

REVIEW

Progress in Reducing Cot Deaths since 1988

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In 1988-89, my research prompted the hypothesis that:

The primary cause of sudden infant death syndrome (SIDS) is anticholinesterase poisoning by gaseous phosphines, arsines and stibines generated by deterioration of cot mattress materials by micro-organisms.

Phosphorus, arsenic and antimony compounds are present in cot mattress materials as plasticizers, fire retardants, preservatives and hygiene agents. The transformation of arsenic compounds in carpets, tapestries and wallpaper into volatile arsines by the fungus *Penicillium brevicaulis*, now known as *Scopulariopsis brevicaulis*, and the toxic action of these arsines, particularly on infants, were first reported by the Italian chemist Gosio in the nineteenth century; indeed, the arsines became known as Gosio's arsenic, and a Royal Commission on arsenical poisoning in the UK at the beginning of the twentieth century warned particularly of the sinister poisoning action of these volatile arsines. As arsenic could be detected in the tissue of victims, particularly in hair and nails, it was believed that these arsines caused normal arsenical poisoning. However, my research indicated that compounds of phosphorus and antimony, the elements adjacent to arsenic in Group V/Vb of the chemical periodic table, could be similarly converted into phosphines and stibines, and that the phosphine, arsine and stibine structures are all anticholinesterase poisons, although some compounds have additional actions, such as the arsenical poisoning by arsines.

The anticholinesterase action of phosphines and arsines has been developed in chemical warfare agents and pesticides by modifying the structures to produce advantageous additional properties, but even the simple trihydride and alkyl phosphines, arsines and stibines are anticholinesterase poisons. In adults they typically interfere with nerve synapses and cause disturbance of the nervous system, but in infants respiration of these gases destroys cholinesterase in the circulating blood, allowing choline to accumulate and slowing the heart rate. In extreme cases this results in cardiac failure and death, and no abnormality or cause can be identified on autopsy. The infant's death is sudden and unexpected and the cause of death is described as sudden infant death syndrome (SIDS).

My hypothesis has been widely recognized as realistic and consistent with all known features of SIDS. Contributory causes have been identified which are easier to understand if it is accepted that I have identified the primary cause. The prone sleeping position results in maximum exposure of an infant to gases generated by microbial infections encouraged by body warmth and perspiration within mattress materials. Overwrapping traps the infant with the generated gases, and overheating accelerates microbial activity and gas generation. Genetic factors, such as a long QT factor, increase susceptibility and cause clusters of SIDS deaths within extended families. Of these contributory factors, the sleeping position is certainly the most important and campaigns recommending avoidance of the prone sleeping

position, such as the Back to Sleep campaign in Britain, have reduced the SIDS rate by about 40% in all geographical regions where they have been promoted. However, in Britain (England and Wales) figures from the Office of National Statistics (formerly the Office of Population Censuses and Surveys) indicate that the SIDS rate has decreased to a much greater extent in a series of distinct steps.

The SIDS rate peaked in Britain at about 2.3 per 1000 births in 1986-88, but following media publicity for my hypothesis and my recommendations that a new cot mattress should be used for every new baby, or old mattresses should be securely wrapped in polyethylene sheet, the rate reduced steadily to about 1.7 per 1000 by 1992. Introduction of the Back to Sleep campaign reduced the rate to about 1.3 per 1000. The Cook Reports on television in 1994 prompted cot mattress manufacturers to eliminate arsenic and antimony compounds from their products and, as these newer mattresses have been more widely used, the rate has decreased steadily to only about 0.4 per 1000 in 2000. It is probable that SIDS could be entirely prevented if the manufacturers also eliminated the phosphorus compounds that they still use as fire retardants and plasticizers. The Department of Health and the Foundation for the Study of Infant Deaths attribute the SIDS reduction to their Back to Sleep campaigns, conveniently ignoring the fact that much of the decrease occurred before their campaigns and the reduction is much greater than the 40% achieved in all other regions where such recommendations have been promoted.

About 20 years ago Japan had infant death statistics similar to those for Britain, except that no SIDS deaths were recorded. However, the large Japanese communities in North America suffered the same SIDS rates as other North American families. Gradually SIDS appeared in Japan and it is now at the same rate as in North America, the explanation being that cotton futons, free from phosphorus, arsenic and antimony compounds, which were used as infant mattresses in Japan have now been replaced by North American-type mattresses containing these dangerous elements. In New Zealand, there were several areas that had the highest SIDS rates in the world, but my recommendations for mattress wrapping to avoid the toxic gases have been adopted and there are now no cot deaths, except where my recommendations are ignored and wrapping is not used.

In Britain, the Department of Health appointed two expert groups to investigate my hypothesis, the first in March 1990 chaired by Professor Paul Turner and the second in 1994 chaired by Lady Limerick. Both groups ignored my recommendations for investigations that I considered most appropriate, apparently because they feared that my hypothesis might be correct and embarrassing to government departments involved in infant mattress controls. Each group concluded that my hypothesis was unproven, even though their investigations disclosed many matters in support of my hypothesis. My main criticism of their investigations was their failure to analyse tissue from SIDS victims, although it had been established through analysis of infant hair in support of the Cook Reports on television that antimony in mattresses can be transferred to infant tissue. In February 2000, UK delegates to the SIDS 2000 conference in New Zealand were accused of murdering infants by insisting that my hypothesis had been disproved. Eventually perhaps common sense will prevail over politics even in the UK: I am not referring to party politics, but the politics which operate in government departments and which are adopted to ensure the satisfactory completion of careers rather than the conscientious achievement of departmental aims, a situation which also affects scientific and medical research.

A SIDS death is very traumatic for the family as the cause of death is not positively identified and the parents wonder whether they caused the death unintentionally. The loss of two or more infants in a family through SIDS is even more horrific, particularly if the mother is then accused of murdering her infants. In most recent trials in the UK, Sir Roy Meadow has given evidence that the chance of a SIDS death is slight and the chance of two SIDS deaths in a family is too remote to be likely, so that it is certain that the infants have been murdered. When challenged, Meadow says that the chance of two SIDS deaths in a family is 1 in 73,000,000. He calculates this as the square of the chance of a single SIDS

death which he considers to be about 1 in 8500, or 0.117 per 1000, although it was actually at least 0.7 per 1000 when he made these calculations. In addition, his calculations do not take family circumstances into account, such as genetic and environmental factors which prompted a first SIDS death and which remain uncorrected so that they then prompt a second SIDS death.

The Department of Health's Confidential Enquiry into Stillbirths and Deaths in Infancy (CESDI) studied 472,823 infant births and deaths in five UK regions. There were 456 sudden unexpected deaths in infancy (SUDI), of which 323 deaths were identified as SIDS, five of these SIDS deaths occurring in families which had previously suffered SIDS deaths, so that the chance of a second SIDS death in a family is actually 5 in 323, or 1 in 64.6. The risk of a second SIDS death in a family is therefore high and it is grossly unfair that Meadow's incorrect evidence is used to wrongly accuse mothers of murder, particularly as Meadow contributed the Preface to the CESDI report on SUDI, so he should be well aware of the true situation. This calculation does not take into account the fact that many mothers do not have a subsequent baby following a SIDS death because the trauma has caused them to separate from their partner or because they are apprehensive that they might lose a second baby. If only 1 in 10 mothers has another baby following a SIDS death, the chance of this further baby dying of SIDS is 1 in 6.46, a very high risk indeed especially if the factors that caused the first SIDS death remain uncorrected.