the addition of antimony and arsenic to materials to which infants are intimately exposed.

Poisoning by trimethylarsine is very well documented, having first been identified by Gosio in 1893. The Limerick Report does not provide sufficient evidence as to why it states there is no potential for the production of trimethylarsine or trimethylstibine in an infant's environment. I am left to conclude that certain SIDS cases may be due to Gosio's disease and that, in some circumstances, the toxic gas hypothesis has validity.

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Anmerkung: siehe auch: Fitzpatric MG. The Evolution of Phosphine from Cot Mattress Materials. Department of Chemistry, University of Auckland, Final Report, December 1997