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Mr P Law
British Plastics Federation
5 Belgrave Square
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Your ref | Our ref | Date

23 May 90

Dear Mr. Law,

SUDDEN INSTANT COT DEATH SYNDROME

I refer to our telephone conversation to-day in respect of the above.

It has proved very difficult to find any information relating to the effects of exposure to any of these three gases which is not occupationally based. However, the following exposure/time relationships have been quoted :

Phosphine (IPCS Environmental Health Criteria 73, 1988) :

2000 ppm, rapidly fatal
500 ppm, death in 30 - 60 mins.
150 ppm, serious effects after 30 - 60 mins.
7 ppm, no serious effect after 30 - 60 mins.

Arsine (Swedish Criteria Group Consensus Report 1988) :

240 ppm, immediate death
24-48 ppm, death within 30 mins.
10 ppm, death following prolonged exposure
0.0165 ppm for 2 hours, report of acute signs and symptoms of arsine poisoning.

Stibine : No data found, but its action is similar to that of arsine.

I hope this information answers your questions.

Yours sincerely,

B BENNETT
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2248021

BPF - Holding Statement

July 89

COT MATTRESS BIODETERIORATION AND SUDDEN INFANT DEATH

1. Introduction

In early June 1989 considerable media coverage focused on the conclusions of a report entitled 'Cot Mattress Biodeterioration and Sudden Infant Death' published by Mr B.A. Richardson of Penarth Research International Ltd. This attempted to demonstrate a link between the use of PVC materials in cot mattress and the tragic phenomenon commonly known as 'cot death'.

This report has been subjected to detailed investigation by the Laboratory of the Government Chemist. This paper summarises the examination of the report separately carried out by the manufacturing companies concerned - the PVC resin manufacturers, additive suppliers and calendered PVC sheet manufacturers - and it approaches the subject from three principal standpoints:

- analytical chemistry
- microbiology
- medical

The principal questions which Mr Richardson's report raises are as follows:

A) What are the constituents of PVC materials used in PVC cot mattresses?

B) Can emissions of arsine, phosphine and stibine take place?

C) Do these emissions actually take place?

D) If so are the emissions injurious to health?

E) Is there any link between the emissions and cot death?

2. What are the constituents of PVC materials used in PVC cot mattresses?

Mr Richardson's report states that "all cot PVC contains antimony trioxide and phosphate plasticisers" (p.4). It is certainly true that triaryl phosphate is used up to 12% weight/weight final compound and antimony trioxide incorporated up to about 1.3% weight/weight final compound. However, they are never used together in a formulation. Mr Richardson is wrong in his assertion that 'manufacturers are now using autimony trioxide as a filler pigment at concentrations well above those used for fire retardancy' (p.4).

It is also untrue, as Mr Richardson implies (p.4) that 10,10' - oxybisphenoxyarsine (OBPA) is used as a biocide (p.4) in PVC cot mattress formulations.

Mr Richardson has not produced any evidence to indicate that he has analysed the PVC coverings to determine their compostion.

3. Can emissions of arsine, phosphine and stibine take place?

Mr Richardson's conclusions rest on the role of a particular fungus, 'Scopuleropsis brevicaulis', in developing on PVC cot mattresses 'if adequate moisture and protein are present, perspiration providing these requirements' (p.4). It is this, he says, which liberates arsine, phosphine and stibine. The strong implication is that he had carried out laboratory investigations himself (p.5).

However, he gives no information about the time-scale of the experiments to detect the presence of phosphine, arsine or stibine through the colour changes experienced by silver nitrate and silver diethyldithiocarbamate, in particular the length of time taken for colour to develop.

The use of silver nitrate test paper itself provides only a qualitative means of detecting the hydrides of phosphorous, arsenic and antimony. This method is by no means specific for these hydrides. A positive test is only indicative of a reducing agent being present.

Hydrogen sulphide also gives as colouration with silver nitrate, test paper. If sulphate-reducing bacteria are present, hydrogen sulphide could be generated. The absence of hydrogen sulphide would have to be proved - lead acetate test paper would probably suffice.

Mr Richardson also claims ammonia can be evolved by a variety of bacteria. Again, the absence of ammonia would have to be proved - it could also discolour silver nitrate test papers. Richardson is adding protein (a source of nitrogen) to the culture dishes.

If there is any degradation of P. V. C. itself then there is a possibility of hydrogen chloride evolution - another potential interference to silvernitrate test papers.

Nowhere in Mr Richardson's paper is there scientific evidence to show the absence of these potential interfering substances. Mr Richardson's detection method is inadequate if phosphorus, antimony and arsenic are present together.

More fundamentally there were no adequate controls in the investigations and the followed issues need to be addressed:

- what happens with fungi growing on the medium with no added Pvc?
- what happens with the indicator strip incubated over uninoculated medium?
- what happens with the indicator strip incubated with infected and sterile Pvc alone in the absence of a growth medium and added fungus?

These controls are particularly important since one of the references cited, 32, indicates that the organism can release arsine from laboratory media. His result therefore may just be an artefact of his methodology.

There is no evidence of identification of the fungus from infected samples. He claims the fungus has been detected but gives no quantitative data such as number per square millimetre or amount of hypha per square millimetre.

4. Do these emissions actually take place?

Mr Richardson's investigations were carried out in a laboratory where the fungus was developed under optimal conditions for its growth. They do not show that these gases could be produced 'in vivo'. The temperature under a baby in a cot would be at about body temperature of 37 C, possibly higher in situations where bed clothes act as insulation. It is not established that the fungus will grow or remain metabolically active at that temperature as many fungi will not grow at 37 C.

5. Are the emissions injurious to health?

The quanities of gases generated in cots under normal conditions are likely to be extremely small and uninjurious to health. This is further supported by the configuration of cots, most of which are open-sided with rails rather than panels. Additionally, mattresses currently manufactured have holes cut into the foam at the head end to facilitate a child's breathing. In these cirumstances, if phosphine, arsine and stibine were generated in the way described by Mr Richardson they would be well diluted.

This is graphically illustrated by the following example. If we assume that the densest of the three gases, stibine was present at the extraordinarily high level of 1000 ppm even then the density of the air/stibine mixture would only be 1.003 compared to air = 1. This is a trivial distance and the stibine would rapidly disperse in normal air movements such as convection currents. Lethal concentrations cannot accumulate.

6. Is there a link between the emissions and cot death?

Mr Richardson's report only remains a hyphthesis.

The statistical information he cites on the incidence of cot deaths (pp1-2) is not based on authoritative source material. Some fundamental weaknesses are illustrated by the following:

- his statement that most cot deaths occur at week-ends is not totally corroborated as several studies do not demonstrate a correlation.
- he does not mention that the incidence in the USA is equivalent to that of Australia and the UK.
- the lower incidence of cot deaths in Scandinavia is more probably due to their higher level of health education and social conditions.
- the rate for Hong Kong of 0.3 per 1000 is more likely to be due to the lower crowded conditions in which large families live, producing a greater awareness of subtle changes in a baby's condition.

- statistics on this subject are notoriously inadequate. This is due to the lack of a post-mortem and death certification, a post mortem being essential to produce a diagnosis of cot death. Statistics are probably at their weakest in Russia, China and India where Mr Richardson quotes a low incidence of cot death (p1).

Authoritative organisations and close students of cot deaths such as the Foundation for the Study of Infact Death believe that there has been no increase in cot deaths and that cot deaths occured in antiquity.

It is additionally, a well known fact that babies who die from 'cot death' do not necessarily die in a cot and are known to die even when in the arms of their parents.

Although there is probably no one single cause of cot death, perhaps one possible cause of cot death relates to the sleeping patterns of babies. Two-thirds of cot deaths take place during the first six months of life. A baby has a deep sleep pattern in this phase, which then changes.

We have to conclude that there is no direct evidence of an association between gaseous intoxication and Sudden Infant Death.

contributory factor in the cause of cot death

From Jacquie Fraser
advised of amendments
[illegible]/7/89

The comment " is that all there is
- that's excellent"

22480025

B. Bennett

PVC
Stuurgroep PVC & Milieu

PRESS RELEASE

Cot Deaths

As a result of reports concerning a possible connection between cot mattresses having a lining of PVC and cot deaths we make the following statement:

The first publications concerning the possible connection between flame retardants made of PVC and cot deaths came out early in 1989 in England, when a scientific investigator at Penearth Research International indicated a possible connection between cot death and flame retarding additives to the PVC lining on cot mattresses.

A publication of the results of research work was rejected by the British Medical Journal due to insufficient scientific foundation. Consequently, the British Ministry of Health, Safety and Environment dismissed the line of reasoning put forward by the researcher. The British Government likewise saw no reason to take any action regarding flame retardants used in the industry.

In June 1989 it was however decided that the laboratory of the British Government should carry out an investigation since it has been considered that every possible indication, no matter how small, should be investigated if it may help to solve the as yet unexplained problem of cot deaths.

Up to the present time no indications have been found concerning a possible connection. In view of the information now known the Stuurgroep PVC & Milieu looks forward with confidence to the outcome of the British investigation. Should however the results not indicate anything positive or be available within the envisaged 6 weeks the Stuurgroep and the Dutch government will together decide to carry out their own investigation.

It need hardly be said that the Stuurgroep PVC & Milieu takes very seriously reports concerning a possible connecting existing between PVC and cot deaths. It is of the utmost importance that all possible efforts be made to solve this mystery! All the same it is also very important that in a matter which involves, understandably, a great deal of emotion the facts must be relegated* first consideration.

Note for the editor: You may obtain more information from H.J. Moonen, chairman of Stuurgroep PVC en Milieu on tel.no. 020-934844.

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* given.