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

2248100

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 FLOWERS: Princess Diana, her arms filled with bouquets from schoolchildren in Hackney 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

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6. 6. 89.

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)

22601112

BFG21131

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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 manufacture 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:

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22480103

BFG21132

BPF

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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 death. They have been sent a report by:

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

who claims that plasticised PVC in cot mattresses gives rise to emissions of Phosphine, Arsine and Stibine. He says that of 9 mattresses examined, 8 contained 2 fungus which develops on plasticised PVC + an adhesive agent.

BFG21134

2480105

Robin Fielder, a toxicologist in BHSS, 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

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

12th June 1989

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Dr P Lewis
Senior Executive, Occupational Health
Product and Regulatory Affairs

Copied to: Mr. H.M. Clayton, Hydro Polymers
Mr. D.M. Middleton, Hydro Polymers

With compliments

BFG21136

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

Date : 8.6.89 Time: 1200 Duration : 3 minutes 10 seconds

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 dot 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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TO: Harold Clayton

FROM: Jeremy Frear

DATE: 9-6-89

NO. OF PAGES (INCLUDING FRONT): 16

IF ANY DIFFICULTIES ARE ENCOUNTERED DURING TRANSMISSION

OUR FAX NUMBER IS: 01-235 8045

MESSAGE: A further copy of the Richardsons report re no information on evidence with more tests used - this to be attached.

2248011M

P.R.I.L.
TECHNICAL SERVICES
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REPORT

COT MATTRESS BIODETERIORATION

AND

SUDDEN INFANT DEATH

Barry A Richardson

P 1

June 1989

COT MATTRESS BIODETERIORATION

AND

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, 60% of the deaths affecting infants between 1 and 3 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 weak-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 retardancy 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 retardancy. 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 phthelate and similar compounds when fire retardancy 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 rubriraticuli*, 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 rubriraticuli* 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 sueceptible, 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

These investigations are an extension of a series of projects on biodeterioration of plasticised PVC. I am particularly grateful to Mr P R Mitchell of Mitchell Marquees who, when warned that arsine generation might occur in stored tentage containing OBPA, prompted these investigations by enquiring whether sudden infant death might be caused in this way. My assistant Mrs S A R Kelly was responsible for the mycological work, and Mr T R G Cox advised on chemical matters. Dr D Allsopp of the CAB International Mycology Institute at Kew supplied the original sample of *Scopulariopsis brevicaulis* which enabled us to establish that phosphine, arsine and stibine could be generated through biodeterioteion of plasticised polyvinyl chloride fabric.

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Evening Standard

Toxic gas theory on cot deaths

GC

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 flexbile.

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 [illegible] released, but [illegible] where [illegible] wrapped [illegible] [illegible] close to the mattress."

"One of the points is babies always seem to die 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?

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

By JANET MENZIES

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

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COT-DEATH 'CLUE' STARTS STORM

Sun 9.6

Mothercare rap boffin

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."

Makers

Dr Richardson, 31, 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 mattress including one covered wi[th] PVC which has been on sale sin[ce] the 1960s.

'We have had lots of calls fro[m] 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 remaine[d] one in 500.

'The fact that old[er] mattresses are more suscep[tible] this points to the higher ri[sk] among second-born childre[n]. Even with first-born victim[s] 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 [illegible], of [illegible], who lost her baby Neil eight years ago, agreed.

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

'Newly bereaved parents get terribly upset at 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?

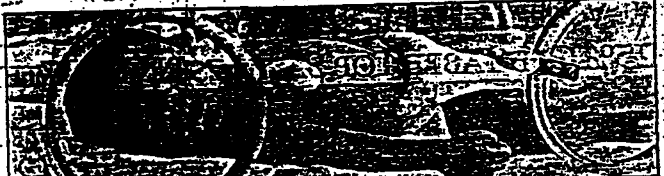
By MARK YOULE

[illegible]

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

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

[illegible]

Premature

Advised

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 Gra[n]ge 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.

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BFG21155

BREAKTHROUGH: Barry Richardson has answer to cot death tragedy

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

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 know 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 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.

Today

MOTHERS IN COT DEATH PANIC

Yesterday's TODAY

They were just two of the 2,500 British babies who die suddenly and mysteriously in their cots each year, victims of SIDS, sudden infant death syndrome.

Lethal

Mr Richardson, 52, has spent £18,000 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 polystyrene 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

406,078

- 8 JUN 1989

Cot death crusader

A DETERMINED scientist Barry Richardson found himself faced with an agonising dilemma over the hugely emotive question of cot death.

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.

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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 throw away tiny ones used to carry cots.

"These should be replaced

Turn to Page 2

NEWSCLIP/APCUT

TODAY

Daily 408,076

9 JUN 1988

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 1980s 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 gases 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 Penguin Research International on Guernsey stumbled on the breakthrough accidentally.

During tests on the deterioration of PVC fabrics they found that a micro-organism aspergillus brakeuils 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 had traces of 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 asthma 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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B. A. RICHARDSON

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 22·8 × 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