Rec'd at BPF Meeting on 23-4-90 for discussion.
Philip Law to amend following discussion & will fax to members for approval by end of week (27th April)

CONFIDENTIAL

COT DEATHS

Draft submission to the Department of Health, 3rd May

1. First use of PVC mattress Covers

Due to breaks in record keeping theory G the evolution of companies the exact date for the introduction of PVC mattress covers is not known. They were certainly in use in the early 1960's. • poss late 1950's

2. Dates on the first use of foam fillings

Information to be supplied by the British Rubber Manufacturers Association but their origins draw similar comments to the response in (1).
Latex for early 1950's prob; PU fillings prob for 1960. New Combonol foams not used before 1989.

3. The composition of materials used for covers.

Over the years the composition of PVC materials used in the manufacture of cot mattress has evolved to reflect changing technologies, regulations and their attendant standards. Typical formulations at different stages would be as follows:

1960's

PVC polymer	60%	
Octyl phthalate	30%	
Epoxy plasticizers	2 %	
Barium/cadmium/zinc stabiliser	1.25%	(might contain phosphite chelator)
Titanium Dioxide pigment	6-6.25%	

1980's

Pvc polymer	59%	
DIDP	33%	10% phosphate plast sometimes used.
Epoxy plasticiser	1.8%	
Barium/zinc Stabiliser	1.25%	Ca/Zn Also
Titanium Dioxide	6%	
Antimony Trioxide (may contain Arsenic as impurity)	1.2%	Not always used eg if phosphate plast used

1990

The level of Antimony Trioxide has been increased to a level of 4.5% to enable materials to meet the 'match test' requirement.

2248058

4. The compositon of materials used for fillings

Information to be supplied by the British Rubber Manufacturers.

5. Changes in the compositon of there materials, particularly in the use of fire retardants which might have taken place in 1986 and 1987.

We are unaware of any changes in the composition of UK PVC mattress covers in this period. with particular reference to fire retard systems.

6. Sales levels of PVC cot mattress in the UK together with any regional variations

Our best estimates of the total UK market sizes for the different types of PVC cot mattress are as follows:

- Cot mattresses : 90,000 - 100,000 units.
- Carry cot mattresses: 80,000 - 100,000 units.
- Push chairs: 250,000.

No statistics are available on the regional pattern of sales. From qualitative data we are unaware of any departure from what could be described as normal distribution.

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Mr P.G Dibb
Department of Health
Hannibal House
Elephant and Castle
London SE1 6TE

2nd May 1990 STRICTLY CONFIDENTIAL

Dear Mr Dibb

I am responding to your request for information on several points relating to the usage of PVC cot mattresses and foam fillings. I understand that you will be passing these data to the Expert Working Group convened by the Chief Medical Officer to advise him on Mr Barry Richardson's hypothesis on the cause of Sudden Infant Death Syndrome (SIDS). A delegation from the British Plastics Federation will of course be meeting the Expert Working Group on the afternoon of May 3rd when we will be pleased to discuss this issue further and to add any necessary amplification.

I should add that owing to the structure of the industry and its trade bodies it is the British Rubber Manufacturers Association (BRMA) which is the focal point for matters relating to foam. We cannot therefore offer detailed comments here on foam but we expect that a representative of the BRMA will join our delegation on May 3rd.

To revert to your specific questions:

1. First use of PVC mattress covers

   Due to breaks in record keeping during the evolution of companies and changes which have affected the structure of the industry the exact date for the introduction of PVC mattress covers is not known. They were certainly in use in the early 1960's and their origins probably lie in the late 1950's.

2. Dates on the first use of foam fillings

   This information will be supplied by the British Rubber Manufacturers Association but the origins of foam fillings draw similar comments to the response in (1).

3. The composition of materials used for PVC mattress covers

   Over the years the composition of PVC materials used in the manufacture of cot mattresses has evolved to reflect changing technologies, regulations and their attendant standards. Typical formulations at different stages in the history of the product would be as follows:

1960's

PVC Polymer	60%
Octyl Phthalate Plasticiser	30%
Epoxidised Soya Bean Oil as Plasticiser	2%
Barium/Cadmium/Zinc stabiliser	1.25%
Titanium Dioxide pigment	6-6.25%

1980's - two formulations in common use:

(A)	PVC Polymer	59%
	Di-isodecyl phthalate plasticicer	33%
	Epoxy plasticiser	1.8%
	Barium/Zinc Stabiliser	1.25%
	Titanium Dioxide	6%
	Antimony Trioxide	1.2%
(B)	PVC Polymer	58%
	Di-isodecyl phthalate plasticiser	23.5%
	Phosphate Plasticiser	8.5%
	Epoxidised Soya Bean Ester as Plasticiser and Co-stabiliser	1.1%
	Calcium/zinc stabiliser	1.1%
	Phospite Stabiliser booster	1.1%
	Titanium Dioxide	6.8%

1990

The level of Antimony Tioxide in formulation (A) above has been increased to a level of 4.5% enable materials to meet the `match test' requirement.

It is worth mentioning in this context that PVC sheet is widely used in hospitals for mattress covers and pillow cases and that fire retardants have been used in these applications to meet DHSS requirements for many years.

4. The composition of materials used for fillings

This information will be supplied by the British Rubber Manufacturers Association.

5. Changes in the compostion of these materials, particularly in the use of fire retardants which might have taken place in 1986 and 1987

We are unaware of any changes either in the composition of PVC mattress covers or the fire retardant systems used in this period.

6. Sales levels of PVC cot mattresses in the UK together with any regional variations

Our best estimates of the total UK market sizes for different types of PVC cot mattress are as follows:

- Cot mattresses: 90,000 - 100,000 units (UK manufacturers have the major share of the market)
- Carry cot mattresses: 80,000 - 100,000 units (There is a substantial level of imports of the sheet used in this category particularly emanating at the moment from East Germany and Israel)
- Push Chairs: 250,000 units (UK manufacturers have the major share of the market)

No statistics are available on the regional pattern of sales. From qualitative data we are unaware of any departure from what could be described as normal distribution.

More broadly Mr Richardson's study has been subjected to close scrutiny by the industrial companies involved. They and other organisations have been unable to reproduce his work. A full appraisal of his initial paper carried out by the British Plastics Federation is attached as Appendix A. A detailed report of experimental work carried out by the British Plastics Federation is also attached as Appendix B.

The British Plastics Federation is extremely anxious to co-operate fully with the Department of Health's Expert Working Group in its investigation to see an early vindication of our products. We did in fact at a much earlier point provide detailed information to the Laboratory of the Government Chemist but unfortunately we have not yet had a formal indication of its results.

We now look forward to some positive feedback from the Department and should you require any further information please do not hesitate to contact me.

With best wishes

Yours sincerely

Philip Law

Philip Law
Head of Planning and Membership Activities

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APPENDIX I

BRITISH PLASTICS FEDERATION'S REVIEW OF MR B.A. RICHARDSON'S REPORT, 'COT MATTRESS BIODETERIORATION AND SUDDEN INFANT DEATH' (JUNE 1989)

AUGUST 24TH 1989

24thAugust 1989

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 a timony 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 compositions.

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 a 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 silver nitrate 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 following 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 difference 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 no 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 over-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.

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APPENDIX II

EXPERIMENTAL WORK CARRIED OUT BY THE BRITISH PLASTICS FEDERATION ON THE BIODETERIORATION OF PVC COT MATTRESS COVERS.

JULY 12TH 1989

Director - D R Jones
A company limited by guarantee
Registered in England No. 282883

BIODETERIORATION OF PVC COT MATTRESS COVERS

INTRODUCTION

A recent report by B.A. Richardson of Beharth Research International Ltd., Guernsey stated that phosphine, arsine and stilbene can be released from plasticised PVC cot mattress covers by biodeterioration caused by reaction of the fungus Scopulariopsis Brevicaulis.

A set of experiments were performed to test this hypothesis.

EXPERIMENTAL

An authentic sample of the fungus Scopulariopsis brevicaulis No. 049528 was obtained from C.A.B. International Mycological Institute, Ferry Lane, Kew, Surrey, TW9 3AF.

Culture media utilised during the investigation were malt extract agar (Oxoid, CM 59) with the addition of 10 g per litre soy peptone (Lab M, MC3) and malt extract broth (Oxoid CM 57) with an identical addition of soy peptone.

The media were prepared and sterilised according to the manufacturer's instructions. The agar medium was dispensed in 20-ml volumes into 90-mm single vent petri dishes. The broth medium was stored in 50-ml volumes in 100 ml Duran screw-capped bottles.

Silver nitrate and mercuric chloride test papers were prepared as in B.A. Richardson's report.

Preparation of Starter Culture

A starter culture was prepared by inoculating soy-enriched malt extract broth with scopulariopsis brevicaulis and incubating on a shaking water bath for 72 hours at 23°C. Subsequent viable counting, after suitable serial dilution, indicated that the resulting suspension contained approximately 6.5×10^5 colony-forming units per millilitre.

Investigation in liquid medium

In order to investigate whether any phosphorus arsenic or antimony hydrides were liberated from appropriately doped broths by the action of scopulariopsis brevicaulis the following experiment was performed:

Four bottles of broth were aseptically inoculated with 0.5 ml of a starter culture. One of these bottles was used as a control, with no further additions. The other three were supplemented by the addition of one of the following:

(a) Corvic S71/102 PVC powder (0.5 g)
(b) Antimony Oxide powder (0.5 g)
(c) Triaryl Phosphate (2.0 ml)

Suspended from the top of each bottle were strips (50 x 5 mm) of silver nitrate test paper and mercuric chloride test paper. The bottles were incubated for 144 hours at 23°C, after which a dense micelial growth was observed in each sample.

Investigation on solid medium

In order to investigate whether the hydrides of phosphorus, arsenic and antimony were released from the materials under investigation by the action of the fungus when grown on a solid medium, the following experiments were set up:

Sixteen petri-dishes containing soy peptone-enriched malt extract agar were inoculated with 0.2 ml of the Scopulariopsis brevicaulis starter culture. This inoculum was then spread uniformly over the surface of the agar. Two plates were used as controls. To the remainder, the following additions were made:

(1) TRIARYL PHOSPHATE (0.5 ml) was spread over the agar.
(2) Corvic S71/102 (0.5 g) was sprinkled over the agar.
(3) Antimony Oxide (0.5 g) was sprinkled over the agar.
(4-14) 25 x 50 mm samples of PVC sheet was placed on the agar. See Table 1 for the composition of the PVC Formulations Ref. 7631-7641.

Widely separated strips of silver nitrate and mercuric chloride test paper (100 x 5 mm) were now placed in the lid of the petri dish, the paper was stretched over the inoculated and impregnated agar surface. All the plates were incubated at 23°C for 144 hours, after which a dense lawn of fungal mycelium covered most of the plate.

EXAMINATION OF TEST STRIPS AFTER TESTS

No stains were observed on the mercuric chloride test papers from both the liquid and solid cultures, i.e. no positive test for arsenic.

There was some yellowing of the test strip from the control but only on the edge of the strip overhanging the edges of the petri-dish.

It was noticeable that the only area of the plates where dense fungal growth had not occurred was that zone immediately below the mercuric chloride test strip. This is presumably because of the potent fungicidal properties of mercury compounds.

No stains were observed on the silver nitrate test papers on the area of strip across the diameter of the petri-dishes. There was a general discoloration of all the silver nitrate test strips which is probably the effect of daylight.

Black stains were observed on the silver nitrate test papers in the area which was bent over the edges of the petri-dishes. These black stains were also observed in the controls.

As there was no staining of the papers in the areas where fungal growth was taking place, these stains on the edges of the test papers cannot be taken as a positive test of hydride generation especially as the same phenomenon occurs in the controls.

On the plates where triaryl phosphate, antimony oxide and PVC powder had been placed in the agar surface, the fungal growth had completely invaded the impregnated areas.

On the plates where plasticised PVC strips had been placed on the agar surface, the fungus did not appear to invade the PVC strips.

There was also no staining of the test strips from the liquid broth experiments.

CONCLUSION

This repeat of Richardson's work was unable to give a positive identification of hydride generation from PVC cot mattress materials. The stains on the test papers at the overhanging outside edges cannot be taken as a positive test as this also occurs in the controls.

Even in the more rigorous conditions of fungal growth in liquid cultures were we NOT able to detect phosphine, arsine or stilbene using Richardson's detection methods.

TABLE 1

FORMULATION DETAILS

CORVIC S71/102	100	100	100	100	100	100	100	100	100	100	100
WHITE LEAD PASTE	4	4	4	4	4	4	4	4	-	-	-
LEAD STEARATE	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25	-	-	-
IRGASTAB-BZ531	-	-	-	-	-	-	-	-	2	2	2
TRIARYL PHOSPHATE	50	-	-	-	-	-	50	-	50	-	-
PHOSPHATE/ADIPATE BLEND	-	50	-	-	-	-	-	-	-	-	-
DOP	-	-	50	-	50	37	-	50	-	50	50
ALKYL DIARYL PHOSPHATE	-	-	-	50	-	-	-	-	-	-	-
ANTIMONY OXIDE	-	-	-	-	5	5	-	5	-	-	5
CERECLOR-S52	-	-	-	-	-	20	-	-	-	-	-
TIOXIDE RFC5	-	-	-	-	-	-	5	5	-	-	-
COMPOUND REF.:	7631	7632	7633	7634	7635	7636	7637	7638	7639	7640	7641

MSB file

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12.4.90

To: BPF MEMBERS INVOLVED IN COT DEATH ISSUE

Gentlemen,

As you know the Chief Medical Officer has asked a group of experts to advise him on Barry Richardson's hypothesis.

The Federation has been invited to attend a meeting of this group on May 3rd (time and location to be advised to us) and has been specifically asked to present the following data in as much detail as possible in the form of a paper:-

1. Dates on the first use of PVC mattress covers.
2. Dates on the first use of foam fillings.
3. The composition of materials used for covers.
4. The composition of materials used for fillings.
5. Any changes in the compositions of these materials, particularly in the use of fire retardants which might have taken place in 1986 and 1987.
6. Sales levels of PVC cot mattresses in the UK together with any regional variations.

The Department Of Health have pledged their confidential treatment of this information. I have naturally accepted the invitation, and I am now taking steps to prepare this paper. Not all of this data will be available from Federation sources, for example The British Rubber Manufacturers Association will be the fount of knowledge on foam fillings. I will be contacting those companies I feel will be best placed to provide this information directly.

I wish to invite you to a meeting of the interested manufacturers at the BPF at 11.00 a.m. Monday 23rd April to review the information which we can collate, determine the compostion of our delegation to the Department Of Health and our approach to the Meeting on May 3rd.

Would you please send me a fax advising me of your attendance.

Regards,

Philip Law
Head Of Planning and Membership Activities

2248073

Hospital mattress covers are a sep. item again
Cut off roll in Hptl... could well be based on phenylate plast. due to (oil)extractn resist. requirements

Query Gf on Dr Cook (Lab Gov Chem) progress: Shows to Stat. but no
BPF should a) criticise untrue statements in Richardson Lancet article.
b) present Ciba Geigy results on phos. plast.
c) emphasise willingness to help in every way incl. request for indep. work (as per Lab Gov Chem.)

May 3rd Party proposed:
Limited Group ~~Rep~~ BPF (4) + BRMA (Buell) rep (for foam fillings).

Phil. Law: Brian Bennet (Medical and PVC supplier EVC.), Robin? (Wide Stores): T. Hyde (Ciba Geig
BPF + PVC supplier (Wild) + Sheet Mfr. + Adds Supplier.

Suggest: To request Mel Repo see Expert Group after Richardson
Resent orig. Aug 89 BPF Critique with formalised issue (BPF handing; Identity of Task Force).

ECVM has been informed of current BPF meeting/involvement

224.8.0074

BPF Mon April 23rd Cot Death Special Meet'

8 present + Philip Laws.. incl. Brit Rub Mfrs Assoc (Mr Rennell) rep

William Hyde Also
Wendle Storey HPL
ICI (Brian Bennett) Cole Geigy
Comm Platters.

DoH Expert Group: reporting to Sir Donald Acheson (Chief Med Officer).

Prof West — Analy Chem (Aberdeen)
Prof Hawksworth — Mycologist (Kew)
Dr. Poovdfoot — Dir. Poisons Unit (Edinburgh R.I).
Dr Jean Keeling — Paediatric Pathologist (Edinburgh)
Prof Milner — Paediatrician (Nottingham)
Prof Eva Alberman — Paediatric epidemiologist

Also there were DTI Assessors.

Meredith Glover (Consumer Safety)
Dr Cooke (Lab Gov Chemist)

Approx 3 months Time Scale expected before this Group Reports.

Richardson appeared on Dutch TV prog recently on Cot Death.
— Also provided Video.

DoH Exp Grp requested meeting on May 3rd with BPF. (Hatfield ST, p.m. but to be confirmed)
Also seeing Richardson and Found for Study of Inf Death
Not all of DoH Exp Grp will be present - 2 or 3 only.

BRMA:
New foam required from March 89 but not yet certain when reached Cot mattresses.
V. short notice to gather info. don't expect to be able to compile all requested info for (first) May 3rd meeting.

BPF Meeting organised to
1) Look at info summarised by PL.
2) Agree group to go to May 3rd meeting.

Storey's 1982 supplying PVC sheet for Carry Cot mattresses with PU foam interest
Identifying when diff mats commenced use is one thing; but getting data on market split not likely to be poss. for these years (y 60's)

Diff suppliers kept to be used for Cots and Carry Cots. latter could be merchanted from
Prob'dom by UK mfrs overseas: cheap market so
Unlikely to be Home standard

2248075

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Patron: Her Royal Highness The Duchess of Gloucester

COT DEATH RESEARCH AND SUPPORT

13th March 1990

STATEMENT TO ALL FSID STAFF FOR INFORMATION

"Sir Donald Acheson, the Chief Medical Officer, announced today (9th March 1990) that he has asked a group of independent experts to investigate the claim that the emission of toxic gases from fungal growth on some cot mattresses is a cause of sudden infant death.

The group will be chaired by Professor Paul Turner who is Chairman of the Government's independent expert advisory committee on toxicity and will include experts in toxicology, neonatal pathology, mycology, epidemiology and analytical chemistry.

Announcing the review, Sir Donald said that the sudden infant death syndrome was almost certainly due to many causes and these are little understood. Any new hypothesis therefore warranted careful consideration. The hypothesis - put forward by Mr B A Richardson, Director of Penarth Research International and Mr P R Mitchell, Director of Mitchell Marquees - is that due to the action of certain fungi, soiled cot mattresses may emit toxic gases.

Sir Donald went on: "The death of an infant is a shattering blow to parents. We have a duty to investigate possible causes. But this needs to be done in a way which, on the one hand avoids alarming parents unnecessarily before claims can be properly assessed and on the other, raising hopes that a 'preventable cause' of sudden infant death has been found".

"Mr Richardson's and Mr Mitchell's work which has not been published in the scientific literature, and therefore has not been subject to the usual scrutiny and discussion, is already being assessed by the Laboratory of the Government Chemist. The independent experts will be examining the laboratory findings as well as the work done by Mr Richardson and Mr Mitchell. I have asked the group to report as soon as possible and will make public their recommendations".

In the meantime, Sir Donald has taken advice from a range of experts including neonatal pathologists and paediatricians. He has been advised that no case of cot death in this country due to the toxic gases arsine, stibine and phosphine has been reported. Nor is there any need for parents to take any special action in respect of cot mattresses. Nevertheless, as a matter of prudence he has invited an independent assessment of the evidence".

The above extract is taken from a Department of Health press release, and follows articles in the Daily Express in February 1990. These articles stated that "startling evidence by government scientists has confirmed a possible new link between cot death syndrome and fire resistant mattresses", that "Ministers begin a series of crisis meetings in Whitehall", that "Health Secretary Kenneth Clarke (has) ordered an immediate enquiry into the possible link between cot deaths and fireproof mattresses" and that "Mr Clarke is prepared to order tens of thousands of baby mattresses to be withdrawn from sale if Government scientists confirm a possible link".

22480076

Registered as Charity No. 262191

The articles correspond to a theory of a cause of sudden infant death syndrome (SIDS) that was widely reported on television, radio and in the press at the beginning of June 1989. Laboratory work by a chemical analyst, Mr Barry Richardson, suggested that toxic gases were being released when biocides (substances acting against infecting organisms) and fire retardants contained in cot mattress coverings of plasticised polyvinyl chloride (PVC) were acted on by a normal household fungus. He believed that the gases might be a significant cause of SIDS and informed both the Foundation for the Study of Infant Deaths and the Department of Health of his views.

Mr Richardson told a meeting of the Scientific Advisory Committee of the Foundation for the Study of Infant Deaths about his work, and was questioned about it by members. After careful consideration, the Committee formed the view that this was an unproven hypothesis for which no direct evidence existed and that this was unlikely to be a major cause of SIDS.

The infant mortality rate (all first year deaths) has continued to fall since plasticised polyvinyl chloride (PVC) was introduced while the SIDS rate has stayed relatively constant.

However, the toxic gas theory was widely publicised on radio and television and in the press before any scientific confirmation of the laboratory work had been undertaken. The Foundation was concerned that any possible such link with SIDS should be quickly and appropriately investigated and for that reason is at present funding a project in London (£10,000 over 6 months) to undertake suitable tests.

Advice to parents:

The Foundation for the Study of Infant Deaths advises parents that they need not feel that they should take any special action following these claims, and warns them NOT to endanger their baby by wrapping polythene around the mattress as has been suggested by Mr Richardson.

For further information please contact:

Erica De'Ath (Chief Executive) or
Tracy Lamb (Research and Information Officer)
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(ENDS)

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Research Note January 1990

Cot mattress biodeterioration and sudden infant death

1. Introduction

1.01 PRIL research in 1988 included an investigation into deterioration of reinforced polyvinyl chloride (PVC) fabric used as tentage. The fabric was found to be infected by various fungi which were utilising the plasticisers as nutrient, particularly *Aspergillus niger* which causes grey staining along the reinforcement fibres and *Streptomyces rubrireticuli* which causes patches of pink staining, despite the presence of the arsenical biocide 10,10'-oxybisphenoxyarsine (OBPA). The clients sponsoring the research were warned by the project director B A Richardson that other fungi might develop such as *Scopulariopsis brevicaulis* which could convert the OBPA biocide into toxic arsine gas, and that accumulations of this gas might occur in folded tentage in winter storage. One of the clients, P R Mitchell of Mitchell Marquees, enquired whether similar infections might occur on PVC cot mattress coverings and whether generated arsine might be the cause of unexplained sudden infant death.

1.02 The fungus *Scopulariopsis brevicaulis* is commonly found infecting protein substrates such as meat, milk and cheese, as well as damp wool, leather and silk (1,2). It has been known since about 1890 that this fungus is able to convert arsenic compounds into arsine (arsenic trihydride) and related alkyl compounds (1-17). Gosio and Sanger reported that some mystery illnesses and deaths were due to arsine generation through this fungus infecting wallpaper printed with the arsenical pigments Paris green and Scheele's green, although another source of arsine on papered walls was horse-hoof size which often contained white arsenic (arsenious oxide) as a rodent repellent. In the United Kingdom the last deaths from these causes were reported in 1932 (12).

1.03 Investigations on PVC cot mattress coverings show that all used mattresses are naturally infected by the fungus *Scopulariopsis brevicaulis* in the area affected by the warmth and perspiration of the sleeping infant. Arsine generation is not usually identified as OBPA biocide is not generally used in PVC cot mattress coverings. However, phosphine (phosphorus trihydride) and particularly stibine (antimony trihydride) and related alkyl compounds are frequently identified, originating from the phosphate plasticisers and antimony trioxide that are used to improve fire retardency. Exposed foam mattresses are infected by a wide range of micro-organisms where they are affected by dribble and vomit, including *Scopulariopsis brevicaulis* which generates phosphine if phosphate fire retardents are present.

2. Research observations

2.01 All used cot mattress coverings are infected by a wide range of the micro-organisms that are commonly found in the domestic environment.

2.02 PVC cot mattress coverings are generally affected by surface soilage infections but *Scopulariopsis brevicaulis* always develops on the area affected by the warmth and perspiration of the sleeping infant. This infection, which is apparently prompted by the nitrogen content in the perspiration, develops in the plasticiser within the PVC sheet and is invisible, the only evidence being progressive loss of plasticity and the development of brittleness and splits. Phosphine, arsine and stibine or related alkyl gaseous compounds are usually detected when samples are incubated. Stibine, originating from the antimony trioxide fire retardent component in the PVC, is most often detected. Phosphine from fire retardent phosphate plasticisers is also often detected. Arsine from the arsenical biocide OBPA is rarely detected but traces of arsine are sometimes detected which are attributable to arsenical impurities in antimony trioxide. Fungal activity and gas generation vary with temperature, the rate of generation at 40°C being about 100 times that at 20°C. Phenolic compounds, released when aryl phosphate plasticisers are degraded by the infection, sometimes cause pink staining in the shape of the sleeping infant.

2.03 Exposed foam mattresses, that is mattresses which comprise foam alone, or covered with net, or partly covered with PVC, are usually infected by a wide range of infections, including many fungi which produce spores or support mites which can prompt allergic respiratory reactions such as asthma. *Scopulariopsis brevicaulis* infections on foams are usually associated with phosphate fire retardents and phosphine generation.

2.04 Unexplained sudden infant deaths or cot deaths usually involve infants in the prone or face down position with generous coverings. Death usually occurs after the infant has remained undisturbed for six hours or more. Death is usually attributed to sudden infant death syndrome or SIDS. The prone position and heavy coverings maximises exposure to any gas generation from the mattress, and hyperthermia in the infant caused by the heavy coverings also increases the rate of gas generation through both the higher temperature and the increased perspiration.

2.05 50 mattresses from 45 SID incidents have been examined, including 1 cotton covered, 26 PVC covered, 15 PVC covered with exposed foam at one or both ends, and 8 exposed foam; 2 mattresses were involved in 5 incidents. All the PVC and foam mattresses were infected by the fungus *Scopulariopsis brevicaulis* and, when samples were incubated, all generated phosphine, arsine or stibine gas. Mattresses had generally been used for a previous infant, the mattresses varying from apparently excellent clean condition to grubby surfaces with brittle and split PVC coverings or heavily stained foam. Five mattresses were involved which had not been used for previous infants. Two of the mattresses involved PVC coverings which were brittle, pink stained and generating phosphine; purchase dates are also known for some of the used mattresses, those generating phosphine and therefore containing phosphate plasticisers apparently deteriorating more rapidly than those containing other plasticisers. Another mattress was exposed foam, pink stained and generating phosphine from a phosphate fire retardent treatment. In all cases the infection and gas

generation were restricted to the area affected by the warmth and perspiration of the sleeping infant. Bed coverings were also sometimes examined. Cotton sheets covering the mattresses were always found to be saturated with perspiration if examined shortly after death.

3. Notes

3.01 Incubation of naturally infected samples of PVC and foam at high humidity is sufficient to cause phosphine, arsine or stibine gas generation. However, incubation of small samples on nutrient plates is necessary for the infection to spread from the samples so that it can be identified. Samples are therefore examined routinely by plating on agar plates containing 5% malt and 5% soya flour, the protein in the soya flour encouraging *Scopulariopsis brevicaulis* growth. Samples are incubated at 22 to 25°C in order to restrict activity and limit the rate of gas generation to avoid danger to staff; headaches are the first indication of excessive gas concentrations.

3.02 Silver nitrate and mercuric bromide (or chloride) papers are used for gas detection; gold compounds can also be used. With silver nitrate phosphine gives a yellow colour, whilst arsine gives a brown or mauve colour and stibine a pink or brown colour; high concentrations eventually cause darkening to grey or black with all three gases. With mercuric bromide phosphine and stibine cause no colour change as white complexes are formed but arsine gives a yellow colour, darkening to orange and red with high gas concentrations. These reactions are quantitative and sensitivity can be improved by the use of optimum loadings on the detection paper and by limiting the size of the paper strips; high humidity is also essential as the reactions do not occur if the papers are too dry. Routine studies involve placing small samples of PVC or foam on malt/soya agar plates in Petri dishes and incubating for several days at 22 to 25°C until active *Scopulariopsis brevicaulis* growth develops, either as beige coloured hyphal growth or the buff coloured slime form which develops under stress. With PVC the fungus develops only on the nutrient medium and the edge of the PVC. Small strips of detection paper about 5mm wide and 25mm long are then slipped over the edge of the dishes and secured with the lid, the colour generated after 1 to 3 hours identifying any phosphine, arsine or stibine generation. Hydrogen sulphide interference, which is indicated by early darkening of the silver nitrate, does not normally occur. Alkyl compounds are also generated by *Scopulariopsis brevicaulis* and account for the characteristic garlic odour associated with phosphine, arsine and stibine, but the silver, mercury and gold complexing method of detection is more sensitive to the trihydrides.

3.03 The increase in the rate of gas generation at higher temperatures can be observed by placing well established open plates in a desiccator in a water bath, conditioning to the required temperature for an hour or more, flushing with air, and determining the rate of gas generation by introducing test paper and observing the rate of development of the detection colour. Silver nitrate and mercuric bromide papers can be used but greater detection sensitivity can be achieved using paper treated with mercuric chloride and a pH indicator, the trihydride gases complexing with mercury forming hydrochloric acid.

3.04 The gases can also be detected using, for example, the termite *Reticulitermes santonensis*. Termites placed on well developed plates with PVC samples generating easily detectable arsine and stibine are generally killed within two hours but phosphine is distinctly less toxic. Did he do that?

3.05 Although the fungus *Scopulariopsis brevicaulis* can survive on simple carbohydrate media such as malt agar, a source of nitrogen will greatly enhance activity and enable the fungus to compete strongly with other organisms. This fungus is often therefore the dominant species on substrates containing protein such as meat, milk and cheese, but substrates containing nitrogen sources such as ammonium compounds and urea are equally suitable. Extracellular enzymes are produced by invading hyphae which convert the nitrogen sources to ammonia (nitrogen trihydride) gas which is then absorbed by the hyphae and utilised for protein formation. However, the extracellular enzymes do not act on nitrogen sources alone but also on other Group V/Vb compounds, generating phosphine, arsine and stibine which are apparently liberated into the atmosphere as they cannot be absorbed by the hyphae. The fungus has an affinity for Group V/Vb compounds; if they are sprinkled on plain agar plates to form a distinctive pattern the fungus will follow the pattern. If the fungus is under stress, usually due to inadequate nitrogen in the substrate, the beige hyphal growth degenerates to a buff coloured slime, reverting when plated onto a more suitable medium. The infection is invisible in PVC fabric because it is in this structureless slime form and causes only slight distortion of the PVC; the infection does not spread over the surface of the PVC.

3.06 Phosphine, arsine and stibine poisoning cannot be readily detected on post mortem investigation. Normal lethal doses will cause increases in phosphorus, arsenic and antimony levels in blood and tissue but these increases are smaller than normal ambient variations and therefore undetectable.

3.07 Standard toxicological references indicate that chronic exposure to phosphine, arsine or stibine causes death by haemolytic anaemia in adults, although a headache is an early symptom of poisoning (32-36). It is suggested that these gases are accumulative but this is not strictly correct; chronic exposure simply causes the anaemia to become progressively more severe. However, SIDS is not normally associated with haemolytic anaemia, although colour changes in the lungs may result from local haemolysis in lung tissue. Infants are sometimes reported to be irritable in their cot several hours prior to death which may indicate headaches. Infants are sometimes provided with monitors which detect cessation of respiration but prompt attempts at resuscitation are apparently unsuccessful. These observations may indicate a poisoning action causing cessation of respiration, perhaps acting through the respiratory centre in the medulla, although the trihydrides are likely to degrade in the blood before reaching the brain, and respiration in young infants is controlled by the carotid sinus rather than the respiratory centre. Whilst the trihydrides may block the carotid sinus, the circumstances of sudden infant death are also consistent with damage to sensitive cells due to oxygen or blood sugar deprivation. The trihydrides are reducing agents and may obstruct oxygen transport by blocking the oxygen acceptor sites on erythrocyte haemoglobin. Alternatively phosphine, arsine and stibine may form phosphonium, arsonium and stibonium hydroxide which may condense onto blood sugar hydroxyl groups, preventing blood sugar utilisation by cells. Research by PRIL in 1968 showed that poisoning by organotin compounds, which caused numerous deaths and brain

injuries in France in the Stalinon affair, involves similar condensation on blood sugar hydroxyl groups (40-42).

4. Further information

4.01 This Research Note will be revised as this project progresses. Revised issues will be distributed only to bona fide research teams. Further information on this project is available from the director B A Richardson.

4.02 This project is not sponsored. PRIL has provided laboratory facilities for the microbiological and entomological work. The project consultants are B A Richardson, T R G Cox and J G Watt. The culture of *Scopulariopsis brevicaulis* involved in the initial work was provided by Dr D Allsopp of the International Mycological Institute at Kew; subsequent research involved cultures from naturally infected samples.

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BAR/JR P1

Cot mattress biodeterioration and SIDS

SIR,—In 1988, various fungi were identified in industrial plasticised polyvinyl chloride (PVC) despite the presence of the preservative 10, 10'-oxybisphenoxyarsine (OBPA). Warnings were given that the PVC might also become infected by arsenic-tolerant organisms which might convert OBPA to toxic arsine gas. Mr P. R. Mitchell, a client of our company, inquired whether arsine generation through biodeterioration of such coverings might be a cause of sudden infant death syndrome (SIDS).

50 mattresses used in forty-five SIDS incidents were examined, including 1 cotton covered, 26 PVC covered, 15 PVC covered with exposed foam at one or both ends, and 8 exposed foam; in five incidents 2 mattresses had been used. All mattresses contained *Scopulariopsis brevicaulis*, but only in the area beneath the infant affected by warmth and perspiration. Incubated samples of the infected materials all generated toxic trihydride gases. PVC coverings usually generate stibine (SbH_3) from a fire retardant additive, antimony trioxide, but also phosphine (PH_3) from the phosphate plasticisers that are used when fire resistance is required. Although OBPA is not generally used in cot mattress PVC, tests indicated arsine (AsH_3) generation when it was used; and small amounts of arsine were sometimes identified when it was not, presumably from arsenical impurities in antimony trioxide. Mixtures of gases were often detected. Phosphine generation from PVC was sometimes associated with the development of a pink stain, usually in the shape of the sleeping infant, apparently caused by liberation of phenol compounds from decomposition of phenyl phosphate plasticisers. Only phosphine was generated from infected cotton and foam samples, apparently through biodeterioration of phosphate fire-retardant treatments. Foam samples and the 1 cotton sample were also infected by various other fungi, especially in the area affected by dribble; many of these fungi produce asthmatic reactions.

The generation of highly toxic trihydride gases from cot mattresses is a serious threat to health. The gases are heavier than air, and infants most at risk are those who remain for long periods in their cots, sleep prone in carry-cots, or are heavily wrapped such that they are in long and close breathing contact with accumulations of gas. Heavy wrapping also induces hyperthermia, which prompts an increase in gas generation. Some infants probably survive because the first symptom of poisoning is headache, which results in irritability, dislodgment of bedding, and improved ventilation; infants of low weight cannot dislodge bedding. SIDS occurs mainly in infants up to age 5 months who sleep prone with heavy wrapping and have indications of hyperthermia. SIDS is only reported in western-style communities where foam and PVC covered cot mattresses are used, and has only been recognised since about 1969, following introduction of these materials during the previous 10–15 years.

It has been reported that SIDS is less likely to occur in first born children, perhaps because mattresses are new and do not become infected while the infant is young and most at risk. 5 first children were involved in the 45 incidents investigated. 2 died on previously used mattresses; 1 of these 2 slept on a mattress immediately after use by an older child. 1 infant died on a new foam mattress that was generating phosphine and 2 died on new PVC mattresses which were affected by exceptionally active fungus and were generating phosphine; two other new mattresses were implicated, both of which were similarly affected.

Infection by *S brevicaulis* is not readily visible in PVC. Severe deterioration, usually associated with phosphate plasticisers, results in brittleness and splitting. In the home the fungus is usually found in protein-containing substances such as cheese and meat, and in damp wool and leather; however, its extracellular enzymes can convert a wide range of compounds containing the elements nitrogen, phosphorus, arsenic, and antimony into the trihydrides ammonia, phosphine, arsine, and stibine. The preferential development of this fungus in cot mattresses should be attributed to the presence of nitrogen compounds in the perspiration. The generation of arsine in this way has been recognised for about 100 years since Gosio[1] discovered that death in children and illnesses in adults were sometimes caused by arsine generated by the action of *S brevicaulis* on arsenical wallpaper pigments, although it is now known that arsine could be similarly generated from the horse-hoof size that was commonly used at that time and which contained white arsenic to discourage rodent damage.[2]

Phosphine, arsine, and stibine are exceedingly toxic with threshold limit values of 0·3, 0·5 and 0·1 ppm, respectively, compared with 100 ppm for carbon monoxide. Relatively high ambient levels prevent the detection of phosphorus, arsenic, and antimony residues at necropsy. However, some pathologists have commented that they usually associate SIDS with abnormal lung colour; this could be associated with local erythrocyte haemolysis due to exposure to these trihydride gases.

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**A Government inquiry, prompted by these findings, was announced on March 9. The inquiry will be chaired by Prof Paul Turner.

MSB

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Mr Martin carried vital statistics that allow his small tailors' shop in Sligo town to take on London competition. Even big Sligo money cannot pay this. Mr Martin admits that the local starting sewn up. Transport is by a 20-seater aircraft — with a change in Dublin — on the first Thursday among bales of tweed, worst and cashmeres, skeins of hi silk and cops of machine threa

Inquiry into 'cot deaths link' to PVC mattresses

The Independent 10.3.90

By Liz Hunt
Medical Correspondent

A GOVERNMENT inquiry into claims that toxic gases released from PVC mattresses can cause cot deaths was announced by Sir Donald Acheson, the chief medical officer, yesterday.

Research has suggested that the gases are produced by a common household fungus which reacts with the chemicals used to make mattresses fire retardant.

It is claimed that the fumes act on a baby's nervous system and suppress its breathing.

Sir Donald said that there was no reason for parents to be concerned, and that the inquiry was "a matter of prudence".

"We have a duty to investigate possible causes," he said.

A team of independent experts, lead by Paul Turner, professor of pharmacology at St Bartholomew's Hospital and chairman of the Government's expert advisory committee on toxicity, will review research carried out by Barry Richardson, a consultant scientist and specialist in the deterioration of materials. Mr Richardson, a director of Penarth Research International in Guernsey, which has funded the £50,000 research, announced his findings last June. They have not yet been published.

The Government team will also look at the results of an investigation into Mr Richardson's claims by the Laboratory of the Government Chemist. The Foundation for the Study of Infant Deaths is carrying out its own investigation. There are about 1,500 cot deaths each year.

Sir Donald said that the findings so far were inconclusive. Government scientists had not "conclusively replicated the original findings".

"This is nothing for parents to worry about. From the expert advice taken, we can reassure them that there is no cause for concern."

Sir Donald said that "no case of cot death in this country due to the toxic gases arsine (from arsenic), stibene (from antimony), and phosphine (from phosphorous) has been reported".

Autopsies had not revealed the expected changes in kidney tissue or in red blood cells caused by acute arsine poisoning, or clinical changes associated with mild poisoning, Sir Donald said.

Mr Richardson began his research in 1988 while advising Peter Mitchell, of Mitchell Marquees, on the deterioration of industrial PVC used in tents and awnings.

Some of the deterioration was caused by a common fungus, *Scopulariopsis brevicaulis*. It was acting on one of the arsenic-containing biosides used to preserve tents, and producing arsine.

A possible link with cot deaths was made by Mr Mitchell, who hires out tents and marquees, when he contacted the manufacturers of the bioside for reass ances about its safety.

"He was told that it was so s that it was used on cot mattres and nappy pants. He rang early one morning and told that he hadn't slept for worry about it," Mr Richardson said.

Mr Richardson began his search when cot matresses start to arrive at his home in Winch ter.

Peter Mitchell had written every coroner in England a Wales asking for the mattres used by cot death victims so th could be examined.

Mr Richardson examined mattresses — and some used healthy babies — and found th were infected with the fungus. E periments showed that the fung on a PVC mattress did produ the gases, as expected, he said.

Mr Richardson claims his th ory fits in with what is alrea known about cot deaths. They c cur in fewer first-born babies, w tend to have new mattresses.

Nuclear staff call for radiation level cuts

A REDU ION in radiation dose limits to one fifth of the cur- level is being demanded by more than 10 milliSieverts in one year, and no more than 5 mSv in any six-month period, the unions

Stepfather 'tortured girl for bed wetting

A STEPFATHER who kicked a girl of four about "l t football", dumped her in cold baths, forced her to stand naked in a

Recorder James Crespi Q sentenced Nequaye, of W worth, south-east London, to fo years. The judge said he "ter

ASG

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COT DEATH

The British Plastics Federation is aware of the article, 'New Clue probed over cot deaths' which appeared in the 'Exclusive' column of the Daily Express on 26th February 1990.

To test the accuracy of the statement made in the article we have been in touch with the Department of Trade and Industry and the Laboratory of the Government Chemist.

From these enquiries it is apparent that a report has not been issued on the subject of an asserted link between the phenomenon of cot death syndrome and fire retardant chemicals by either the Department or the Laboratory. Crisis meetings, involving government ministers, as reported in the Express article, have not taken place.

The Laboratory of the Government Chemist is proceeding with its investigation in this field. We are awaiting a full report from the Laboratory but we do understand that the allegations made in the article, whatever their source, cannot be substantiated.

For further information contact:

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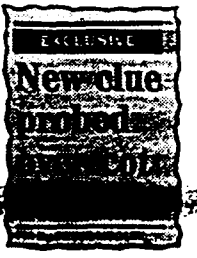
Clarke acts over mattresses alert



HEALTH Secretary Kenneth Clarke yesterday ordered an immediate investigation into the possible link between cot deaths and children's fireproof mattresses.

The move followed yesterday's exclusive revelations in the Daily Express that Government scientists had discovered fire retardants can react to emit deadly quantities of deadly gases around a sleeping infant.

There is increasing concern at Westminster that civil servants have not kept Ministers up to date on vital independent research findings.

As Chief Medical Officer Sir Donald Acheson met top scientists yesterday, the Department of Health said evidence for a link between fire-retardant chemicals and the 2,000 cot deaths a year was still being checked.

Earlier this month Health Minister Virginia Bottomley ordered Government scientists to investigate the possibility.

Toxic

But the Daily Express can reveal that results of at least two experiments confirm independent research to which the Government was first alerted 12 months ago.

Ministers were also told yesterday that cot deaths rose 12 to 15 per cent in 1986 and have continued to increased since.

It was then that foam manufacturers, increasingly worried about toxic fumes given off in household fires, started treating their products with fire retardants.

Mr Clarke is prepared to order tens of thousands of baby mattresses to be withdrawn from sale if Government scientists confirm a possible link.

"He is determined to find out what is going on and will not hesitate to act," an aide said last night.

Sir Donald Acheson was under pressure to issue urgent guidelines to parents who have already bought fireproof mattresses, which have to conform to new fire safety laws from September.

"The Government's failure to grasp the nettle over this is simply frightening," said independent biochemist Barry Richardson.

He has spent £50,000 of his own cash and thousands of hours since he discovered the cot death link by chance.

"What I discovered is not remarkable. It is so blindingly obvious that it is amazing it was not considered before," he said.

Danger

"I now hope the Government will take note and ensure all new mattresses do not contain the fire retardants that give rise to danger."

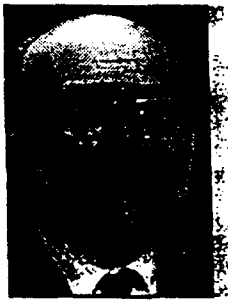
Mr Richardson's findings link cot deaths with a chemical reaction between a common invisible household fungus and phosphorus and antimony compounds used in fire-retardant mattresses.

The reaction can also be triggered by preservatives used in PVC mattresses, which some scientists now say may be responsible for the huge rise in British cot deaths since 1971.

The Department of Trade and Industry was urged last night by baby product firms to make a statement.

"We are in a cleft stick," said Nursery Shop boss Corrine Flounders, a qualified health visitor.

"Many of us are extremely worried about this. But if we do not comply with government regulations we will be breaking the law."


Acheson: Pressure


Bottomley: Probe

WHAT PARENTS SHOULD DO

LEADING biochemist Barry Richarson, who pioneered research on mattresses, yesterday issued his own safety code for parents.

He urged:

- **All new mattresses should be washed down once a week with disinfectant.**
- **All old and second-hand mattresses should be securely wrapped in a clear polythene sheet, obtainable from ironmongers.**
- **The polythene should be wrapped tightly and properly secured with adhesive tape on the bottom.**

"I advise people with carry-cot mattresses to throw them away and use a doubled-up cotton sheet instead," he said.

He tested 45 mattresses on which babies had died.

They were made from foam, PVC and cotton. All had been fire-proofed. In tests all gave off potentially lethal doses of poison gases.

By PETER HOOLEY
Whitehall Correspondent

Now Bush guns for Castro

From Page One

almost 11 years in power. U.S. intelligence reports indicated the result will have a pulverising effect in Havana, already quaking over the worldwide disintegration of Communism.

Cuba's state-run radio, TV and newspapers had predicted a near-certain win for Ortega's Leftist Sandinista movement.

Castro, 63, was devastated as the first returns came in. His gloom deepened when Ortega conceded defeat. Cuba's Radio Rebelde described his surrender of power as "one of the bravest political lation were born after he took over in 1959.

He has erected signs along country roads exhorting: "Long live rigidity!" and "Socialism or Death".

His rhetoric was beginning to sound like a death-rattle, especially in Washington where his every move is monitored.

Austerity

"People who lived through the revolution had a sense of what they were fighting against," said a State Department analyst.

"The kids who have grown up since don't. All they have been told is austere style, is only 90 miles away in Florida.

Yesterday's pounding of the Communists in Nicaragua was beamed to them from U.S. radio and TV stations in Miami where 700,000 Cuban exiles are growing increasingly optimistic about Castro's downfall.

The U.S. Congress gave mother-of-four Mrs Chamorro more than £1 million for her low-key campaign and she is believed to have received five times that amount through the backdoor.

Almost everyone in Nicaragua is on the poverty line. It has been torn by war for most of the past decade with the U.S. ploughing in millions of pounds in aid to

New IRA warning

THE GANG responsible for two town centre blasts in a week may go for civilian targets next, security chiefs warned last night.

The alert followed the explosion at an Army recruiting centre in Halifax, West Yorkshire, late on Sunday night. Nobody was hurt. Police are linking it with the IRA bombing of an Army van in Leicester last week.

Anti-terrorist chiefs

from Robin Chaze

EXCLUSIVE

New clue probed over cot deaths

By PETER HOOLEY
Whitehall Correspondent

STARTLING evidence by government scientists has confirmed a possible new link between cot-death syndrome and fire-resistent mattresses.

It could lead to emergency guidelines to parents — or the recall of tens of thousands of recently-sold fire-retardant baby mattresses.

Ministers begin a series of crisis meetings in Whitehall today after it was found that chemicals used in fire-proofing to comply with new fire laws, designed to make cots safer, could actually cause the deaths of babies.

Scientists have found the fire retardants can form part of a chemical reaction which emits minute quantities of some of the most deadly gases known to man and create an invisible cloud of death around a sleeping infant.

Many thousands of such

Page 2 Column 1

Target 36, Sport 38 - 44

Eric Forth: Talks

Cot death: New clue

From Page One

mattresses have already been produced and sold in Britain's baby shops in readiness for new fire safety laws on babies' furniture which come into force from September 1 this year.

The findings could force Consumer Minister Eric Forth to change his mind and give a last-minute emergency exemption from the new match-test rules for all nursery furniture.

And Health Minister Virginia Bottomley will be pressed to issue urgent new guidelines

The cot-death syndrome, which results in the death of 2,000 of the 80,000 babies born in Britain every year, has never been fully explained.

Reaction

Scientists have noted an increase in victim numbers since the fire retardant chemicals became widely used.

Whitehall sources said yesterday research by Laboratory of the Government Chemist is believed to have confirmed the possible link.

A chemical reaction occurs between an invisible and common household fungus, usually present on mattresses, and the chemicals used in the fire retardant, phosphorous and antimony compounds.

Fungal activity was made worse by the nitrogen content in perspiration and babies' urine.

● A young mother's tragic mistake with an electric blanket led to the deaths of her four-week-old twin boys. Kurt and Damon Hardman died from heat exhaustion after Mrs Karen Hardman, of Kirkham, Lancashire forgot to switch off the blanket.

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Speed up cot death research, Prime Minister urged

WINCHESTER M.P., Mr. John Browne has asked the Prime Minister to speed up the Government chemist's inquiry into the cause of cot deaths.

In the meantime, he has called for a Government warning to be issued to parents as a matter of urgency.

Mrs. Margaret Thatcher, the Prime Minister, replied this week: "I share the great concern of parents and Health Service staff about the incidence of unexplained cot deaths, the suddenness of which is especially tragic for the families.

"The Department of Health has promptly commissioned the laboratory of the Government chemist to undertake a scientific assessment, which is under way."

Continued Mrs. Thatcher: "I understand that, so far, those tests have not reproduced the result obtained by the independent researcher who proposed a link with fire resistant chemicals."

She added that the Government would consider whether it would be right to issue warnings in the light of the scientific assessments now going on.

Afterwards, Mr. Browne said one of his constituents, Mr. Peter Mitchell had raised the question with him at a meeting and had referred to the research of another constituent, Mr Barry Richardson.

Mr. Browne said: "Mr. Richardson's researches show that when a baby's body liquids come into contact with fire resistant additives (e.g. antimony trioxide) and preservative additives (e.g. OBPA) the resulting reaction can, over a period of time, cause the growth of a fungus. If this fungus is then heated by the body temperature of a subsequent baby, highly toxic gases can be given off such as Stibine, Phosphine and Arsine."

Mr. Browne added: "It appears, and I stress the word appears from samples provided by Mr. Mitchell and studied by Mr. Richardson, that these gases were the likely cause of the majority of cot deaths, currently about 2000 each year in the United Kingdom.

"Obviously, a used mattress was much more dangerous because the fungus had time to grow before being heated by subsequent babies. It was interesting to note that the incidence of cot deaths increased in winter when babies are heavily wrapped and central heating switched on. The images show that stronger babies were able to throw open some of their bedding and escape the effect of the toxic fumes.

Mr. Browne said: "I realise that the Government is looking into the matter, but there is need for more urgency. I myself have friends who have suffered the trauma of a cot death and I know the anguish that this causes, especially for the mother.

"In the meantime I think the Government should issue a warning to parents advising them to be careful of second-hand or previously used cot mattresses, that may have been soiled and may contain the deadly fungus."

The British Plastics Federation
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JCF/AC

24 August 1989

Status and publication details of the Laboratory of the Government Chemist research into the allegations made by Mr Richardson

The BPF has been advised to liaise directly with Dr Raymond Cooke of the LGC every 2 months (links have already been established) and it is anticipated that the LGC work will take approximately 4 months to complete.

It is likely that the research will be described in an article in Chase Bulletin however, no conclusions will be drawn. Conclusions will only be made available to the DTI's CSU directly and will not be made public. The CSU may well retain a statement in its files which could, for instance, be used to respond to Parliamentary questions.

Details of the research may also appear in the house journal of the LGC, published quarterly. This information is then passed on to the DTI, the Government Research Establishment and other academic/research bodies.

If an industry has been closely involved in a particular issue being researched by the LGC then it is usual for them to be consulted before details of the report are finalised. It is therefore probable that the BPF will have sight of the LGC's draft report.

It is the current view of the CSU and Department of Health that Mr Richards reports is very much a 'dead-letter' with little, if any, foundation on fact.

Jacky Freer
Group Executive

cc MSB

The Vinyl Institute

A Division of The Society of The Plastics Industry, Inc.

TELECOPY TRANSMITTAL FORM

TO: Harold Clayton @ Hydro Polymers

FROM: Roy T. Gottesman

DATE: April 17, 1990 #/PAGES: One plus cover

SUBJECT: British Report on "Cot Deaths"

COMMENTS: Harold:

I have just learned that there was a radio report on NBC by a Roger Field dealing with the Lancet report on "cot deaths" and the interpretation that stibene, arsine, etc. liberated by fungi acting on the fire retardants in PVC cot mattresses are suspects in causing these deaths. Today, Pat Toner, V.P. Technical of SPI, talked with Mr. Field who is now working on a story on this for the New York Times. In response to Mr. Field's inquiries, Pat Toner advised Fields that all we have seen is a "letter to the Editor" in Lancet which is lacking in technical details and that until our technical experts have an opportunity to review the technical work on which this report is based, we'd have no comment.

Continued .....

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Would you please gather together copies of all information you have on this subject and bring them with you to the Brighton Conference? I'd greatly appreciate that as it will provide Pat and me a total package of information for our review. Also I would look forward to the opportunity of discussing this with you at Brighton.

Thanks in advance for your assistance in this and I look forward to seeing you next week. Kindest personal regards.

Roy

cc: H. Patrick Toner, SPI -Washington

MSB file

29th March 1990

PVC
Stuurgroep PVC & Milieu

[Netherlands Action Group PVC and Environment]

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.

2248094

* given.

PVC
&

Stuurgroep PVC & Milieu

PERSBERICHT

Wiegedood

Naar aanleiding van berichten over een mogelijk verband tussen kindermatrasjes met een PVC bekleding en wiegedood het volgende:

De eerste publicaties over het mogelijk verband tussen in PVC toegepaste brandvertragers en wiegedood verschenen begin 1989 in Engeland, waar een onderzoeker van Penearth Research International een mogelijk verband aangaf tussen wiegedood en brandvertragende toevoegingen aan de PVC omhulling van kindermatrasjes.

Een publicatie van de onderzoeksresultaten werd door de British Medical Journal geweigerd op grond van te magere wetenschappelijke onderbouwing.
Het Engelse Ministerie voor gezondheid en veiligheid en milieu(HSE) verwierp vervolgens de door de onderzoeker naar voren gebrachte argumentatie.
De gepresenteerde onderzoeksresultaten waren dan ook voor de Britse overheid geen reden om enige maatregel te nemen tegen de door de industrie gebruikte brandvertragers.

In juni 1989 werd echter alsnog besloten tot onderzoek door het laboratorium van de Engelse overheid, daar men terecht van mening was dat elke mogelijke aanwijzing, hoe gering ook, die tot de oplossing van een tot nu toe onverklaarbaar probleem kon leiden in ieder geval onderzocht diende te worden.

Tot op heden heeft men geen aanwijzingen kunnen vinden voor een mogelijk verband.
Gezien de nu bekende gegevens ziet de Stuurgroep PVC & Milieu de uitkomsten van het in Engeland uitgevoerde onderzoek met vertrouwen tegemoet.
Indien echter de resultaten niet tot een duidelijke conclusie leiden, of na de nu voorziene ca. 6 weken niet bekend zijn, zal de Stuurgroep, eventueel samen met de Nederlandse overheid, tot een eigen onderzoek besluiten.

Het spreekt vanzelf dat de Stuurgroep PVC & Milieu berichten over een mogelijk verband tussen PVC en wiegedood zeer serieus neemt. Het is buitengewoon belangrijk dat alle mogelijke inspanningen worden verricht om het mysterie tot een oplossing te brengen. Het is echter van groot belang dat juist in een zaak waar, begrijpelijkerwijs, emoties een grote rol spelen de feiten een hoofdrol blijven vervullen.

Noot voor de redactie: voor meer informatie kunt U zich wenden tot H.J. Moonen, voorzitter van de Stuurgroep PVC en Milieu, Tel. 020- 934844

p.a. NFK
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JF/AC

24 August 1989

STRICTLY CONFIDENTIAL

To: Participants in BPF Forum on Cot Mattress Biodetioration

You will recall that at our meeting on 12th June a Task Force was established to produce a critique of Mr Barry Richardson's study. Initial work has now been completed and the second draft is attached for your information.

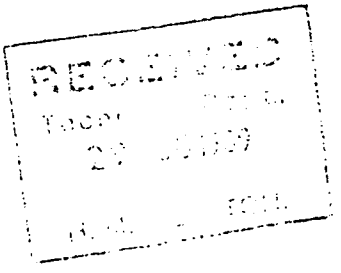
Would you please study the report and, if you feel that there are any questions still to be addressed or areas which require clarification or revision, please contact me immediately.

Obviously, as this is such a sensitive issue the report is strictly confidential to member companies involved. Please do not discuss it outside your company.

Also attached are brief notes to a conversation with a representative of the DTI's Consumer Safety Unit which outline the status and intended publication details of the Laboratory of the Government Chemist's research.

A meeting of interested parties will be called in approximately 4 weeks to take stock of the situation. I will be contacting you shortly to establish a convienent date.

Jacky Freer
Group Executive



2248096

Director - D R Jones
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COT MATTRESS BIODETERIORATION AND SUDDEN INFANT DEATH - 2ND DRAFT

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 pPVC materials in cot mattress and the tragic phenomenon commonly known as 'cot death'. His report is the subject of 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

- medicine

Five principal questions are raised by Mr Richardson's report:

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

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

iii) Do these emissions actually take place in situ?

iv) If so, are the emissions injurious to health?

v) 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 has not produced any evidence to indicate that he has analysed the pPVC cot mattress coverings to determine their composition - he has assumed their composition only from the results of his experiments.

The report states that "all cot PVC contains antimony trioxide and phosphate plasticisers" (p.4). Whilst 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 they are never used together in a formulation. Mr Richardson is wrong in his assertion that 'manufacturers are now using antimony 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.

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

Mr Richardson's conclusions rest on the role of a particular fungus, 'Scopulariopsis brevicaulis', which he says develops on pPVC cot mattresses 'if adequate moisture and protein are present, perspiration providing these requirements'. It is this, he says, which liberates arsine, phosphine and stibine and the strong implication is that he has carried out laboratory investigations (however vague) (p.5) to verify this (p.4). However, he presents no evidence of identification of the fungus from samples of infected mattresses. He claims the fungus has been detected but gives no quantitative data about its presence, such as numbers per square millimetre or amount of hypha per square millimetre.

All experiments to which Mr Richardson refers and from whose results he bases his conclusions are dated, crude and not specific to the detection of the gases in question. He gives no information about the time-scale of the experiments or the particular length of time taken for colour to develop.

His use of silver nitrate test paper to indicate the presence of the hydrides of phosphine, arsine and stibine is very crude. Silver nitrate only provides a qualitative means of detecting the presence of hydrides. Hydrogen sulphate, ammonia or hydrogen chloride (whose absence would have to be positively ascertained) can also discolour silver nitrate and, in addition, this paper naturally turns black in the atmosphere over time anyway.

Mr Richardson does not scientifically prove the absence of potential interfering substances (for which credible sources in his experiments exist) and in addition has no adequate controls in his experiment which could address the following important points:

- 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 results therefore may just be artefacts of his methodology.

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 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 such a temperature as many fungi will not grow at 37°C.

5. Are the emissions injurious to health?

The quantities of gases generated in cots under normal conditions are likely to be extremely small and uninjurious to health (Mr. Richardson did not measure these). Mattresses currently manufactured have holes cut into the foam at the head end to facilitate a child's breathing and in these cirumstances, if phosphine, arsine and stibine were generated in the way described by Mr Richardson, they would be well diluted in air. 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's density of 1. Such a difference is trivial 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 hypothesis.

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 probably due to their higher level of health education and social conditions.

- the rate for Hong Kong of 0.3 per 1000 is likely to be due to the over-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).

One would expect a post-mortem to indicate the presence of toxins in a childs body and Mr Richardson's argument around this is spurions.

Authoritative organisations and close students of cot deaths, such as the Foundation for the Study of Infant 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 contributory factor in its cause 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 conclude that there is no direct evidence of an association between gaseous intoxication and sudden infant death.

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24 August 1989

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It is the current view of the CSU and Department of Health that Mr Richards reports is very much a 'dead-letter' with little, if any, foundation on fact.

Jacky Freer
Group Executive

2248109

2248101

Chatty Di opens anti-drug centre

by Peter Woolrich

PRINCESS Diana opened a drug rehabilitation centre in Hackney today.

Hundreds of onlookers cheered as the princess arrived at the Lorne House centre in the Lower Clapton Road.

Wearing an aquamarine suit with a trendy high-cut jacket, she spent an hour touring the purpose-built unit and talking to care experts.

As the Princess left she surprised security men by going over to the crowds to shake hands, and chatting with Salvation Army workers next door.

Lorne House is the first centre of its kind to provide a unified approach for 18-to-25-year-olds with combined problems of alcohol and drug dependence.

After her chat over a garden fence with the Salvation Army workers, the Princess moved on to accept dozens of bouquets from excited schoolchildren.

She blushed as one older girl told her that she looked "absolutely gorgeous", and asked the princess to say hello to Charles.

SAYING IT WITH schoolchildren in l

LOWERS: Princess Diana, her arms filled with bouquets from ckney today

Picture: ALISON McDOUGALL

Toxic gas theory on cot deaths

by Alan Massam

THE mystery cot death syndrome may be caused by babies breathing toxic gases generated from PVC mattress coverings, scientists now believe.

A team of researchers have come up with the explanation for sudden infant death syndrome, which kills 2000 babies each year in Britain.

The discovery was announced today by Guernsey-based Penarth Research International. But Britain's leading research body into the problem, The Foundation for the Study of Infant Deaths, condemned it because it was "only an unproven hypothesis".

"We have been inundated with calls from worried parents," a spokeswoman said.

Penarth claim that mattresses tested included those only half covered with PVC. It was found that they were likely to harbour fungi which were capable of giving off gases like arsenic trichloride when they reacted with the plasticisers used to make PVC flexible.

The company believed that the usual circumstances of sudden cot deaths were consistent with poisoning by gas heavier than air accumulating in bedding. Infants usually died face down when they were heavily wrapped against the cold.

Deaths often occurred on mattresses which were not new and in which fungi organisms could have become established.

Mattresses supplied by parents who had recently lost babies were contaminated with the fungus scopulariopsis brevicaulis, particularly in stitching.

The researchers also considered that foam coverings of the top half of mattresses supported fungal infections which could provoke allergic reactions.

The Foundation for the Study of Infant Deaths stressed that epidemiological evidence did not support the Penarth claims because cot deaths occurred long before PVC was used on mattresses.

The Penarth research is supported by Dr Peter Fleming of Bristol Maternity Hospital's Department of Child Health.

BPF

6.6.89.

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Attention: Mr H.M. Clayton

Harold

Please find attached the text of a holding statement which BPF issued to the media tonight on the cot-deaths issue.

Regards

Philip Law

(2 sheets)

22480102

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Fax 01-235 8045

The British Plastics Federation is aware of the research report that has been produced which links the incidence of Sudden Infant Death Syndrome ("cot deaths") with certain additives applied to PVC used in the manuracture of children's bedding.

The BPF is obviously concerned and has requested an independent assessment of the report's findings as well as obtaining its own experts' opinion.

After initial reading, the report appears not to involve PVC directly with these tragedies.

An immediate appraisal of the materials used is being carried out with the manufacturers.

In the meantime concerned parents should follow the quite normal hygiene procedures when dealing with infants and if in any doubt seek medical advice.

For more information:

Philip Law
British Plastics Federation
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London SW1X 8PH
England
Tel: (01) 235 9483

The British Plastics Federation
5 Belgrave Square · London SW1X 8PH
01-235 9483 · Telex 8951528 Plaied G

TO: H. M. CLAYTON

FROM: PHILIP LAW

DATE: 4.6.89.

NO. OF PAGES (INCLUDING FRONT): 3

IF ANY DIFFICULTIES ARE ENCOUNTERED DURING TRANSMISSION,

OUR FAX NUMBER IS: 01-235 8045

MESSAGE: Harold

The essence of our phone message attached

Regards

Philip Law

2480104

Have been contacted by the Consumer Affairs Unit of the D.T.I. asking us to comment on an asserted link between PVC and cot deaths. They have been sent a report by:

Barry Richardson.
Technical Director
Penarth Research International Ltd.
St. Peter Port.
Guernsey

who claims that plasticized PVC in cot mattresses gives rise to emissions of Phosphine, Arsine and Stibine. He says the of 9 mattresses examined, 8 contained 2 fungus which develops on plasticized [illegible] adhesive agent.

2480105

Robin Fielder, a toxicologist in DHSS. doesn't feel that the report can be ignored and we have been asked to supply data on the antimony trioxide and phosphorous content of plasticised PVC.

Meanwhile Richardson claims to have an article prepared for British Medical Journal and has been interviewed by Sky Net.

We will keep you posted.

Regards,

Philip Law

224801116

PVC fume link to cot deaths dismissed

By Pearce Wright
Science Editor

A theory that cot deaths are linked to poisonous fumes released from PVC plastic cot mattresses has been widely dismissed by experts.

The *Department of Health* says there is "no adequate validation" for the idea, which has caused alarm among many parents.

The scientific advisory committee to the Foundation for the Study of Infant Deaths, which has spent £4 million in the past 17 years on research, says the hypothesis contradicts the findings of painstaking investigations into factors that may trigger the deaths. The *British Medical Journal* also rejected the theory.

Dr John Grange, consultant bacteriologist at the Brompton Hospital, west London, said in a devastating assessment of the report for *The Times*, which was circulated this week, that more detailed studies were needed to provide a more definitive answer.

The controversy centres on the details of a report by Mr Barry Richardson, director of a scientific consultancy service, which describes the laboratory experiments on which the suggestions about cot deaths are based.

Dr Grange described the experimental methods as "using crude equipment, lacking an obvious control experiment that would reveal flaws and irrelevant as a representation of a cot mattress".

Mr Richardson maintains that a particular type of micro-organism common in homes can react with traces of chemicals used in the manufacture of PVC to generate poisonous fumes.

A key to the process is mattresses must also have a protein content on which the micro-organisms thrive.

The laboratory experiments involved placing strips of PVC and organisms on a rich nutrient mixture of soya flour and malt. Dr Grange said that that bore no relation to mattress conditions and traces of protein it might contain.

Moreover, advanced instruments would be needed to identify the type and amount of gases generated. The report contains no figures of the quantity of gases which came from heating those cultures.

Dr Grange said they could have been generated from the nutrient mixture rather than the PVC. He suggested that Mr Richardson had conducted for the best of motives a completely irrelevant

(12)

12th June 1989

Chemical Industries Association Limited

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With compliments

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AUTO879

TELLEX
THE BROADCAST REPORTING SERVICE
REPORT

PVC - LINKS WITH COT DEATHS

For :	BRITISH PLASTICS FEDERATION		KEITH HANMAN
Prog :	HARALD FUCHS	Service :	BBC SHROPSHIRE Serial : 066232/JD
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TELLEX Monitors Limited 36 Paradise Street, Birmingham B1 2AJ Telephone: 021-643 4177

HARALD FUCHS:

PVC mattresses could be the cause of cot deaths, according to a British research company. Penarth Research Laboratory tested twenty one mattresses, including some from Shropshire, on which cot deaths occurred. Jonathan Beale asked the Technical Director of the company, Barry Richardson, what his investigations had revealed.

BARRY RICHARDSON:

Our research was part of a programme of investigating deterioration of PVC fabric and we became aware that a particular fungus might infect fabric and might attack some of the additives that are commonly put into the fabric as preservatives and fire retardents (sic). And this fungus would then cause toxic gases to be produced from the fabric.

JONATHAN BEALE:

So what would actually happen to the baby then lying on the bed?

B.R:

Well in the case of the cot mattresses investigations that we did we found that the infection is always naturally present underneath the baby. It develops after a few months. The... depending upon how long a baby is left on the mattresses actually, and it only develops just beneath the baby where the mattress is warm and moist from perspiration. The gas, we find, is always produced from the in...from infected mattresses. Everyone that we have examined is producing this gas when we incubate it. That means we put it in conditions that are similar to cot conditions. The gas is produced, the main one is called dibean* and it's exceedingly toxic. A thousand times more toxic than carbon monoxide. So I'm afraid I'm not talking about anything that is mildly toxic or anything like that. We're talking about one of the most toxic gases we know. What we actually have is the babies who are dying are generally sleeping apparently in the face down position, and they're generally quite heavily wrapped during cool weather.

J.B:

(Phrase inaudible)...to the incidence of cot death, particularly in relation to Shropshire?

*Phonetic spelling

B.R:

When deaths have occurred some of the coroners offices who have heard of our work have asked the parents whether they could provide us with the mattresses on which the infants were lying. We've received mattresses, including mattresses from Shropshire.

J.B:

What's your reaction to people who've actually disclaimed this theory? For example, the Foundation for the Study of Infant Deaths says that your work is unproven. What's your reaction to that?

B.R:

The work that I have done on the generation of the gas from plastic is quite firmly proven. What is unproven is the significance that this work may have, or this poisoning may have in relation to actual cot deaths. That's not my job. I'm afraid the medical people must take my work into account and see whether it does relate to the cot deaths that they're investigating.

J.B:

What precautions can parents take?

B.R:

Well I think in the long term, of course, parents will have to replace mattresses with safe mattresses. There's no objection to PVC, I must emphasise that. The problem lies with the additives that are used in current PVC. So obviously the mattress manufacturers will producing safe mattresses I imagine quite shortly.

H.F:

Barry Richardson of Penarth Research Laboratory talking about his investigations into cot deaths.

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REPORT

COT MATTRESS BIODETERIORATION

AND

SUDDEN INFANT DEATH

Barry A Richardson

P 1

June 1989

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SUDDEN INFANT DEATH

Barry A. Richardson

Introduction

Sudden infant death, commonly described as cot death, accounts for a significant proportion of postneonatal mortalities, usually involving infants which are not observably ill prior to death. The causes remain unexplained despite extensive research, many recent studies concentrating on statistical interpretations of the circumstances of death as obvious explanations seem to have been eliminated.

It has been generally established that sudden infant death syndrome is the commonest cause of death between 1 and 12 months, and accounts for about 2 deaths per 1,000 live births in the United Kingdom, 2 to 4 per 1,000 generally in western countries, and about 6 per 1,000 in New Zealand, but only about 0.3 per 1,000 in Hong Kong, and very low incidence in Russia, China, India and Africa, and amongst infants of African or Asian origin in England and Wales (1,2,3,4,5,6). The very high death rate in New Zealand is due to exceptional rates of about 11.5 per 1,000 amongst Maori infants, the rate for Caucasian infants at about 1.6 per 1,000 being lower than most western countries (7). Deaths occur unexpectedly during sleep, 80% of the deaths affecting infants between 1 and 5 months. There are proportionally more deaths in the winter and more at weekends, with boys more at risk in countries where high death rates occur. The risk is higher with premature births at low birth weights, twins, smoking mothers, unemployed fathers, single mothers and poor housing conditions, perhaps because all these factors tend to result in infants with lower weights than normal for their age. Deaths sometimes follow observations of general malaise when infants have digestive disorders, refuse to feed or are difficult to wake.

Recent hypotheses have concentrated on the upper respiratory tract. It has been noted that there has been a threefold increase in sudden infant deaths in The Netherlands from 0.46 per 1,000 for 1969-71 to about 1.31 per 1,000 since 1978 (8). The obvious explanation for this increase is that sudden infant death syndrome is now recognised statistically, but this increase has been attributed instead to the general adoption of a prone or face-down sleeping position following recommendations that it was preferable at a paediatric conference in 1971. It has been suggested that sleeping in the prone position presents more risk of obstruction of the upper respiratory tract (9,10). Viral and bacterial infections have also been reported, and may be associated with the prone sleeping position (11,12). In a recent review it was pointed out that sleeping in the supine position may avoid these problems but create others (13). It has also been suggested that the prone position might result in hyperthermia as the face is the most important cooling surface and temperature control element, particularly in infants protected in the winter by extra coverings (14,15).

Another possibility with heavy bed coverings and cots with high impermeable sides is that the infant is affected by accumulations of an heavier-than-air gas, the prone position aggravating exposure. The most obvious gas is carbon dioxide generated by the infant itself. High carbon dioxide concentrations would normally stimulate hyperventilation, although infants at risk may suffer longer attacks of apnoea and may not be able to hyperventilate to a sufficient extent to clear the accumulating gas from a cot and bedclothes reservoir(16,17). In such circumstances the carbon dioxide would be converted eventually to toxic carbon monoxide, such a conversion perhaps explaining why sudden infant deaths are particularly associated with week-ends when an infant may remain unchecked in the cot for a longer period(6). Carbon monoxide poisoning has not been reported and is unlikely as this gas is lighter than air and likely to disperse, but such an explanation would be consistent with the slow release or formation of a toxic heavier-than-air gas. Recent investigations by Penarth Research International Limited into biodeterioration of the plasticisers in polyvinyl chloride indicated that three extremely toxic heavier-than-air gases phosphine, arsine and stibine might be generated, and this report therefore considers the possibility that these toxic gases from mattress covers, cot sides and nappy pants might be a cause of sudden infant death.

Phosphine, arsine and stibine

Group V/Vb elements form gaseous trihydrides. Nitrogen trihydride is best known as ammonia, but phosphorus, arsenic and antimony similarly form phosphine, arsine and stibine gases which are 1.17, 2.68 and 4.29 times heavier than air respectively. The threshold limit values for phosphine, arsine and stibine are usually quoted as 0.3, 0.05 and 0.1ppm respectively, about 300, 2000 and 1000 times more toxic than carbon monoxide which has a TLV of 100ppm. All three gases are colourless. Phosphine has a 'dead fish' or 'garlic' odour, arsine is more distinctly 'garlic', and stibine odour is usually described only as 'very unpleasant'; with arsine the garlic odour, which may be associated with the formation of alkyl compounds, is only detectable at concentrations well in excess of the TLV.

Arsine

The typical symptoms of acute arsine poisoning in adults, which usually develop between 2 and 24 hours after exposure, are headache and dizziness with general weakness and malaise, followed by thirst, nausea, vomiting, diarrhoea and abdominal pain, often with a burning sensation of the face and chest discomfort accompanying dyspnea and pulmonary oedema(18). Red urine may develop 4 to 6 hours after exposure, followed by oliguria, anuria and anaemia. Hepatomegaly and jaundice follow after 24 to 48 hours. Polyneuropathy and encephalopathy have also been reported.

Chronic exposure is usually reported to have a cumulative effect. This is not strictly correct as arsine is eliminated by conversion and excretion in the urine, but anaemia caused by chronic exposure obviously becomes progressively more severe; at very low air concentrations poisoning is characterised by shortness of breath and general weakness resulting from this anaemia. The mode of poisoning includes erythrocyte haemolysis through glutathione reduction which continues after exposure ceases, followed by haemorrhagic hepatitis and

nephritis with tubular necrosis. Arsine is a depressant of the central nervous system, and prolonged exposure may affect the bone marrow. Exposure to 10ppm for 1 minute or 3ppm for 30 minutes is sufficient to cause fatal or severe toxic reactions, and 1ppm for long periods can be exceedingly dangerous.

Arsine poisoning is usually diagnosed through acute haemolitic anaemia with ghost erythrocytes and increased plasma haemoglobin. Arsenic in the urine usually exceeds 0.05mg/l in acute poisoning, although in chronic poisoning continuous elimination usually means that the arsenic concentration does not exceed normal ambient levels. It has long been advised that cases of 'toxic jaundice' and 'haemolytic anaemia' should be investigated for possible arsine poisoning(19). Mild chronic arsine poisoning may be easily missed and attributed to food poisoning(20).

This description of arsine poisoning is based on observations in adults. Young infants can be expected to be more susceptible because of their low body weight but there may be other physiological factors which may further increase susceptibly such as sluggish respiration and blood circulation in deeply sleeping infants, and which may cause local rather than general anaemia in cases of acute poisoning.

Phosphine and stibine

Phosphorus trihydride or phosphine and antimony trihydride or stibine are not as well known as arsine, but they are exceedingly poisonous heavier-than-air gases with the same mode of action and symptoms as arsine. The main differences are associated only with differences in density and toxicity.

Phosphine, arsine and stibine from plasticised polyvinyl chloride

Phosphine, arsine and stibine can be generated by biodeterioration of organic and inorganic phosphorus, arsenic and antimony compounds; alkyl compounds may also be produced which are also exceedingly toxic gases. *Scopulariopsis* fungi are usually involved but *Paecilomyces* species and some wood destroying fungi such as *Lentinus squamosus* and some bacteria can also act in this way(32-52). Published reports refer only to arsine generation but phosphine and stibine can be similarly formed. Micro-organisms can only convert phosphorus, arsenic and antimony compounds to hydrides if an adequate nutrient source is also available which will provide the necessary energy.

Scopulariopsis brevicaulis, also known as *Penicillium brevicaule*, is the fungus species which is most frequently reported as generating arsine in this way in domestic situations. It is usually described as protein tolerant but it is actually protein dependent, and was usually identified in the past on silk, a protein fibre. Several classical cases of arsine poisoning have been attributed to this fungus causing biodeterioration of old wallpaper printed with Scheele's green or Paris green arsenical pigments. Napoleon's death and a mystery illness suffered a few years ago by the US Ambassadress to Italy have both been attributed to chronic arsine poisoning caused in this way, and this could be an explanation for some of the unexplained sudden infant deaths reported in the 19th century. Although this fungus is usually associated in reference books with biodeterioration of silk, it actually occurs in normal domestic air and will

develop on any suitable protein source, such as wool in carpets on damp floors or clothing stored in damp wardrobes, as well as on leather (53-58).

Scopulariopsis brevicaulis can develop on polyvinyl chloride cot mattress coverings if adequate moisture and protein are present, perspiration usually providing these requirements. Other fungi and bacteria can deteriorate PVC plasticisers, particularly *Aspergillus niger* which causes grey discolouration and *Streptomyces rubrireticuli* which causes bright pink staining; these organisms have no toxic effect but their presence indicates that conditions are suitable for the development of *S. brevicaulis* and other organisms that can generate phosphine, arsine and stibine (23,24,25,26,27,31).

The arsenic source in plasticised PVC is usually 10,10'-oxybisphenoxyarsine (OBPA), a biocide that is incorporated to prevent biodeterioration of the plasticiser in tentage and similar PVC fabric exposed to the weather, although it is also sometimes used in PVC furniture coverings for hospitals as a sanitizing agent. OBPA will control most organisms at normal biocidal use concentrations but it is slightly volatile and it is also degraded by ultra-violet light, concentrations typically decreasing progressively until colonisation by arsenic-resistant organisms such as *S. brevicaulis* becomes possible.

The antimony source is usually antimony trioxide, a white pigment which is added to PVC to improve fire retardency as it acts with the chlorine in the PVC as a flame suppressant by formation of antimony chlorides, but it appears that some manufacturers are now using antimony trioxide as a filler pigment at concentrations well above those required for fire retardency. Antimony trioxide is not significantly biocidal and stibine can be generated if conditions are otherwise suitable for microbial development. The phosphorus source is usually a phosphate plasticiser, used in preference to phthalate and similar compounds when fire retardency is required. Regulations introduced in the United Kingdom in March 1989 require PVC for nursery purposes to be fire retardent and this means that all cot PVC contains antimony trioxide and phosphate plasticisers; this is an unfortunate response to fire risk as fire rarely initiates in PVC items, and fire retardent PVC heated by fire from another source will generate much more toxic smoke than ordinary PVC.

Investigations

Phosphine, arsine and stibine generation by *Scopulariopsis brevicaulis* can be demonstrated in the laboratory. This fungus is protein dependent and will not develop strongly in the absence of a protein nutrient source. A convenient culture medium can be prepared by stirring 5% soya flour into 5% malt agar, although the soya flour can be replaced by other nitrogen sources such as ammonium citrate. Incubation should be at about 23°C. If a phosphorus, arsenic or antimony source is added to the medium or as a patch of material such as impregnated filter paper or fabric, generation of phosphine, arsine or stibine will occur. These gases are reducing agents and can be detected by darkening of silver nitrate; phosphine initially forms a lemon yellow complex, and arsine and stibine form brown complexes before black silver deposits are formed at higher concentrations. If greater sensitivity is required the culture air can be circulated through silver diethyldithiocarbamate reagent, producing a brown or orange colour for phosphine and red colours for arsine and stibine (21).

Mercuric chloride or bromide will react with arsine to produce yellow to red complexes, depending on concentration, but phosphine and stibine do not produce coloured products with these reagents. The most convenient method of detection of these gases from Petri dish cultures is to use two small strips of detection paper tucked over the edge of each dish after the growth has become strongly established, a silver nitrate paper which will turn initially yellow with phosphine and brown with arsine or stibine and eventually black with all three gases, and a mercuric chloride or bromide paper which will turn yellow or red if arsine is present. Silver nitrate paper is prepared by soaking filter paper in a 5% aqueous solution and mercuric chloride or bromide paper is prepared by soaking filter paper in a 5% alcohol solution.

PVC is often naturally infected and deteriorated by organisms, particularly *Aspergillus niger* and *Streptomyces rubrireticuli*, and laboratory experiments have demonstrated that these infections can develop if the arsenical biocide OBPA is present at an inadequate concentration and even if antimony trioxide is present at high concentrations, provided that a suitable nutrient source is available externally or within the PVC as plasticiser. It has also been demonstrated in laboratory experiments that the fungus *Scopulariopsis brevicaulis* can develop on the same PVC samples in similar conditions, a high protein nutrient source being necessary, a soya/malt agar medium being the most convenient way to introduce an external protein source in routine laboratory experiments.

Exposing small discs of PVC fabric to *S. brevicaulis* Petri dish cultures on soya/malt agar with silver nitrate and mercuric chloride paper detection is a simple means for checking whether samples contain phosphorus, arsenic or antimony sources which might cause toxic hydride gas generation under appropriate biodeterioration conditions. Checking PVC involved in sudden infant death to confirm whether toxic gas generation was probable can involve a similar technique but without inoculation, relying upon the natural infection on the fabric.

Initial cot investigations were on PVC fabric from a new mattress which did not contain any biocide, according to the manufacturer. Samples were inoculated with *S. brevicaulis* and generated gas gave a brown colour with silver nitrate and a faint yellow colour with mercuric chloride, indicating a reducing gas, probably stibine from antimony trioxide, with some arsine, probably from the arsenic contamination that is usually present in antimony trioxide. A doctor who had lost an infant then provided the mattress on which it had died. The mattress was a Visivent carry cot type mattress comprising foam with the lower two-thirds covered with PVC and the upper third covered only with a net. The mattress had been used for a previous infant but was in excellent clean condition. The PVC fabric was sampled and placed on soya/malt plates, producing the normal *Penicillium*, *Aspergillus* and other fungal cultures that are normal from domestic articles, but heavy growth of *Scopulariopsis brevicaulis* also developed, generating gas causing brown or black colouration with silver nitrate but no detectable colouration with mercuric chloride, probably stibine from antimony trioxide. Foam was sampled under the net, that is under the head of the infant, giving heavy *S. brevicaulis* infection but no colour change with silver nitrate; several other micro-organisms were also found on the foam, including *Streptomyces rubrireticuli* which is a pink staining organism which is often associated with deterioration of plasticised PVC.

The initial investigations included 21 mattresses from sudden infant death incidents, including one cotton covered mattress but all others involving PVC and some with exposed foam areas covered only by net. All PVC covers were infected with *S. brevicaulis* which was particularly heavy at stitching, although also always present in the PVC covering immediately beneath the infant; all samples infected with *S. brevicaulis* also generated gas which caused brown or black colouration with silver nitrate, probably stibine from antimony trioxide. Some samples were also infected by *S. rubrireticuli*, although the characteristic pink stain was not always visible; in one case on a mattress only 2 years old the pink stain had clearly formed under the infant and followed the body shape. Foam samples from areas covered only by net were heavily infected with a number of fungi, including *S. brevicaulis* although gas generation was not detected; the foam does not contain suitable sources of phosphorus, arsenic or antimony. It was observed that some of the fungi on the foam produce spores which are well known as causes of allergic reactions and which might cause fatal asthmatic attacks in sensitized infants (53,57,59-64).

Discussion

Phosphine, arsine or stibine gas generation through biodeterioration of plastiser in polyvinyl chloride fabric is a possible explanation for sudden infant death, particularly if infants are in closed cots or heavily wrapped. Toxic poisoning in this way is consistent with the reported circumstances of sudden infant death syndrome. A prone sleeping position, heavy coverings in cool weather, and longer undisturbed periods in the cot at week-ends all increase the risk of chronic poisoning from a heavier-than-air gas. The increasing incidence over the last 20 to 30 years is consistent with the increasing use of plasticised PVC in nursery products, and significantly lower incidence in some countries such as Russia may be associated with lack of use of PVC products in this way. The frequently reported malaise, digestive disorders and anorexia prior to death are consistent with chronic poisoning; it might be expected that some darkening of the urine would be reported in such circumstances but it is possible that death occurs before it develops. The risk is least with new cots and mattresses as they are less likely to be supporting infection.

The symptoms of phosphine, arsine and stibine poisoning have been generally reported for adults with the cautions that poisoning is not easily suspected or diagnosed (18,19). Infants are more susceptible because of their lower body weight; in an accident on the *Thermopylis*, a ship carrying grain fumigated with phosphine, 29 crew and 2 children were affected but it was a child that died (22). Infants with low birth weights might be expected to be particularly susceptible, whatever the cause of their low birth weight, because of their lower weight in relation to age during subsequent development. The high incidence at less than 12 months is probably associated with low body weight coupled with minimal ventilation due to lack of body disturbance during sleep, but the peak incidence between 1 and 5 months might be associated with physiological blood or respiration features associated with that age group.

Hydride formation by micro-organisms is well known. Sulphate reducing bacteria such as *Desulfovibrio* species can convert sulphates to sulphur dihydride, more commonly known as hydrogen sulphide, but only usually in anaerobic conditions in which the bacteria utilises sulphate as a source of oxygen; hydrogen sulphide is

generated in faeces in this way. Nitrogen trihydride or ammonia can be generated from nitrogen sources such as nitrates and urea by a variety of bacteria such as *Thiobacillus denitrificans*, some acting in aerobic situations; ammonia is generated in this way from urine soilage of nappies and clothing. Some bacteria are capable of converting suitable arsenic sources into the trihydride arsine (28,29,30). In all cases these bacteria also require a suitable nutrient carbon source. Fungi can also generate phosphine, arsine and stibine, *Paecilomyces* and particularly *Scopulariopsis* species being the most likely cause in domestic nursery situations in which the protein-dependent *S. brevicaulis* is likely to be present as an infection of damp wool carpeting or clothing. Infection of plasticised PVC is apparently dependent on perspiration and develops mainly on the area immediately beneath the infant, infection being followed by toxic gas generation if suitable sources of phosphorus, arsenic or antimony are present; stibine formed from antimony trioxide seems to be the most commonly generated gas. The risk of infection on PVC in this way is higher on older materials which are more likely to be affected by established infections, although an infection will only generate significant amounts of gas when it is particularly active, perhaps not until several weeks after the mattress is first used.

Vented mattresses in which the foam at the head end is covered only with a net support massive mixed fungal infections which produce spores which can cause allergic reactions, including severe asthmatic attacks in sensitized infants.

Conclusions

Phosphine, arsine and stibine gas generation through biodeterioration of plasticised PVC is a possible cause of sudden infant death. Infants are more susceptible than adults because of their low body weight which makes it easier to accumulate a lethal dose and particularly if sleeping in the prone position with the face close to the gas accumulating on the mattress. Poisoning is difficult to detect as the phosphorus, arsenic or antimony introduced into the body through phosphine, arsine or stibine poisoning would not increase levels beyond normal ranges.

Investigations should involve incubation of samples of PVC mattress coverings at about 23°C on high protein agar plates, such as 5% malt agar with the addition of 5% soya flour. Positive identification of organisms is not essential, but it is necessary to confirm whether an infection is present which may be generating phosphine, arsine or stibine, most conveniently by incubating the samples on Petri dish plates with silver nitrate and mercuric chloride or bromide test paper strips tucked over the edge of each dish when an active infection has developed; silver nitrate paper changes initially to yellow with phosphine and brown for arsine and stibine, and subsequently to black with all three gases, arsine also causing yellow or red colouration of mercuric chloride or bromide paper.

It should be appreciated that heavy fungal infections on exposed foam in vented mattresses may produce spores that can cause allergic reactions, including severe asthmatic attacks in sensitized infants.

Recommendations

Sudden infant death investigators should recognise phosphine, arsine and stibine poisoning from PVC biodeterioration, and asthmatic reactions to fungal spores, as possible causes of death. PVC mattress coverings should be checked for fungal infections and gas generation, and exposed foam and porous mattress fabrics should be checked for fungal infections.

PVC used in mattress coverings, cot sides and nappy covers should be resistant to fungal and bacterial infections. Resistance can be achieved most reliably and permanently by using resistant plasticisers, rather than by adding biocides. Components containing phosphorus, arsenic and antimony must not be used; the present limits for arsenic and antimony in British Standard BS 1877:Part 10:1982 *Domestic bedding; Part 10, Specification for mattresses for children's cots, perambulators and similar domestic articles* are too high and there are no limits for phosphorus.

Exposed foam mattresses should not be used.

Temporary protection against gas and spore generation can be achieved by wrapping mattresses in clear polythene sheet, secured on the underside using adhesive tape. Carry cot mattresses can be replaced with a folded large sheet.

Acknowledgements

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Evening Standard 6.6

Toxic gas theory on cot deaths

by Alan Massam

THE mystery cot death syndrome may be caused by babies breathing toxic gases generated from PVC mattress coverings, scientists now believe.

A team of researchers have come up with the explanation for sudden infant death syndrome, which kills 2000 babies each year in Britain.

The discovery was announced today by Guernsey-based Penarth Research International. But Britain's leading research body into the problem, The Foundation for the Study of Infant Deaths, condemned it because it was "only an unproven hypothesis".

"We have been inundated with calls from worried parents," a spokeswoman said.

Penarth claim that mattresses tested included those only half covered with PVC. It was found that they were likely to harbour fungi which were capable of giving off gases like arsenic trichloride when they reacted with the plasticisers used to make PVC flexible.

The company believed that the usual circumstances of sudden cot deaths were consistent with poisoning by gas heavier than air accumulating in bedding. Infants usually died face down when they were heavily wrapped against the cold.

Deaths often occurred on mattresses which were not new and in which fungi organisms could have become established.

Mattresses supplied by parents who had recently lost babies were contaminated with the fungus scopulariopsis brevicaulis, particularly in stitching.

The researchers also considered that foam coverings of the top half of mattresses supported fungal infections which could provoke allergic reactions.

The Foundation for the Study of Infant Deaths stressed that epidemiological evidence did not support the Penarth claims because cot deaths occurred long before PVC was used on mattresses.

The Penarth research is supported by Dr Peter Fleming of Bristol Maternity Hospital's Department of Child Health.

D. Telegraph 7.6

Cot deaths theory will be examined

By Our Health Services Staff

Health officials are "taking seriously" a theory that some cot deaths may be caused by PVC in mattresses emitting heavier-than-air noxious gases.

The Department of Health said yesterday that the claim, by a Guernsey research company, would be evaluated scientifically, but a spokesman added: "Current thinking is that cot deaths are due to more than one cause and the possible explanation that has been put forward is just another possibility."

Hope for parents

D Express 7.6

COT deaths bring tragedy to thousands of parents every year. They are all the more distressing for being shrouded in such mystery.

Researchers are not sure whether they stem from one cause or many. All of which does nothing to reassure millions of anxious mothers.

Moreover the mystery helps create suspicion in the minds of outsiders — thus increasing the agony of the grieving parents.

For these reasons the prospect of major breakthrough into the cause of cot death is a matter for rejoicing. If the research team on the Channel Islands really has come up with the answer, they will deserve all our thanks.

D. Express 7.6

Fungus poison gas link to cot deaths

By MARK YOULE

BRITISH scientists claimed yesterday to have tracked down a major cause of cot death, which kills more than 1,000 babies a year.

Researchers in the Channel Islands have discovered a fungus which attacks PVC mattress covers, giving off a cocktail of deadly gases.

The scientists said the amount of poisonous gas has increased because of chemicals added to PVC to comply with recent fire laws.

Laboratory tests on 21 mattresses on which babies died were carried out by Penarth Research International in Guernsey.

Twenty had PVC coverings and each of these had fungal infections which were generating the lethal gases arsine and stibine.

The scientists said more investigation was needed, but research company director Barry Richardson was "absolutely convinced" he has discovered one of the causes of cot death.

Risk

He advised parents to throw away all PVC covered mattresses and replace them with a folded cotton sheet.

Mr. Richardson said: "Very [illegible] amounts of gas are released, but [illegible] where a baby is wrapped [illegible] accumulate to high levels close to the mattress."

"One of the points is babies always seem to lie in face down positions where they are close to the liberated gas."

The news caused panic among parents after Mr. Richardson was interviewed on Radio Four's Today programme.

Hundreds jammed phone lines to the Foundation for the Study of Infant Deaths pleading for help on how to protect their babies. Many callers were in tears.

The Department of Health promised an investigation — and advised parents not to start ripping off PVC mattress covers.

D Express 7.6

Cot deaths: Can this be the answer?

By JANET MENZIES

A COT DEATH is one of the most traumatic tragedies that a young family can suffer. The distress and shock can be so devastating that some parents have been driven to commit suicide as a result.

So the news that scientists have come up with a new and plausible reason for Sudden Infant Death Syndrome (SIDS) is very welcome. The findings show that the PVC-covered mattress on the cot itself could be responsible.

Research by Barry Richardson, of Penarth Research International in Guernsey, revealed yesterday, suggests cot death victims may be being literally gassed by their own cots as fungi, growing on PVC-clad mattresses, give off toxic fumes. The fungi cannot be killed by ordinary disinfectant and are naturally present in the home, especially in wools. It is a terrible irony that many parents have switched to PVC-covered mattresses to beat other possible causes of cot death.

Cot death still remains an enigma. Dr Elizabeth Taylor, senior lecturer in community paediatrics at Sheffield University, is one of the foremost researchers in the field. She says: "Cot death is probably due to a lot of factors which come together and then hit a baby who is already vulnerable. We've got to look at everything."

Cot death was only officially classed as a cause of death in 1971. Before then, the syndrome was not commonly recognised.

Vulnerability

It is now thought that about one in 500 babies die from cot death. Two thirds of these deaths occur in babies of four to 16 weeks old, and slighty more boys are victims than girls. Research has shown that 66 per cent of all cot deaths happen between October and March.

The high winter rates have led researchers to believe that overheating may be a cause, or even suffocation because of overwrapped babies. Children under three months old have difficulty in regulating their temperature-control mechanism. But the researchers came to a halt when looking at older victims.

The new research links the incidence of children lying face downwards and swaddled in covers with their increased vulnerability to fumes accumulating in the PVC bedding.

Further support for the theory appears to come from New Zealand, which has one of the highest cot death rates in the world. The custom there is to place the baby face down on a sheepskin rug covered with blankets — and the micro-organisms which can contaminate PVC and eventually give off toxic gas can also be found in wool.

A common theory is that the baby's oxygen supply is in some way inhibited, either from outside, by gasses perhaps, or from internal problems.

Dr Kevin Forsyth, of the Institute of Child Health in London, has conducted a survey which he believes shows respiratory infection as a factor. His research revealed that many cot death victims had had minor respiratory infections.

Six babies died withing 30 hours in the Home Counties in January this year. Researchers felt that it was caused by a lung problem associated with the after-effects of a minor respiratory tract infection.

Dr Taylor confirms: "Breathing is very important. The mechanism which controls the baby's breathing can be interrupted. Some of my research suggests this is because of respiratory infection. Allergy has also been put forward as a possible reason."

Susceptible

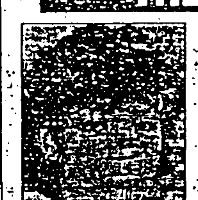
A new book published tomorrow suggests that parents anxious about cot death could take the baby to sleep in their own bed with them. This, an age-old practice in Africa, India and Japan, is linked to the baby following the proximity of the mother's breathing patterns.

In her book Three In A Bed (Bloomsbury, £9.99), writer Deborah Jackson says that in the first year of life, a baby's breathing slows from 87 to around 17 breaths per minute. "And between two and four months; the time when a baby is changing breathing patterns, he is most susceptible to SIDS."

Sleeping next to an adult can stimulate regular breathing in a baby and gas exchange may also be an important factor. "The baby sleeping next to his parents is likely to breathe in carbon dioxide, a chemical stimulant to take the next breath."

Studies quoted in Three In a Bed show that babies sleeping with their mothers display different breathing patterns from children sleeping alone. In fact it would appear that the child's breathing follows the mother's

THE DAY MY BABY DIED



Sue Fellowes ... guilt

LITTLE David Fellowes used to wake so regularly in the morning that he was like an alarm clock for his parents Sue and Graham.

On June 18 last year they overslept. There had been no alarm clock. Sue went into the nursery. Graham was horrified to hear her scream. "I couldn't see David at first, he was under the covers," Sue says. "He was still warm from being under his quilt. I tried giving him mouth to mouth resuscitation and massaging his heart," says Graham. But David was dead.

The post mortem said that there was nothing wrong with David although there was a slight infection of the lung.

"One of the worst things afterwards is the guilt you feel," Sue says. Sue, 21, and Graham, 28, are now expecting a new baby, due in October. They're thrilled but worried, and will have a monitor in the cot which sounds if the baby's breathing is interrupted. The baby will also sleep in their room.

For more information, contact The Cot Death Research Appeal, 8A Alexandra Parade, Weston-super-Mare, tel 0934-413333 or 0934-613333 or on their 24 hour helpline 0836-219010.

ROSEMARY CARPENTER

22480125

COT-DEATH 'CLUE' STARTS

STORM

Mothercare rap boffin

Sun 9·6

By SUN REPORTER

[illegible]P scientist landed in a row last night w[illegible] he claimed to have discovered one of the main causes of cot deaths.

Dr Barry Richardson said mattresses covered by PVC were to blame for many of the cot tragedies which have baffled researchers for years.

But bosses of the giant Mothercare store chain, which stocks a range of PVC-covered foam mattresses, were furious.

A spokesman said: "We don't want parents to worry about something that is not proven."

Mokers

Dr Richardson, 51, head of a research firm in Guernsey, said he found that 20 mattress covers on which babies had died were affected by a fungus giving off toxic gases.

Mothercare WILL discuss his findings with mattress makers.

The Little Ones cot death charity said: "We remain sceptical."

RESEARCHERS UNDER ATTACK FOR CAUSING A PANIC

Fear and anger over cot deaths mattress alert

D Mail 9·6

SCIENTISTS faced an angry backlash yesterday after claiming to have discovered a major cause of the mysterious cot death syndrome.

The Mothercare chain was besieged by calls from parents anxious about the report that PVC-covered mattresses might be to blame.

Doctors were furious that the researchers had jumped the gun and revealed their findings in advance of more stringent tests.

By LIN JENKINS

But last night the director of Penarth Research International was standing by his claim of a possible breakthough in the baffling syndrome which kills one baby in every 500.

Mr Barry Richardson explained how, during research into fabrics for marquees and tents, his team accidentally stumbled across a fungus in PVC-covered mattresses which gives off highly toxic stibine gas when in contact with elements of the PVC.

The mattresses of cot death victims were then studied and it was found that the gas, which is heavier than air, could accumulate in bedding and was more common in older mattresses.

The Penarth report has been submitted to the British Medical Journal where it is being examined by a referee to decide if it is suitable for publication.

But the Foundation for the Study of Infant Death, whose experts have studied the findings, dismissed the 'unproven hypothesis'.

Vice-chairman Lady Limerick said: 'Ideas like this which are unsubstantiated should not be thrown at the public. It is very irresponsible and will cause enormous distress. Cot death has been with us a lot longer than PVC mattresses.'

Medical experts are sceptical about the theory because it would not account for the fact that more boys than girls die in cot deaths, that second-born children are more susceptible and that the risk is greatest between the ages of two and four months.

Mothercare sells three types [of] mattress including one covered w[ith] PVC which has been on sale si[nce] the 1960s.

'We have had lots of calls [from] worried parents, but we are telli[ng] them there is no risk,' a spokesm[an] said.

'Our PVC is supplied by a leadi[ng] European nursery specialist a[nd] our mattresses meet stringe[nt] requirements relating to toxicity.'

Mr Richardson said: 'I faced [a] real dilemma in whether to t[ell] people. As a scientist I would wa[nt] to prove it outright, and I do[n't] want to be accused of scar[e]mongering. But we ha[ve] found something that nee[ds] investigation.

'PVC came in between 1960 and 1970, and it w[as] only around 1970 that une[x]plained infant death reached such a level th[at] the syndrome was reco[g]nised. For the past 20 yea[rs] the figures have remain[ed] one in 500.

'The fact that old[er] mattresses are more suscep[t]ible points to the higher ri[sk] among second-born childre[n]. Even with first-born victi[ms] we found the mattress[es] were passed on from fami[ly] or friends.'

Theories add to grief, say mothers

A MOTHER who lost her five-month-old daughter in a cot death said conflicting theories added to her grief.

Fiona Katzler, from Elstead, Surrey, whose daughter Katie died in January, said: 'I have heard so many explanations, it just becomes ridiculous. Everything from electro-magnetic fields to the baby sleeping the wrong way up and viruses.

'All these different views are very worrying for parents. I believe cot deaths are caused by a combination of factors and if there was one easy solution it would have been found long ago.'

Her daughter slept on a mattress with a partial PVC covering, but she said: 'My two other children slept on the same mattress and came to no harm.'

Clare Loughrey, of Belfast, who lost her baby Nell eight years ago, agreed.

'There are far too many of these reports. I don't think they should be publicised until they are conclusive.

'Newly bereaved parents get terribly upset by hearing so many different views as to why their child died.'

But Alison Keegan, of Owlsmoor, Berkshire, whose six-month-old son Ben died in January, welcomed the investigations.

Ben slept on a foam mattress with PVC covering at the lower end. 'If these findings save lives then it has to be good news for all parents,' she said.

Cot deaths: Can this be the answer?

D Exp. 8·6

[illegible]

THOUSANDS of anxious mothers yesterday faced the dilemma of throwing away their babies' PVC mattresses, which have been [li]nked with cot deaths.

The controversy has brought conflicting [ad]vice. Cot-death specialists are angry at [sc]ientist Barry Richard[so]n for "going pub[li]c" with "shock [re]sults after six [m]onths of research."

By MARK YOULE

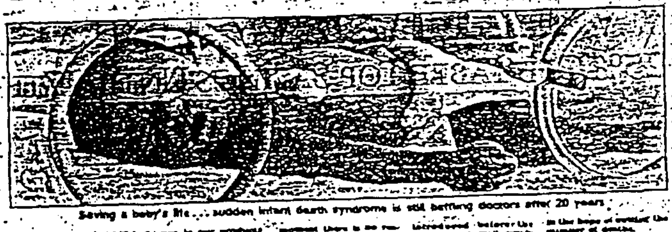
[illegible]

EXPERTS AS SCIENTIST DEFENDS HIS RESEARCH LINKING TOLL WITH 'POISON GAS MATTRESSES'

D Ex 8·6

Cot death dilemma

'Mums are worried about what they're told'



Saving a baby's life ... sudden infant death syndrome is still baffling doctors after 20 years

£½m race to find the cause

EXPRESS REPORTER

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The Times 9·6

PVC fume link to cot deaths dismissed

By Pearce Wright
Science Editor

A theory that cot deaths are linked to poisonous fumes released from PVC plastic cot mattresses has been widely dismissed by experts.

The Department of Health says there is "no adequate validation" for the idea, which has caused alarm among many parents.

The scientific advisory committee to the Foundation for the Study of Infant Deaths, which has spent £4 million in the past 17 years on research, says the hypothesis contradicts the findings of painstaking investigations into factors that may trigger the deaths. The *British Medical Journal* also rejected the theory.

Dr John Grange, consultant bacteriologist at the Brompton Hospital, west London, said in a devastating assessment of the report for *The Times*, which was circulated this week, that more detailed studies were needed to provide a more definitive answer.

The controversy centres on the details of a report by Mr Barry Richardson, director of a scientific consultancy service, which describes the laboratory experiments on which the suggestions about cot deaths are based.

Dr Grange described the experimental methods as "using crude equipment, lacking an obvious control experiment that would reveal flaws and irrelevant as a representation of a cot mattress".

Mr Richardson maintains that a particular type of micro-organism common in homes can react with traces of chemicals used in the manufacture of PVC to generate poisonous fumes.

A key to the process is mattresses must also have a protein content on which the micro-organisms thrive.

The laboratory experiments involved placing strips of PVC and organisms on a rich nutrient mixture of soya flour and malt. Dr Grange said that that bore no relation to mattress conditions and traces of protein it might contain.

Moreover, advanced instruments would be needed to identify the type and amount of gases generated. The report contains no figures of the quantity of gases which came from heating those cultures.

Dr Grange said they could have been generated from the nutrient mixture rather than the PVC. He suggested that Mr Richardson had conducted for the best of motives a completely irrelevant experiment.

22480126

Dilemma of the doctor who triggered mothers' death-panic

As a scientist I had to PROVE cot link . . as a father I had to warn world

BREAKTHROUGH: Barry Richardson has answer to cot death tragedy

Yesterday's TODAY

THE scientist at the centre of the cot death panic is a factually precise, mild-mannered man, not given to dramatic or emotional statements.

But when Barry Richardson discovered that fungus growing on PVC mattresses caused cot deaths, his instincts as a parent took over.

"You can imagine the situation I was in," he says. "I had a terrible dilemma. Should I go on as a scientist and completely prove a link I was certain existed or should I warn people? I still didn't have proof, but as a father and potential grandfather I realised I had to let people know.

Hundreds of panic-stricken mothers jammed a help hotline after Mr Richardson issued his warning.

"I knew there might be a panic but I couldn't have lived with my conscience if I had done nothing. I just hope it saves lives."

A consultant and director of a scientific research company, Mr Richardson first discovered in January that a common household fungus reacted with chemicals in PVC mattresses to produce the gas stibine, 1,000 times more poisonous than carbon

by PENNY WARK

monoxide. "Out of 20 PVC cot death mattresses we examined from Moses baskets, prams, cots, carry cots and of different types of PVC, every one was infected with the fungus in the surface beneath the baby," he says.

As he speaks at his home in Winchester, Mr Richardson cradles two small mattresses. Each was donated by the parents of children who died.

They were just two of the 2,000 British babies who die suddenly and mysteriously in their cots each year, victims of SIDS, sudden infant death syndrome.

Lethal

Mr Richardson, 51, has spent £15,000 researching his theory since the presence of arsenic preservative in plastics first caught his attention last year.

Tests proved the same chemical was not used in cot fabric, but closer examinations revealed stibine was generated when exposed to fungus.

Medical research shows cot death risk is highest for babies lying face down during the winter months or cool summer days.

"This means these babies are well wrapped up. The fungus grows in the warmth and perspiration, the well-wrapped baby breathes in the gas and there is nowhere for it to escape to," says Mr Richardson.

And he maintains his theory is backed up by statistics. "Cot deaths became more noticeable in 1970. They had increased very rapidly during the previous ten years, when PVC began to be used as a mattress covering."

He also points out that SIDS, which affects between two and four babies per thousand, is a purely Western phenomenon.

In Russia and China, where cotton mattresses are used, the syndrome is not recognised.

Although the Foundation for the Study of Infant Deaths has not accepted Mr Richardson's findings, he insists the results speak for themselves.

"I hope PVC mattress manufacturers will change the additives they use. It's as simple as that," he says.

"While we are still conducting tests to prove the link with babies' deaths, we have established that lethal gas is generated by cot mattresses.

"We haven't found an exception yet."

We're no baby killers

THE makers of PVC mattresses denied they produced baby killers.

Relyon, who supply Britain's biggest baby goods chain Mothercare, branded the claim "irresponsible scaremongering".

Quality control manager Richard Marke said: "It is a very dangerous statement to make and can cause unnecessary panic."

He also blasted advice to use folded up sheets or polythene instead as non-PVC mattresses are no longer made.

Relyon's PVC supplier Wardle Storeys ridiculed the findings. "It is a preposterous statement and will get everyone in a panic," said a spokesman. "PVC does not cause cot death.

"We meet standards and do not feel responsible for making an unsafe product, because we don't."

The Foundation for Study into Infant Deaths said the report was "unproven, premature and lacked detailed research".

NEWSCLIP/APCUT

TODAY

Daily

408,078

- 8 JUN 1989

Cot death crusader

RESEARCH scientist Barry Richardson found himself faced with an agonising dilemma over the hugely emotive question of cot deaths.

A tip from a friend sent him on what might have turned into a wild goose chase. PVC in children's bedding could be a source of a gas which just might be the cause of this most tragic and elusive of all fatal conditions in children.

Mr Richardson was not able to establish definitive links between the PVC and cot deaths. But he came across some highly suggestive evidence.

A lethal gas called stibine was produced when PVC came into contact with a common household fungus. Cot deaths had increased rapidly during the ten years PVC coverings have been used in children's mattresses. While in other countries where PVC was not used, cot death was not a recognised killer.

A detective would not be able to get a conviction on evidence like this. But Mr Richardson was not after a conviction. He wanted something much more important. He wanted to save lives.

So, should he have waited until he had made a watertight case? Or should he have made his suspicions public, even at the risk of causing alarm among parents and bringing the manufacturers tumbling down on him with a ton of lawsuits?

He decided to go public. And he was absolutely right to do so. If his analysis is correct and he had waited till he could prove it, more children might have died. If he is wrong, nobody will be the worse off for his mistake.

The principle in such difficult cases must always be to let the public know as much as possible as soon as possible. Then the public can decide.

Secrecy serves nobody. Mr Richardson has earned the public's profound thanks. Other researchers must follow his example. Not only with his kind of painstaking research but with his boldness in telling the world of his suspicions.

2248127

MOTHERS IN COT DEATH PANIC

by ANDREW YOUNG

HUNDREDS of panic-stricken mothers jammed a hotline yesterday after babies' mattresses were blamed for cot deaths.

They pleaded for advice after scientists claimed the bedding's PVC covering could give off a gas 1,000 times as lethal as carbon monoxide.

The danger has soared since new fire-retardant linings were introduced in March, researchers said.

Flooded

Horror spread as radio bulletins announced the latest theory to explain why 2,000 babies every year die in their sleep.

The leading cot death charity, the Foundation for the Study of Infant Deaths, was flooded with calls.

A counsellor said: "Mums were extremely upset to hear the results of the study. Some were so distressed they were crying on the phone."

All infant mattresses are at least partly covered by PVC.

Mr Barry Richardson, who led the research in Guernsey, urged parents to thrown away tiny ones used in carry cots.

"These should be replaced

Turn to Page 2

NEWSCLIP/APCUT

TODAY

Daily 408,078

9 JUN 1989

Mothers panic over mattress link to cot deaths

From Page 1

with a folded sheet," he said. "In the case of a larger drop-sided cot, the mattress should be covered in a polythene sheet and securely taped."

Parents must also ensure that babies' rooms are well ventilated.

But caution was urged by Peter Fleming of Bristol Maternity Hospital, considered one of the country's leading paediatricians.

He said: "The mere possibility that this has occurred, when all the products have been tested for safety, clearly gives us cause for concern.

"The tests were carried out under controlled laboratory conditions and there is no hard evidence that this occurs in the cot.

"We must be cautious. It would be foolish for mothers to throw away PVC mattresses or change the way they attend to their children."

One of the biggest retailers, Mothercare, announced an immediate investigation.

Spokeswoman Jane Bookham said: "The foam mattress has been in our range since the 1960s and we have no record of any cot death attributed to the use of PVC.

"We will investigate the report's conclusions with our manufacturers."

The Foundation for the Study of Infant Deaths admitted it had known about the research for a month.

A spokeswoman said: "The hypothesis that cot deaths might be caused by the release of poisonous gages from PVC cot mattresses has been carefully considered by our Scientific Advisory Committee.

"It must remain as yet a completely unproven hypothesis which does not fit with what is known of sudden infant death syndrome."

There have been many theories to explain cot death since it was first identified more than 30 years ago.

Most of the young victims are under eight months old, with more boys dying than girls. The mortality rate soars during the winter months.

Most previous explanations for the mystery centred on the babies themselves, with such causes as a missing enzyme, a lung malfunction, a deficiency of sleep hormone or abnormal red blood cells.

Babies breathe in killer gas

A SLEEPING baby could be gassed to death by a common household fungus reacting with the PVC on cot bedding.

Scientists at Penarth Research International on Guernsey stumbled on the breakthrough accidentally.

During tests on the deterioration of PVC fabrics they found that a micro-organism *scopulariopis brevicaulis* reacted with chemicals used in production methods.

The bug can give off three lethal gases, all of which are heavier than air. Youngsters lying face down are starved of oxygen as they sleep, the scientists suggest.

Manufacturers use an agent to kill off the fungus during production. But this deteriorates with age.

The potential danger has grown since March when a fire-retardant chemical, antimony trioxide, was introduced into PVC production.

The fungus reacts with the chemical to produce into the highly toxic gas stibine.

Research base director Barry Richardson said: "We found all mattresses new and old harboured the fungus.

"Stibine is 1,000 times more deadly than carbon monoxide and attacks the red blood cells rendering them unable to absorb oxygen.

"The exact cause of death would be anaemia.

"The risk to infants is very high. It is only a hypothesis, but we believe it to be a great step forward in need of greater research." Mattresses with foam pillow headpieces could also cause asthmas attacks, say doctors.

Cot mattress biodeterioration and SIDS

SIR,—In 1988, various fungi were identified in industrial plasticised polyvinyl chloride (PVC) despite the presence of the preservative 10, 10'-oxybisphenoxyarsine (OBPA). Warnings were given that the PVC might also become infected by arsenic-tolerant organisms which might convert OBPA to toxic arsine gas. Mr P. R. Mitchell, a client of our company, inquired whether arsine generation through biodeterioration of such coverings might be a cause of sudden infant death syndrome (SIDS).

50 mattresses used in forty-five SIDS incidents were examined, including 1 cotton covered, 26 PVC covered, 15 PVC covered with exposed foam at one or both ends, and 8 exposed foam; in five incidents 2 mattresses had been used. All mattresses contained *Scopulariopsis brevicaulis*, but only in the area beneath the infant affected by warmth and perspiration. Incubated samples of the infected materials all generated toxic trihydride gases. PVC coverings usually generate stibine (SbH_3) from a fire retardant additive, antimony trioxide, but also phosphine (PH_3) from the phosphate plasticisers that are used when fire resistance is required. Although OBPA is not generally used in cot mattress PVC, tests indicated arsine (AsH_3) generation when it was used; and small amounts of arsine were sometimes identified when it was not, presumably from arsenical impurities in antimony trioxide. Mixtures of gases were often detected. Phosphine generation from PVC was sometimes associated with the development of a pink stain, usually in the shape of the sleeping infant, apparently caused by liberation of phenol compounds from decomposition of phenyl phosphate plasticisers. Only phosphine was generated from infected cotton and foam samples, apparently through biodeterioration of phosphate fire-retardant treatments. Foam samples and the 1 cotton sample were also infected by various other fungi, especially in the area affected by dribble; many of these fungi produce asthmatic reactions.

The generation of highly toxic trihydride gases from cot mattresses is a serious threat to health. The gases are heavier than air, and infants most at risk are those who remain for long periods in their cots, sleep prone in carry-cots, or are heavily wrapped such that they are in long and close breathing contact with accumulations of gas. Heavy wrapping also induces hyperthermia, which prompts an increase in gas generation. Some infants probably survive because the first symptom of poisoning is headache, which results in irritability, dislodgment of bedding, and improved ventilation; infants of low weight cannot dislodge bedding. SIDS occurs mainly in infants up to age 5 months who sleep prone with heavy wrapping and have indications of hyperthermia. SIDS is only reported in western-style communities where foam and PVC covered cot mattresses are used, and has only been recognised since about 1969, following introduction of these materials during the previous 10-15 years.

It has been reported that SIDS is less likely to occur in first born children, perhaps because mattresses are new and do not become infected while the infant is young and most at risk. 5 first children were involved in the 45 incidents investigated. 2 died on previously used mattresses; 1 of these 2 slept on a mattress immediately after use by an older child. 1 infant died on a new foam mattress that was generating phosphine and 2 died on new PVC mattresses which were affected by exceptionally active fungus and were generating phosphine; two other new mattresses were implicated, both of which were similarly affected.

Infection by *S brevicaulis* is not readily visible in PVC. Severe deterioration, usually associated with phosphate plasticisers, results in brittleness and splitting. In the home the fungus is usually found in protein-containing substances such as cheese and meat, and in damp wool and leather; however, its extracellular enzymes can convert a wide range of compounds containing the elements nitrogen, phosphorus, arsenic, and antimony into the trihydrides ammonia, phosphine, arsine, and stibine. The preferential development of this fungus in cot mattresses should be attributed to the presence of nitrogen compounds in the perspiration. The generation of arsine in this way has been recognised for about 100 years since Gosio[1] discovered that death in children and illnesses in adults were sometimes caused by arsine generated by the action of *S brevicaulis* on arsenical wallpaper pigments, although it is now known that arsine could be similarly generated from the horse-hoof size that was commonly used at that time and which contained white arsenic to discourage rodent damage.[2]

Phosphine, arsine, and stibine are exceedingly toxic with threshold limit values of 0·3, 0·5 and 0·1 ppm, respectively, compared with 100 ppm for carbon monoxide. Relatively high ambient levels prevent the detection of phosphorus, arsenic, and antimony residues at necropsy. However, some pathologists have commented that they usually associate SIDS with abnormal lung colour; this could be associated with local erythrocyte haemolysis due to exposure to these trihydride gases.

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1. Gosio B. Azione di alcune muffe sui composti fissi d'arsenico. *Riv d'Igiene e San* 1892; 3: 201–30, 261–73.
2. Thom C, Raper KB. The arsenic fungi of Gosio. *Science* 1932; 76: 548–50.

**A Government inquiry, prompted by these findings, was announced on March 9. The inquiry will be chaired by Prof Paul Turner.

Acute renal failure after colloidal bismuth subcitrate overdose

SIR,—Nephropathy due to acute and chronic use of bismuth salts is well recognised,[1] but oral ingestion of colloidal bismuth is said to be safe since it is less readily absorbed than other salts.[2] We report a case of acute renal failure associated with an overdose of colloidal bismuth subcitrate.

A 76-year-old man who had been taking tripotassium dicitratobismuthate ('De-Nol') for four weeks for haemorrhagic gastritis and duodenitis was admitted having taken an overdose of 6 'Deteclo' (tetracycline hydrochloride 115·4 mg, chlortetracycline hydrochloride 115·4 mg, and demeclocycline hydrochloride 69·2 mg per tablet) and 80 de-nol tablets 4 h earlier. The only other prescribed drug ingested in the month before admission was fluoxetine. His pulse rate was 84 per min and blood pressure was 170/90 mm Hg. He had mild epigastric tenderness and was slightly confused. Plasma creatinine was 145 µmol/l, serum sodium 137 mmol/l, serum potassium 5·4 mmol/l (haemolysed sample), haemoglobin 13·9 g/dl, white cell count $22 \cdot 8 \times 10^9$/l, and platelet count 212×10^9/l. Bismuth was detectable in the blood (1600 µg/l). An abdominal X-ray film showed opacification of the colon by ingested bismuth and a chest X-ray film showed no evidence of free gas under the diaphragm. Since the patient had vomited profusely at home and in the accident and emergency department, gastric lavage was not done. After admission to the ward he was noted to be oliguric and four hours later began passing bloody stools, but no source was seen at sigmoidoscopy. He was started on ranitidine and regular antacid, and magnesium sulphate enemas were prescribed to purge his colon of bismuth. He had a brief episode of hypotension (80/35 mm Hg lasting about 5 min) 12 h after admission, but mean blood pressure was 149/80 mm Hg with a venous central pressure of +10 cm water. He was transfused with two units of blood though his haemoglobin never dropped below 11·3 g/dl. After 48 h of oliguria plasma creatinine was 516 µmol/l and serum potassium was 8·1 mmol/l and he was dialysed for 3 days, during which time he continued to pass bloody stools and needed further transfusion. Acute abdominal pain then developed with absent bowel sounds but he was judged unfit for surgery and died 4 days later. Necropsy revealed a perforated duodenal ulcer and "pale kidneys" which proved to contain bismuth (11 mg/g and 16 mg/g).

This patient had toxic serum bismuth concentrations 4 h after ingestion of 80 de-nol tablets and bismuth proved to have accumulated in his renal tissue. Bismuth nephropathy probably contributed to acute renal failure, although there was the additional insult of gastrointestinal bleeding. We are aware of only one previously recorded case of acute renal failure secondary to colloidal bismuth subcitrate ingestion,[3] in which a 27-year-old man recovered following colonic purging and haemodialysis. Chronic

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