

# EXHIBIT T

## **Inquiry into the Convictions of Kathleen Megan Folbigg**

1. I prepared a report for Mr Peter Krisenthal, who was a solicitor with Legal Aid NSW. He appeared for Ms Folbigg. My report was originally prepared in 2004 and updated in 2006 (**Attachment A**). I repeat some of what is said in that report.
2. In 2000 while on sabbatical leave from the University of Edinburgh, I was approached by Craig Folbigg via telephone. He called me to inquire about the study on genetic assessment of families in which there had been a sudden infant death. This was in response to an article in a local newspaper about the study I and others were conducting on genetic assessment of families. Mr Folbigg said that his wife was accused of murdering their four children; and, he asked that if they participated in the study, could the results provide an explanation to the deaths. I advised Mr Folbigg that I could not provide them with the results as the ethics committee required all participant information be anonymous. Neither Mr Folbigg nor Mrs Folbigg provided samples for the study.
3. After this telephone conversation with Mr Folbigg, I received a visit from Detective Bernie Ryan who asked for information about sudden infant deaths. He said that there was a family that had four infant deaths and he was investigating the circumstances of the deaths. At the time, I was located in the Newcastle Royal Hospital as a visiting Professor of Immunology and Microbiology. I was based in the clinical diagnostic immunology at the Newcastle Royal Hospital, and Mr Ryan spoke to me in Professor Maree Gleeson's office in the same building. She was then the deputy director of Immunology Services.
4. Both Prof Gleeson and I were at the meeting with Bernie Ryan. He was alone. He informed me he was investigating the circumstances of four infant deaths. I recall being surprised that Mr Ryan wanted information about four infant deaths in one family, because Mr Folbigg had approached me a few days prior about the four infant deaths in his family. I told Mr Ryan that Mr Folbigg had inquired about the study we were conducting but had decided not to take part.
5. I asked Mr Ryan about the results of the autopsies but did not see the reports at this time. The only suggestion I could make was based on work recently published by our research group. I advised him that my research team had screened tissues and body fluids from infants who died of unexplained causes and identified toxins of *Staphylococcus aureus* in over half of the SIDS/SUDI infants tested. More recent findings have indicated that *S. aureus* is one of the major isolates from infants who die suddenly and unexpectedly [Weber *et al.*, 2008; Goldwater, 2009; Kruger *et al.*, 2018].

6. I informed him that we could do enzyme linked immunosorbent assays (ELISA) for the toxins at the University of Edinburgh where the tests had been developed [Zorgani *et al.*, 1999]. I suggested that the costs would be modest; these included a fresh batch of commercially available reagents to exclude any potential contamination from previous studies and the time for my senior research assistant to carry these out. He said it was too expensive as there would need to be someone accompanying the samples and to watch each step of the process. I indicated that there would be no objection to an observer. He did not ask about the cost of the assays.
7. I have since looked at the cost and calculated the amounts (from documents) that are approximately 2008 cost estimates in US dollars. On my calculations it would have cost approximately \$5000 US Dollars (**Attachment B**).
8. Since I had not seen the autopsy files, I could only suggest the toxin testing, which was a recent research advancement; the first article was published in 1999. The study included samples from sudden deaths in Scotland, France, and Australia. Similar results were obtained with samples later sent to Edinburgh from Germany and Hungary [Blackwell *et al.*, 2002].
9. It should be noted that assays for toxins or inflammatory mediators proposed to play a role in some sudden infants are not standard diagnostic tests but used by research groups investigating SIDS/SUDI. These tests would be important in assessing if a child had an inappropriate or overwhelming inflammatory response to a “mild” respiratory infection/sniffle often reported by parents. Two reasons for not including these tests are 1) the additional costs to pathology services, 2) the role of infection and inflammatory responses is not widely held to be important (see letter in which Dr. Cala dismisses the role of infection and inflammation as “junk science”) (**Attachment C**). Perhaps Dr Cala was unaware of papers in this area published prior to 2003 [Morris *et al.*, 1987; Sayers *et al.*, 1995; Bentley *et al.*, 1997; Harrison *et al.*, 1999; Morris, 1999; Zorgani *et al.*, 1999]; however, there is increasing evidence that these factors need to be considered [Morris, 2004; Blackwell *et al.*, 2004; 2005; 2015; Goldwater, 2004; 2009; Goldwater and Bettleheim 2013; Opdal, 2018].
10. I agree to be bound by the UCPR (signed and dated 5 March 2019) (**Attachment D**).
11. I outline a summary of my professional experience and qualifications, and CV (**Attachment E**).

### **What matters could possibly be eliminated or require further investigation in relation to the deaths of the Folbigg children?**

12. **Metabolic disorders:** The genetic analyses and general health of the children do not indicate any evidence for the rare metabolic disorders sometimes associated with

sudden death in infancy; however, further investigations in this area should include genetic assessments by whole genome sequencing.

13. **Statistics relating to multiple infant deaths:** The comments by Dr. Ophoven in her expert certificate statement (6 October 2000) must be reviewed with extreme caution. On page 10 she states categorically, “It is well recognised that the SIDS process is not a hereditary problem and the statistical likelihood that 4 children could die from SIDS is in excess of 1 in a trillion.” (**Attachment F**). The argument is similar to that used by Prof. Sir Roy Meadow in the Sally Clark trial. This has been significantly challenged by Prof. Ray Hill [<http://plus.maths.org/issue21/features/clark/>; Hill, 2004; 2005]. Prior to 2003, there were reports of four or more SIDS deaths in families [Diamond, 1986; Oren *et al.*, 1987].
14. **Sleep or respiratory disorders:** There is little evidence that sleep disorders were involved in the deaths of these infants.
15. **Swollen uvula:** The swollen uvula in Sarah might have resulted from inflammatory responses to a respiratory infection.
16. **Encephalitis in Patrick:** All the laboratory evidence for herpes encephalitis was negative: no virus was isolated; there was no immune response, either IgG or IgM to herpes virus; most importantly, there was no evidence of inflammatory responses in the cerebrospinal fluid (CSF) samples taken. Evidence of inflammatory responses would indicate that an infective process had taken place, but there was no evidence for this. In view of new rapid methods for screening for viral DNA it would be useful to assess any remaining autopsy samples for molecular evidence of other viral pathogens if they have been stored under conditions that preserve nucleic acids. Reassessment of the pathology slides of his CSF would be useful in relation to the paper [Morris *et al.*, 2012] in which lymphocytosis in CSF was associated with infection. At 11:30pm the night before he died, Patrick was vomiting, had a fever and was sweating (Case record of the Regional Medical Genetics Laboratory, A Colley, ref no. 1564) (**Attachment G**).
17. Recent evidence for associations between SIDS and Sudden Unexplained Death in Epilepsy (SUDEP) have been reported. “SUDEP is sudden, unexpected, non-traumatic death of individuals with or without evidence of seizure, in whom postmortem examination does not reveal a structural or toxicological cause for death” [Brownstein *et al.*, 2018]. The hypothesis proposed is that death due to seizures initiates pathogenic signalling between the brain and heart resulting in lethal cardiac arrhythmias [Jehi and Najm, 2008; Ravindran *et al.*, 2016].
18. Genetic analyses have identified markers suggested to be associated with some sudden expected deaths.

19. **SCN1A** mediates the voltage-dependent sodium ion permeability of excitable membranes. Mutations can result in generalized epilepsy with febrile seizures plus (GEFS+) and Dravet syndrome. In one published family history (pedigree), one child had Dravet syndrome and the other had sudden death after a history of febrile seizures. Both children had inherited a pathogenic variant of **SCN1A** from their unaffected father [Halvorsen *et al.*, 2016]. **SCN1B** codes for changes in a sodium channel beta-1 subunit. It is associated with GEFS+, Brugada syndrome and defects in cardiac conduction (Tan *et al.*, 2010). A case report found a **SCN1B** variant (R214Q) in assessment of three individuals, including one female SIDS [Hu *et al.*, 2012].
20. **DEPDE5** is associated with familial epilepsy and pathogenic variants in **SUDEP** (Nascimento *et al.*, 2015). Other variants identified in **SUDEP** include **KCNA1** (Klassen *et al.*, 2014), and **SCN8A** [Poduri, 2015].
21. **Immunodeficiencies:** According to Dr. Charles Hii, there are no appropriate samples to assess this hypothesis (**Attachment H**). There are indications in the children's medical histories to suggest that they had more frequent or more severe bouts of infection. If whole genome sequencing is to be carried out, the cytokine gene polymorphisms need to be reviewed in relation to potential dysregulation of inflammatory responses [Opdal, 2018].

### What factors need further consideration?

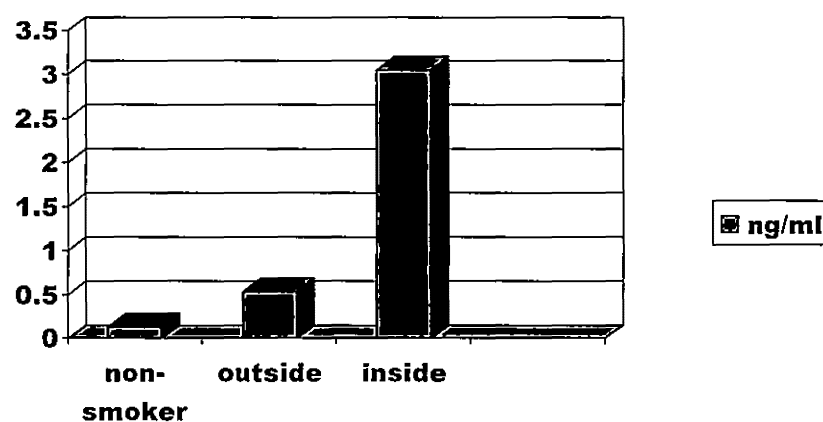
22. **No reports in medical or scientific literature of 4 infant deaths in the same family:** None of the expert witnesses questioned at trial said they had personal experience of four infant deaths in a family, nor could they cite any report in the scientific or medical literature. Each appears to have over looked the original communication by Diamond [1986] of five consecutive siblings whose deaths were diagnosed as SIDS. Oren and colleagues published in 1987, "Familial occurrence of sudden infant death syndrome and apnoea of infancy." In this study, there were two families who had four SIDS and two families who had three SIDS deaths. There was no history to suggest these deaths had been other than natural. The paper was published by the Pediatric Pulmonary Group at the Massachusetts General Hospital in Boston.
23. **SIDS and SUDI:** The emphasis has been on Sudden Infant Death Syndrome. The currently accepted definition of SIDS is restricted in relation to age and the types of investigations carried out to eliminate explainable causes. The strict age range for SIDS (1 month to 1 year) is arbitrary, although some experts are extraordinarily rigid in their use/interpretation of this guideline. The diagnosis of SIDS for the Folbigg children has been criticised on the basis of age: Caleb, 19 days; Patrick, 8 months; Sarah, 10.5 months; Laura, 19 months. To avoid future distraction in relation to age,

the term Sudden Unexpected Death in Infancy (SUDI) or undetermined might be a more useful terms to use.

24. **The interleukin-10 (IL-10) gene hypothesis:** The genetic analyses by Dr. Drucker and Prof. Hutchinson were confined to the IL-10 gene. It is unfortunate that the press labelled their findings as “the cot death gene”. There was good reason for assessment of this factor as IL-10 is an important cytokine which contributes significantly to control of damage elicited by the inflammatory responses to infection and other environmental factors. There is a growing body of evidence that the genetic background of the individual and interactions with environmental factors contribute to potentially lethal responses to infection [Westendorp *et al.*, 1997; Vege and Rognum, 2004; Blackwell *et al.*, 2004; 2005; 2015].
25. There is also evidence that the IL-10 response is significantly decreased by cigarette smoke, a major risk factor for sudden infant death, and that some genetic variants of the IL-10 gene are affected to a greater than others [Moscovis *et al.*, 2004a]. The findings of Dr. Drucker and Prof. Hutchinson [Summers *et al.*, 2000] were confirmed by additional studies with samples from their region of northwest England [Korachi *et al.*, 2004] but not in other studies on IL-10 polymorphisms in populations in other countries [Opdal *et al.*, 2003; Opdal, 2004; Moscovis *et al.*, 2004a]. Their findings cannot, however, be dismissed as other cytokine gene polymorphisms (interleukin-6, TNF- $\alpha$ ) have been shown to differ in SIDS infants from different geographic areas [Moscovis *et al.*, 2006; Moscovis *et al.*, 2015a; Blackwell *et al.*, 2005, 2015; Ferrante *et al.*, 2008; Opdal, 2018].
26. **Inflammation, infection and SUDI:** In relation to SIDS or SUDI, the genetic background of a child needs to be assessed in relation to the infectious agents eliciting an inflammatory response, the developmental state of the child and environmental factors such as cigarette smoke which can affect some of the inflammatory responses [Moscovis *et al.*, 2004a,b; Blackwell *et al.*, 2004; 2005; 2015]. Some sudden deaths in infancy are likely to result from a combination of risk factors or infectious agents and cannot be attributed to a single gene, single infection or other individual factor. In relation to the variety of microorganisms found in Sarah’s lungs, it is important to consider the synergistic effects of mixed infections on induction of inflammatory responses as demonstrated in a model system for SIDS [Sayers *et al.*, 1995] or the combination of infection and exposure to components of cigarette smoke [Blood-Siegfried, 2015].
27. Exposure of infants to cigarette smoke is an important risk factor in all studies of SUDI. It can affect susceptibility to infection and severity of infection in several ways. Smokers are more susceptible to respiratory infections. Their respiratory mucosa are more heavily colonised by bacteria as components in cigarette smoke enhance stickiness of the mucosal surfaces for bacteria. Virus infections can enhance bacterial colonisation by induction of new cell surface components to which bacteria

can bind. One of the inflammatory mediators switched on by virus infection, interferon- $\gamma$  (INF), can enhance other inflammatory responses to bacterial components [Moscovis *et al.*, 2015a].

28. Although many parents who smoke state that they smoke outside the home, there is still some exposure to the smoke which is reflected in cotinine levels in infants; cotinine is a break down product of nicotine. The figure illustrates the levels of serum cotinine detected in children of non-smokers, parents who smoke outside the home and those who smoke inside. This is important because components of cigarette smoke can affect both pro- and anti-inflammatory responses [Moscovis *et al.*, 2004a,b], in particular reducing the anti-inflammatory cytokine IL-10 which helps to moderate the levels of other components of the inflammatory response that can alter physiological mechanisms proposed to explain SUDI. Cytokines produced in the inflammatory responses to infectious agents could have an impact on most of the mechanisms of death proposed: anaphylaxis; poor arousal; hypoxia and apnoea; shock; cardiac arrhythmias; hyperthermia; hypoglycaemia [Blackwell *et al.*, 2015].



29. Assessment of genotypes in the Folbigg children was limited to one polymorphism of the IL-10 gene. A broader assessment of both pro-and anti-inflammatory cytokine gene polymorphisms is needed to provide a more accurate background for further consideration. Some polymorphisms in the IL-6 gene and the TNF- $\alpha$  gene were significantly higher among Australian SIDS infants compared with controls [Moscovis *et al.*, 2006; Moscovis *et al.*, 2015 b; Blackwell *et al.*, 2015].

30. **The role of infectious agents in SIDS and SUDI:** Because of effective childhood immunisation programmes, the majority of children do not experience the classic infectious diseases that accounted for high infant mortality in the past. Epidemiological studies in the United States [Hoffman *et al.*, 1987] and in Britain [Fleming *et al.*, 2001] identified a protective effect for immunisation with the incidence of SIDS being significantly lower among infants who had been immunised at the appropriate time with the diphtheria-tetanus-pertussis (DPT) vaccine. This indicates that infection is involved in some cases of SIDS/SUDI but agents other than the classic bacterial or viral infections of childhood need to be considered.

31. Because invasive bacterial diseases would be explained causes of death, research has concentrated on the more subtle mechanism of deaths mediated by bacterial toxins that can exert their effects in the absence of live bacteria or whole organisms [Morris *et al.*, 1987]. Toxic shock syndrome caused by fever inducing (pyrogenic) toxins of *Staphylococcus aureus* is an example of this phenomenon. The toxic shock syndrome toxin (TSST) and/or other related staphylococcal toxins have been consistently identified in tissues of more than half of SIDS infants from five different countries including Australia [Zorgani *et al.*, 1999; Blackwell *et al.*, 2002].
32. These toxins can be detected by ELISA in body fluids, fresh or frozen tissues or fixed tissues if they are tested with 1-2 years following fixation. These toxins cannot be dismissed as post mortem artefacts; the toxins are produced only between 37-40°C and must have been produced by the bacteria before the body has cooled after death or been refrigerated. The toxins cannot be a contaminant produced during the autopsy procedure, a criticism directed to many microbiological findings in relation to the role of infection in sudden infant death. The toxin has been identified in a child who was about 6 years of age at the time of death whose clinical history was similar to that reported for Sarah Folbigg, *e.g.*, a respiratory infection that resulted in a visit to the GP and antibiotic treatment. Toxin producing *S. aureus* was isolated from his respiratory tract and the TSST identified in his tissues [Bentley *et al.*, 1997]. This was not SIDS but it was a sudden unexpected death in a young child, possibly mediated by the same mechanisms as toxic shock syndrome.
33. Several reviews of microbiological findings in relation to autopsy indicate that if the specimens are taken under appropriate aseptic conditions, the results are of significant value in assessing the cause of death [Morris *et al.*, 2006]. The longer the interval between death and obtaining a sample for microbiological analysis, the lower the chances of isolation of bacteria [Telford *et al.*, 1989; Weber *et al.*, 2010]. In a report by Weber *et al.* [2010], in over 500 autopsies, the percentage of positive cultures decreased from 83% for samples obtained within 24 hours of death to 67.5% when post mortem interval equated to more than 5 days. Significantly fewer polymicrobial cultures were obtained if the samples were collected after a longer interval 61% decreasing to 46% ( $P=0.0003$ ). This evidence does not support the hypothesis of post mortem translocation from mucosal surfaces leading to microbial overgrowth. *Escherichia coli* was found in significantly more samples for children with unexplained diagnosis than those who died of non-infective causes ( $P = 0.003$ ) [Weber *et al.*, 2008].
34. The paper by Weber *et al.* [2008] concluded the high rate of group 2 pathogens, particularly *E. coli* and *S. aureus* in otherwise unexplained cases of SUDI suggests the bacteria could be associated with this condition.



35. There was no microbiology report for Caleb. Microbiology sampling of each of the other three children yielded evidence of coliforms in normally sterile sites.
36. Patrick - Three organisms were found in his blood: *Escherichia coli*; and two species of *Enterococcus*. Both species have surface components, lipopolysaccharide and lipoteichoic acid, that induce powerful inflammatory responses; unfortunately, many of the inflammatory responses induced are short lived, but their role in these deaths is under investigation [Opdal, 2018].
37. The autopsies were carried out on Sarah and Laura at 30 and 12 hours respectively after discovery of the deaths.
38. Sarah - In the lungs, Sarah had profuse numbers of coliforms and alpha haemolytic streptococci and scanty numbers of *S. aureus*. The lungs are normally considered to be sterile sites. In model systems of SIDS (animal or human tissue cultures), mixed infections or virus plus bacteria induced stronger inflammatory responses than those elicited by individual microbes [Sayers *et al.*, 1995; Blood Siegfried, 2015; Moscovis *et al.*, 2015a]. The report recorded **profuse** numbers of coliforms. Coliforms are not normal flora of the respiratory tract. One report indicated a finding of coliforms in an infant conferred an increased relative risk for SIDS of 29 [Gilbert *et al.*, 1992]. The comment about profuse numbers of coliforms is important. For most bacterial infections, the numbers of microbes is important; exposure to low numbers of bacteria might result in clearance and development of immunity to a second exposure. Exposure to larger numbers are more likely to result in infection or damage [Nash *et al.*, 2000].
39. Laura – Laura had profuse numbers of coliforms and in the spleen, profuse numbers of alpha-haemolytic streptococci and moderate numbers of *S. aureus*. Coliform bacteria are not part of the normal flora of the respiratory tract or spleen of healthy adults or infants and are unlikely to have been lung contaminants acquired through resuscitation efforts. It should be noted that in 1992, a paper published by the group of investigators at Bristol University in the UK identified isolation of coliform organisms from the respiratory tract at autopsy had a relative risk for SIDS of 29 [Gilbert *et al.*, 1992].

## Controversies

40. Despite some reluctance to accept that minor infections can trigger SUDI or SIDS, there is a growing body of evidence that infection plays a role in these infant deaths [Goldwater, 2004; 2009; Weber *et al.*, 2008; Morris, 2004; Blackwell *et al.*, 2004; 2005; 2015; Kruger *et al.*, 2018]. In his assessment of the IL-10 gene polymorphism IL-10 –592A in SIDS, Professor Berry (29 April 2003) (**Attachment I**) points out that this genotype confers about a three fold risk of SIDS but that the increased risk associated with smoking is eight fold and that for having the head covered 30 fold. A

study by the group with which Prof. Berry worked found that isolation of coliform bacteria from the respiratory tract similar to those identified in both Sarah and Laura was associated with a 29-fold increase relative risk for SIDS [Gilbert *et al.*, 1992].

41. **Letter from S.E. O'Connor (13 March 2003) (Attachment C)** – Dr. Cala has ignored the evidence implicating infection and inflammation in SUDI published prior to 2003 [Morris *et al.*, 1987; Sayers *et al.*, 1995; Bentley *et al.*, 1997; Harrison *et al.*, 1999; Morris, 1999; Zorgani *et al.*, 1999]. Since 2003, there is increasing evidence that these factors need to be considered. This is not, as Dr. Cala implied, a vague theory by microbiologists or “junk science”. The findings have been reported in well respected, peer-reviewed scientific and medical journals and presented at major national and international meetings [Goldwater, 2004; 2009; Goldwater & Bettelheim, 2013; Blackwell *et al.*, 2004; 2005; 2015; Weber *et al.*, 2008; 2010].
42. Ignoring the microbiological findings was a major oversight in the prosecution of Sally Clark in which the pathologist relied on the hypothesis promoted by Roy Meadow rather than the hard evidence of *S. aureus* isolated from multiple normally sterile sites in the second child. Microbiological analyses found *S. aureus* in a number of sites (internal organs, respiratory tract and cerebral spinal fluid) of the second child. Each of these staphylococcal isolates from the different sites (isolated as a pure culture from sites) was identical to the strain of *S. aureus* isolated from the upper respiratory tract of the child when he was examined on arrival at the accident and emergency department. Each of the isolates from different sites had the identical phage type as determined by the Public Health Laboratory in London. These were facts, not vague hypotheses.

## Summary of findings

43. **Caleb:** As there are no records of samples taken for microbiological analysis or results, it is not possible to make any comment on any potential infectious trigger in his death.
44. The assessment of Prof. Cordner and Prof. Duflou agreed an appropriate diagnosis of SIDS.
45. Further investigation is being carried out by me in relation to eosinophilic exudate noted in the lungs of Caleb Folbigg at autopsy. If there is further information, it will be provided as a supplementary report.
46. **Patrick:** The hypothesis that there was a viral encephalitis is not supported by any microbiological or immunological evidence. The main injuries appear to be neurological. There were, however, three organisms isolated from his blood culture obtained at autopsy - *E.coli*, *Enterococcus faecalis* and *Enterococcus alium*. As the post-mortem examination was carried out two hours after death, it is difficult to

dismiss the findings as contamination as there would have been little time for breakdown of mucosal barriers. If this were contamination from the gut, a larger variety of organisms would be predicted. Evidence of infection prior to death was noted. At 11:30 the night before he died Patrick was vomiting, had a fever and was sweating (Case record of the Regional Medical Genetics Laboratory, A Colley, ref no. 1564) (Attachment J). ↻

47. Both Prof Cordner and Prof Duflou agree that his death was brought on by the consequences of hypoxic-ischaemic encephalopathy resulting from an acute life threatening event (ALTE).
48. **Sarah:** Time of death was estimated at about 1:30 am on 30 August 1993. The rectal temperature of the body at 11 am when it arrived at the mortuary was 25°C. The body would have been refrigerated, minimising bacterial growth until the samples were obtained during the autopsy. The autopsy was carried out at 8 am on 31 August 1993. In the lungs, Sarah had **profuse** numbers of coliforms (see comments p. 9), alpha-haemolytic streptococci and scanty numbers of *S. aureus*. Other findings consistent with SUDI/SIDS include petechial haemorrhage in lungs, pericardium and thymus. There were some signs of inflammation in the lungs including polymorphonuclear (PMN) cells which are found in early stages of infection. There was some aspiration of gastric contents which might be an artefact or possibly vomiting associated with infection or toxin production.
49. Both Prof. Cordner and Prof Duflou agree that Sarah's death can be classified as Sudden Infant Death Syndrome, Category 2.
50. **Laura:** The autopsy on Laura was performed within 12 hours of death. According to the autopsy reports, in the heart there was moderately dense lymphocyte infiltration with degenerate muscles cells. The spleen showed a marked increase in lymphocytes in the red pulp. There was an increase in lymphocytes in the lungs. Laura had profuse numbers of coliforms in the lungs (see comments regarding Prof. Berry's study) and in the spleen, **profuse** numbers of alpha-haemolytic streptococci and moderate numbers of *S. aureus*. The finding of increased numbers of lymphocytes in the lungs and spleen indicate that the child had mounted an inflammatory response and that the bacteriological findings are not *post mortem* contamination as these must occur before death. (See comments above for Sarah in relation to the findings of profuse numbers of bacteria in relation to their potential role in pathogenesis).
51. The reports of reviews of the evidence by Prof. Cordner and Prof. Duflou conclude that the most likely cause of death was myocarditis. Prof. Duflou notes that as in the case of Laura where the inflammation was also predominantly lymphocytic in type, research has found this form of myocarditis tends to have a worse prognosis than other forms [Cooper, 2009].

## Conclusions

52. For the reasons outlined above, my conclusions, particularly for Sarah and Laura, agree with the statement of Prof. Byard in his letter of 18 October to Peter Krisenthal (Attachment JK). "The unusual background of this family with many issues of concern does not negate the fact that potentially significant organic illness was present in these children." The reviews of the evidence and pathology specimens in these cases by Prof. Cordner and Prof. Duflou are in agreement that there are underlying physical factors that could have contributed to these deaths and that homicide is not the default conclusion.

Professor Cecelia Caroline Blackwell



5 March 2019

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## Instructions

This report has been prepared upon instructions of Peter Krisenthal, Solicitor of Legal Aid NSW Criminal Indictable Section, Haymarket, NSW, Sydney in respect to his client, Kathleen Megan Folbigg. I have read the following documents which were supplied by Mr. Krisenthal:

- 1) Medical records of the four children and their mother;
- 2) Transcripts of the relevant pages of the trial report pertaining to expert witnesses and medical evidence;
- 3) Report of Prof. P.J. Berry (November 2001);
- 4) Supplementary report of Prof. P.J. Berry (29 April 2003);
- 5) Reports of Dr. J. Ophoven (6 October 2000 and 1 December 2001);
- 6) Pathology/Autopsy reports of all four children and supplementary reports or specialist tests or examinations;
- 7) Report of Dr. Bridget Wilcken (16 January 2000);
- 8) Statement of Prof. I.V. Hutchinson (17 April 2003);
- 9) Report of Dr. D.B. Drucker (18 February 2003);
- 10) Letters Dr. D.B. Drucker (7 April 2003 and 12 March 2003);
- 11) Statement of Dr. Charles Hii (31 March 2003);
- 12) Letters regarding IgG testing from Department of Public Prosecution (S.E. O'Connor (2 April 2003 and 4 April 2003);
- 13) Letters and attachments regarding IL-10 gene theory of SIDS from Department of Public Prosecution, S.E. O'Connor (13 March 2003 and 23 March 2003);
- 14) Comments by Prof. Berry, Dr. Beal and Prof. Herdson on questions asked of Dr. Cala at trial;
- 15) Supplementary report by Prof. Berry regarding IL-10 gene polymorphism theory (29 April 2003);
- 16) Comment by Dr. Alyson Kakakios on IL-10 gene polymorphism theory and immunodeficiency theory (29 April 2003);
- 17) Neuropathology report on Sarah Folbigg by Dr. R. Pamphlett (pm no. 93/1673);
- 18) Opinion of Prof. R. Byard on deaths of the four children (18 October 2002);
- 19) Expert certificate Dr. B. Wilcken (14 January 2000
- 20) Letters to Dr. B Wilcken from Dr. A. Colley (4 December 1991 and 27 February 1992);
- 21) Letter to Dr. A. Colley from Dr. B. Wilcken (10 December 1991);
- 22) Letter from Gregory Coles to Peter Barnett (15 February 2002);
- 23) Letter to B. Ryan from Dr. A. Cala (19 June 2001);
- 24) Expert certificate Prof. P.B. Herdson (17 February 2002);
- 25) Expert certificate Dr. S.M. Beal (8 December 1999);
- 26) Dr. Beal's report on evidence in the Western Australia case of Scotchmer (21 May 2002);
- 27) Expert certificate Dr. R. Ouvrier (28 October 2002).

## Professional experience and qualifications

I am Professor Cecelia Caroline Blackwell. I am currently con-joint professor in Immunology and Microbiology, School of Health, University of Newcastle, NSW. I also hold professorial appointments in the meningitis reference laboratory which I helped to found at The National School of Public Health, Athens, Greece and in the Institute of Forensic Medicine, Semmelweis University School of Medicine, Budapest, Hungary. Until I took early retirement from the University of Edinburgh, Scotland in December 2001, I was Reader in Medical Microbiology. My previous appointments were in the United States: Assistant Professor, Department of Microbiology, Medical College of Ohio; Postdoctoral Fellow in Infectious Diseases and Associate in Medicine, Beth Israel Hospital and Harvard University School of Medicine.

My qualifications include the following degrees and fellowships: BS in Microbiology, Louisiana State University, USA; PhD in Medical Microbiology, Stanford University School of Medicine, USA; DSc in the Faculty of Medicine, University of Edinburgh; membership and fellowship of the Royal College of Pathologists, UK based on my research in susceptibility to infectious diseases.

My research has been focussed on genetic, developmental and environmental factors that make individuals more susceptible to infectious diseases and conditions in which infection has been implicated such as sudden unexpected death in infancy. I have nearly 300 publications in refereed journals and refereed abstracts and am invited to present my research at national and international meetings on sudden infant death. I have been invited to contribute chapters on the role of infection and inflammation to major books on sudden infant death. I have recently edited a special issue of *FEMS Immunology and Medical Microbiology* (vol. 42 pp. 1-145) on the role of infection and inflammation in sudden infant deaths.

In 2000, I was asked to review the material relevant to the deaths of Christopher and Harry Clark whose mother, Sally Clark, had been convicted of their murder. It was my observation that the microbiology report was missing from Harry Clark's file that led to its recovery and to the evidence that he had suffered from a disseminated infection with *Staphylococcus aureus*. This and other reassessments of the medical evidence in relation to the infection led to the acquittal of Mrs. Clark at the High Court in London in January 2003.

**What factors can be eliminated from further investigation in relation to the deaths of the Folbigg children?**

1. **Metabolic disorders:** The genetic analyses and general health of the children do not indicate any evidence for the rare metabolic disorders sometimes associated with sudden death in infancy. Further investigations in this area would most likely yield negative results.
2. **Statistics relating to multiple infant deaths:** The comments by Dr. Ophoven in her expert certificate statement (6 October 2000) must be reviewed with extreme caution. On page 10 she states categorically, "It is well recognised that the SIDS process is not a hereditary problem and the statistical likelihood that 4 children could die from SIDS is in excess of 1 in a trillion." The argument is similar to that used by Prof. Sir Roy Meadow in the Sally Clark trial. This has been significantly challenged by Prof. Ray Hill in an article in Plus which can be found at the website <http://plus.maths.org/issue21/features/clark/>.
3. **Sleep or respiratory disorders:** There is little evidence that sleep disorders were involved in the deaths of these infants.
4. **Swollen uvula:** There is little evidence that the swollen uvula in Sarah was associated with her death.
5. **Encephalitis in Patrick:** All the laboratory evidence for herpes encephalitis was negative: no virus was isolated; there was no immune response, either IgG or IgM to herpes virus; most importantly, there was no evidence of inflammatory responses in the cerebrospinal fluid (CSF) samples taken. Evidence of inflammatory responses would indicate that an infective process had taken place, but there was no evidence for this.
6. **Immunodeficiencies:** According to Dr. Charles Hii, there are no appropriate samples to assess this hypothesis. There are indications in the children's medical histories to indicate that they had more frequent or more severe bouts of infection.

**What factors need further consideration?**

1. **SIDS and SUDI:** The emphasis has been on Sudden Infant Death Syndrome, or in the case of Patrick a well defined infection. The currently accepted definition of SIDS is restricted in relation to age and the types of investigations carried out to eliminate explainable causes. The strict age range for SIDS (1 month to 1 year) is arbitrary, although some experts are extraordinarily rigid in their use/interpretation of this guideline. The diagnosis of SIDS for the Folbigg children has been criticised on the basis of age: Caleb, 19 days; Patrick, 8 months; Sarah, 10.5 months; Laura, 19 months. To avoid future distraction in relation to age, the term Sudden Unexpected Death in Infancy (SUDI) or undetermined might be a more useful terms to use.
2. **The interleukin-10 (IL-10) gene hypothesis:** The genetic analyses by Dr. Drucker and Prof. Hutchinson were confined to the IL-10 gene. It is unfortunate that the press labelled their findings as "the cot death gene". There was good reason for assessment of this factor as IL-10 is an important cytokine which contributes significantly to control of damage elicited by the

inflammatory responses to infection and other environmental factors. There is a growing body of evidence that the genetic background of the individual and interactions with environmental factors contribute to potentially lethal responses to infection [Westendorp *et al.*, 1997; Vege and Rognum, 2004; Blackwell *et al.*, 2004; 2005]. There is also evidence that the IL-10 response is significantly decreased by cigarette smoke, a major risk factor for sudden infant death, and that some genetic variants of the IL-10 gene are affected to a greater than others [Moscovis *et al.*, 2004a]. The findings of Dr. Drucker and Prof. Hutchinson [Summers *et al.*, 2000] were confirmed by additional studies with samples from their region of northwest England [Korachi *et al.*, 2004] but not in other studies on IL-10 polymorphisms in populations in other countries [Opdal *et al.*, 2003; Opdal, 2004; Moscovis *et al.*, 2004a]. Their findings cannot, however, be dismissed as other cytokine gene polymorphisms (interleukin-1 $\beta$  and interleukin-6) have been shown to differ in SIDS infants from different geographic areas [Moscovis *et al.*, 2004b; Moscovis *et al.*, 2005; Blackwell *et al.*, 2005].

3. **Inflammation, infection and SUDI:** In relation to SIDS or SUDI, the genetic background of a child needs to be assessed in relation to the infectious agents eliciting an inflammatory response, the developmental state of the child and environmental factors such as cigarette smoke which can affect some of the inflammatory responses [Moscovis *et al.*, 2004a,b; Blackwell *et al.*, 2004; 2005]. Some sudden deaths in infancy are likely to result from a combination of risk factors and cannot be attributed to a single gene, single infection or other individual factor. Assessment of genotypes in the Folbigg children was limited to one polymorphism of the IL-10 gene. A broader assessment of both pro-and anti-inflammatory cytokine gene polymorphisms is needed to provide a more accurate background for further consideration. These tests are currently available in the laboratory of Prof. Rodney Scott, University of Newcastle.
4. **The role of infectious agents in SIDS and SUDI:** Because of effective childhood immunisation programmes, the majority of children do not experience the classic infectious disease that accounted for high infant mortality in the past. Epidemiological studies in the United States [Hoffman *et al.*, 1987] and in Britain [Fleming *et al.*, 2001] identified a protective effect for immunisation with the incidence of SIDS being significantly lower among infants who had been immunised at the appropriate time with the diphtheria-tetanus-pertussis (DPT) vaccine. This indicates that infection is involved in some cases of SIDS/SUDI but agents other than the classic bacterial or viral infections of childhood need to be considered. Because invasive bacterial diseases would be explained causes of death, research has concentrated on the more subtle mechanism of deaths mediated by bacterial toxins that can exert their effects in the absence of live bacteria or whole organisms. Toxic shock syndrome caused by fever inducing (pyrogenic) toxins of *Staphylococcus aureus* is an example of this phenomenon. The toxic shock syndrome toxin (TSST) and/or other related staphylococcal toxins have been consistently identified in tissues of more than half of SIDS infants from five different countries including Australia [Zorgani *et al.*, 1999]. These toxins cannot be dismissed as post mortem artefacts; the toxins are produced only between 37-

40°C and must have been produced by the bacteria before the body has cooled after death or been refrigerated. The toxins cannot be a contaminant produced during the autopsy procedure, a criticism directed to many microbiological findings in relation to the role of infection in sudden infant death. The toxin has been identified in a child who was about 6 years of age at the time of death whose clinical history was similar to that reported for Sarah Folbigg, *e.g.*, a respiratory infection that resulted in a visit to the GP and antibiotic treatment. Toxin producing *S. aureus* was isolated from his respiratory tract and the TSST identified in his tissues [Bentley *et al.*, 1997]. This was not SIDS but it was a sudden unexpected death in a young child, possibly mediated by the same mechanisms as toxic shock syndrome.

A recent review of microbiological findings in relation to autopsy indicate that if the specimens are taken under appropriate aseptic conditions, the results are of significant value in assessing the cause of death [Morris *et al.*, 2006]. The longer the interval between death and obtaining a sample for microbiological analysis, the lower the chances of isolation of bacteria [Telford *et al.*, 1989]. The autopsies were carried out on Sarah and Laura at 30 and 12 hours respectively after discovery of the deaths. In the lungs, Sarah had profuse numbers of coliforms and alpha haemolytic streptococci and scanty numbers of *S. aureus*. Laura had profuse numbers of coliforms in the lungs and in the spleen, profuse numbers of alpha haemolytic streptococci and moderate numbers of *S. aureus*. Coliform bacteria are not part of the normal flora of the respiratory tract of healthy adults or infants and are unlikely to have been lung contaminants acquired through resuscitation efforts.

Despite some reluctance to accept that minor infections can trigger SUDI or SIDS, there is a growing body of evidence that infection plays a role in these infant deaths. In his assessment of the IL-10 gene polymorphism IL-10 –592A in SIDS Professor Berry (29 April 2003) points out that this genotype confers about a 3 fold risk of SIDS but that the increased risk associated with smoking is eight fold and that for having the head covered 30 fold. A study by the group with which Prof. Berry worked found that isolation of coliform bacteria from the respiratory tract similar to those identified in both Sarah and Laura was associated with a 25 fold increase in risk of SUDI [Gilbert *et al.*, 1992].

- a. **Letter from S.E. O'Connor (13 March 2003)** – Dr. Cala has ignored the evidence implicating infection and inflammation in SUDI. This is not a vague theory by microbiologists or “junk science”. The findings have been reported in well respected, refereed scientific and medical journals and presented at major national and international meetings [Blackwell *et al.*, 2004; Goldwater, 2004; Blackwell *et al.*, 2005]. This was the crux of the defence case for the successful appeal against the conviction of Sally Clark. Microbiological analyses found *S. aureus* in a number of sites (internal organs, respiratory tract and cerebral spinal fluid) of the second Clark child. Each of these staphylococcal isolates from the different sites (isolated as a pure culture from sites) was identical to the strain of *S. aureus* isolated from the upper respiratory tract of the child when he was examined on

arrival at the accident and emergency department. Each of the isolates from different sites had the identical phage type as determined by the Public Health Laboratory in London.

**Findings for Caleb:** As there are no records of samples taken for microbiological analysis or results, it is not possible to make any comment on any potential infectious trigger in his death.

**Findings for Patrick:** The hypothesis that there was a viral encephalitis is not supported by any microbiological or immunological evidence. The main injuries appear to be neurological.

**Findings for Sarah:** Time of death was estimated at about 1:30 am on 30 August 1993. The rectal temperature of the body at 11 am when it arrived at the mortuary was 25°C. The body would have been refrigerated, minimising bacterial growth until the samples were obtained during the autopsy. The autopsy was carried out at 8 am on 31 August 1993. In the lungs, Sarah had profuse numbers of coliforms (see comments regarding Prof. Berry's study p. 5), alpha haemolytic streptococci and scanty numbers of *S. aureus*. Other findings consistent with SUDI/SIDS include petechial haemorrhage in lungs, pericardium and thymus. There were some signs of inflammation in the lungs including polymorphonuclear (PMN) cells which are found in early stages of infection. There was some aspiration of gastric contents which might be an artefact or possibly vomiting associated with infection or toxin production.

**Findings for Laura:** The autopsy on Laura was performed within 12 hours of death. According to the autopsy reports, in the heart there was moderately dense lymphocyte infiltration with degenerate muscles cells. The spleen showed a marked increase in lymphocytes in the red pulp. There was an increase in lymphocytes in the lungs. Laura had profuse numbers of coliforms in the lungs (see comments regarding Prof. Berry's study p. 5) and in the spleen, profuse numbers of alpha haemolytic streptococci and moderate numbers of *S. aureus*. The finding of increased numbers of lymphocytes in the lungs and spleen indicate that the child had mounted an inflammatory response and that the bacteriological findings are not *post mortem* contamination as these must occur before death.

## Conclusions

For the reasons outlined above, my conclusion, for Sarah and Laura, agrees with that of Prof. Byard in his letter of 18 October to Peter Krisenthal. There is evidence of significant infectious illness and these conditions cannot be ignored as potential causes of death despite the unusual background of the family and the many other issues of concern raised.

Prof. C.C. Blackwell, PhD FRCPath DSc

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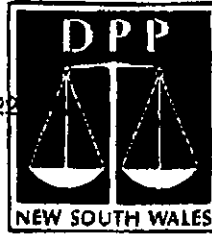
Staphylococcal Antitoxins						
Name	Catalog #		1 mL			
Anti - SEA	AS101		\$ 321			
Anti - SEB	BS202		\$ 321			
Anti - SEC1	CS111		\$ 321			
Anti - SEC2	CS222		\$ 321			
Anti - SEC3	CS333		\$ 321			
Anti - SED	DS303		\$ 321			
Anti - SEE	ES404		\$ 321			
Anti - SEH	SEHS - 1		\$ 321			
Anti - TSST-1	TS606		\$ 321			
Anti - DNase	NS101		\$ 321			
Affinity Purified	IgG		IgG HRPO Conj.		IgG Biotinylated	
Name	Catalog #	1 mg	Catalog #	0.5 mL	Catalog #	0.5 mL
Anti - SEA	LAI101	\$ 321	LAC101	\$336	BAC101	\$336
Anti - SEB	LBI202	\$ 321	LBC202	\$336	BBC202	\$336
Anti - SEC	LCI111	\$ 321	LCC111	\$336	BCC111	\$336
Anti - SED	LDI303	\$ 321	LDC303	\$336	BDC303	\$336
Anti - SEE	LEI404	\$ 321	LEC404	\$336	BEC404	\$336
Anti - SEH	LSEHI-1	\$ 321	LSEHC-1	\$336	BHC-1	\$336
Anti - TSST-1	LTI101	\$ 321	LTC101	\$336	BTC101	\$336
Anti - DNase	LNI101	\$ 321	LNC101	\$336	BNC101	\$336

Normal Rabbit Serum Tested to be Free of Antibodies to SEA-E & TSST-1			
Cat. # NRS-5	5 mL, \$87		Cat. # NRS-10      10 mL, \$168

OUR REFERENCE

YOUR REFERENCE  
Laurel Baglee (02) 9285 8954 (02) 9267 22

DATE Folbigg



**OFFICE OF THE DIRECTOR  
OF PUBLIC PROSECUTIONS**

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13 March 2003

Legal Aid  
FAX: 9219 5906

Attention: Peter Krisenthal

Dear Mr Krisenthal,

**R-v-Kathleen FOLBIGG  
IL-10 GENE THEORY**

We advise the following by way of disclosure.

Professor Hilton advised us today that he has received the e-mail from Dr Drucker which is dated 11 March 2003, which is addressed to Peter Krisenthal and which contains a report of results of genetic testing in respect of Sarah Folbigg. Professor Hilton said that the results do not necessarily mean that Sarah Folbigg died from cot death. He said that, in his opinion, the results make it harder to exclude cot death as the cause of Sarah Folbigg's death.

Dr Cala advised us today that the theory associating the IL-10 gene with SIDS is nothing more than a theory. It is in no way accepted by the forensic community or other people generating the medical literature around the world. He does not give the theory any credence. It is just a theory at this stage.

Dr Cala stated that anyone can go fishing for a gene and suggest a genetic link with SIDS. The issue is whether or not the theory is accepted by the wider medical community. Dr Drucker is a microbiological researcher. This theory has not been accepted by the forensic or SIDS people at all. Dr Cala regards it as "inherently dangerous" when a researcher refers to a gene in this situation as the so-called "cot-death gene". He regards the theory which associates the IL-10 gene as "a vague theory by microbiologists". At this stage, it is no more than "junk science".

Dr Cala said that the medical and forensic issues in this case are, in his opinion, incomparable with Sally Clark's case in the United Kingdom. —?

Yours faithfully,

**SE O'Connor, Solicitor for Public Prosecutions**

per: *Laurel Baglee.*

OUR REFERENCE

Laurel Baglee (02) 9285 8954 (02) 9267 2224 (fax)

YOUR REFERENCE  
Folbigg

DATE

22 April 2003



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Peter Krisenthal  
Legal Aid Commission  
DX 5 SYDNEY

Re: R v Kathleen Megan Folbigg  
Cases No. 2114320  
Listed: 01/04/2003  
Listed for Trial  
At Sydney Supreme Court

I refer to the above matter. Please find by way of service further information received from Professor Berry, Dr Beal and Professor Herdson. Oral responses were sought from each of the expert witnesses to the questions asked of Dr Cala at trial.

Should you require any further information please do not hesitate in contacting me on 9285 8954.

Yours faithfully

S E O'Connor  
Solicitor for Public Prosecutions  
per:

*Laurel Baglee*

Professor HERDSON

Q. You have read the other documents in relation to the deaths of the other three children?

A. Yes

Q. Could we start with the first child to die, Caleb Folbigg?

A. Yes

Q. You were aware from your reading of the documents that at some stage of his life Caleb is alleged to have had a floppy larynx?

A. Yes

Q. Have you, in your own experience, ever had a child who has died of a floppy larynx?

A. No

Q. Have you, in all your reading of the medical literature, ever read of a child who has died of a floppy larynx?

A. *I don't believe in a floppy larynx.*

Q. From your discussions with colleagues, both here in NSW and outside NSW, have you ever heard of a child that has died of a floppy larynx?

A. *I don't know of a floppy larynx.*

Q. If you, yourself, had conducted the post mortem examination of Caleb, without any knowledge of what happened to the other children subsequently, if you had conducted his post mortem, what would your diagnosis have been as the cause of death?

A. *SIDS or might have said undetermined in this case for Caleb. (Has never used diagnosis of undetermined in the context in which we're speaking - so tough on relatives or police. The main point of these children's deaths is that no-one could be certain that any one child died from murder when taken by themselves.)*

Q. And in your view were the findings on Caleb's post mortem examination consistent with him having been deliberately suffocated?

A. *I cannot distinguish between those two - SIDS and deliberate suffocation.*

Q. Are you able to say whether or not Caleb died from a catastrophic asphyxiating event of unknown causes?

A. *Of course he did.*

Q. Thank you. I would now like to proceed to Patrick. If you were examining Patrick's ALTE on its own, without looking at any other children, are you able to say whether or not his ALTE was consistent with him having been deliberately smothered?

A. Yes

Q. Was it?

A. Yes

Q. And are you able to say whether or not Patrick's ALTE was a result of an acute catastrophic asphyxiating event?

A. Yes

Q. Looking now at Patrick's death, again if you are looking only at Patrick on his own without any knowledge of what had happened to the other children, if you had conducted the post mortem examination on Patrick what would your diagnosis have been?

A. SIDS

Q. And are you able to say whether Patrick's death was consistent with having been caused by deliberate smothering?

A. Yes

Q. What are you able to say about whether Patrick's death was a result of an acute catastrophic asphyxiating event of unknown causes?

A. Yes

Q. Moving now to Sarah. If you had conducted the post mortem examination of Sarah, again without any knowledge of what had happened to the other children, what in your view would have been the diagnosis of her cause of death?

A. SIDS

Q. In your view are the findings, as reported by Professor Hilton in relation to the post mortem examination of Sarah, consistent with her having been deliberately smothered?

A. Yes - it can't be excluded.

Q. Doctor, as a pathologist is one of the roles that a pathologist performs during the course of a post mortem examination to exclude death from unnatural causes?

A. Of course it is.

Q. Unnatural causes including both deliberate and accidental trauma?

A. Of course it is.

Q. In your view did Professor Hilton, during his post mortem examination of Sarah, exclude deliberate or accidental trauma?

A. I don't know about that. Would need to go through reports. He made a diagnosis of SIDS - so then he did exclude those things - it's a diagnosis of exclusion. He had every right to make that diagnosis when looking in that case in isolation.

Q. In your view what was the most likely cause of Sarah's death, again looking at her death on its own?

A. By itself, I couldn't get past SIDS. But if I knew two other kids in same family had died, I would have to review that diagnosis.

Q. And looking at her death on its own, in your view was it appropriate to find her cause of death as being from SIDS?

A. Yes

Q. And again, looking at her case on its own, why was it not appropriate to find SIDS as her cause of death?

A. Not applicable.

Q. If you had been conducting Sarah's post mortem examination and you had seen two punctate abrasions under her lower lip, would you have taken photographs of them?

A. *I'm not a great photographer. I would have recorded it, but would not necessarily have taken photographs. If I had noted punctate abrasions it would have immediately worried me.*

Q. *Would you still have persisted with a diagnosis of SIDS.*

A. *If I'd noted that fact, it would have alerted me to wonder - it would have depended on the circumstances - so much depends on what the pathologist is told by the police. Suspected SIDS - subjected to a thorough site inspection.*

Q. And are there facilities in NSW that you are aware of for the taking of photographs during post mortem examinations?

A. Yes

Q. Are those facilities that are only available to police officers or are they also available to the pathologists?

A. Pathologists

Q. Why do you think it was of any importance to take photographs of these punctate abrasions?

A. Not applicable

Q. What do you say about whether or not Sarah, the third child, died of an acute and catastrophic asphyxiating event?

A. *Of course she did.*

Q. In relation to Laura the diagnosis was that her cause of death was undetermined?

A. *By itself, I would have said, until I saw the histology, I would probably have said SIDS, but I would have been concerned that she was 19 months old. Therefore I would at autopsy have said cause undetermined. But when I would have seen the histology, I would have been concerned.*

Q. That it was consistent with smothering?

A. You couldn't exclude it.

Q. Including deliberate smothering?

A. You couldn't exclude it.



Q. And that she probably died from an acute catastrophic asphyxiating event of unknown causes?

A. *Yes of course she did.*

Q. Now, putting those four individual children together is this correct, that they all died from what in your view should have been diagnosed as undetermined causes?

A. *Yes -- but there would come a point if I knew of the previous deaths -- at child 3, definitely 4 -- then murder. Child 1 - Undetermined -- but would call it SIDS. Child 2 -- Undetermined causes -- but would wonder. Child 3 -- Undetermined causes -- but would really wonder. Child 4 -- Murder.*

Q. That they all died in circumstances consistent with deliberate smothering?

A. *Yes -- you couldn't exclude it.*

Q. And that they all possibly died from an acute and catastrophic asphyxiating event of unknown causes?

A. *Yes*

Q. Is there any natural cause of death that could account for all those four deaths and the ALTE?

A. *In my experience, no.*

Dr BEAL

*Area of expertise is epidemiology, which looks at patterns of disease – especially in AIDS – and multiple deaths in a family*

Q. You have read the other documents in relation to the deaths of the other three children?

A. Yes

Q. Could we start with the first child to die, Caleb Folbigg?

A. Yes

Q. You were aware from your reading of the documents that at some stage of his life Caleb is alleged to have had a floppy larynx?

A. Yes

Q. Have you, in your own experience, ever had a child who has died of a floppy larynx?

A. No, none in the 500 cot deaths that I've seen. Floppy larynx could cause symptoms. But never heard of a child dying from it.

Q. Have you, in all your reading of the medical literature, ever read of a child who has died of a floppy larynx?

A. No. If the problem had been serious – he would have had surgery and been sick for a long time. The kid would be very sick before you'd do surgery. The condition is common and mostly not a problem. If severe, the child is in severe stridor for a long time and can be treated surgically.

Q. From your discussions with colleagues, both here in NSW and outside NSW, have you ever heard of a child that has died of a floppy larynx?

A. No.

Q. If you, yourself, had conducted the post mortem examination of Caleb, without any knowledge of what happened to the other children subsequently, if you had conducted his post mortem, what would your diagnosis have been as the cause of death?

A. I wouldn't think floppy larynx was of any great significance. If child was on back, with no covers on its face, you would look at whether there was a minor congenital heart lesion or the child was murdered.

Q. And in your view were the findings on Caleb's post mortem examination consistent with him having been deliberately suffocated?

A. Oh yeah, consistent with that.

Q. Are you able to say whether or not Caleb died from a catastrophic asphyxiating event of unknown causes?

A. Yes. If he didn't have a heart lesion, a catastrophic asphyxiating event is the most likely.

Q. Thank you. I would now like to proceed to Patrick. If you were examining Patrick's ALTE on its own, without looking at any other children, are you able to say whether or not his ALTE was consistent with him having been deliberately smothered?

A. *Certainly very significant. You can't possibly decide whether he had epilepsy at that point. Imposed suffocation was likely to have caused this event.*

Q. Was it?

A. *See above.*

Q. And are you able to say whether or not Patrick's ALTE was a result of an acute catastrophic asphyxiating event?

A. *Yes. But it might have been precipitated by a seizure -- a possibility, but I don't think it's likely.*

Q. Looking now at Patrick's death, again if you are looking only at Patrick on his own without any knowledge of what had happened to the other children, if you had conducted the post mortem examination on Patrick what would your diagnosis have been?

A. *Unknown -- undetermined. (But would say intentional suffocation was likely cause because of the original event)*

Q. And are you able to say whether Patrick's death was consistent with having been caused by deliberate smothering?

A. *Certainly consistent with that.*

Q. What are you able to say about whether Patrick's death was a result of an acute catastrophic asphyxiating event of unknown causes?

A. *Yes, that's exactly what it was.*

Q. Moving now to Sarah. If you had conducted the post mortem examination of Sarah, again without any knowledge of what had happened to the other children, what in your view would have been the diagnosis of her cause of death?

A. *Most likely diagnosis was that this child had been intentionally suffocated as not found prone and her head wasn't covered and no heart lesions found.*

Q. In your view are the findings, as reported by Professor Hilton in relation to the post mortem examination of Sarah, consistent with her having been deliberately smothered?

A. *Yes.*

Q. Doctor, as a pathologist is one of the roles that a pathologist performs during the course of a post mortem examination to exclude death from unnatural causes?

A. *They often can't. There is usually no way of distinguishing between SIDS and intentional suffocation -- you have to go on family history and crime-scene examination to distinguish.*

Q. Unnatural causes including both deliberate and accidental trauma?

A. *Any diagnosis of SIDS has to include the possibility of intentional suffocation.*

Q. In your view did Professor Hilton, during his post mortem examination of Sarah, exclude deliberate or accidental trauma?

A. *No he can't. No pathologist can.*

Q. In your view what was the most likely cause of Sarah's death, again looking at her death on its own?

A. *Intentional suffocation.*

Q. And looking at her death on its own, in your view was it appropriate to find her cause of death as being from SIDS?

A. *Yes - with the understanding that a diagnosis of SIDS includes intentional suffocation. If you prove beyond reasonable doubt that there was intentional suffocation, then you wouldn't call it SIDS. But you make that diagnosis on the history.*

Q. And again, looking at her case on its own, why was it not appropriate to find SIDS as her cause of death?

A. *Not applicable.*

Q. If you had been conducting Sarah's post mortem examination and you had seen two punctate abrasions under her lower lip, would you have taken photographs of them?

A. *I think most people would have photographed them.*

Q. And are there facilities in NSW that you are aware of for the taking of photographs during post mortem examinations?

A. *I presume so.*

Q. Are those facilities that are only available to police officers or are they also available to the pathologists?

A. *Not my area.*

Q. Why do you think it was of any importance to take photographs of these punctate abrasions?

A. *Because nobody can review the findings now.*

Q. What do you say about whether or not Sarah, the third child, died of an acute and catastrophic asphyxiating event?

A. *Yes she did.*

Q. In relation to Laura the diagnosis was that her cause of death was undetermined?

A. *Would think that diagnosis was on history.*

Q. That it was consistent with smothering?

A. *Yes - they all are.*

Q. Including deliberate smothering?

A. *Absolutely.*

Q. And that she probably died from an acute catastrophic asphyxiating event of unknown causes?

A. *Think she probably did. Can't tell you about the myocarditis - I'd depend on the pathologist.*

Q. Now, putting those four individual children together is this correct, that they all died from what in your view should have been diagnosed as undetermined causes?

A. *No. First: SIDS probably, Second: undetermined, Third: suspect intentional suffocation, Fourth: proven.*

Q. That they all died in circumstances consistent with deliberate smothering?

A. *Yes*

Q. And that they all possibly died from an acute and catastrophic asphyxiating event of unknown causes?

A. *Yes*

Q. Is there any natural cause of death that could account for all those four deaths and the ALTE?

A. *Yes - terribly unlikely, metabolic disorders - but here, they would have been excluded. So answer is "no".*

PROFESSOR BERRY

Q. You have read the other documents in relation to the deaths of the other three children?

A. Yes

Q. Could we start with the first child to die, Caleb Folbigg?

A. Yes

Q. You were aware from your reading of the documents that at some stage of his life Caleb is alleged to have had a floppy larynx?

A. Yes

Q. Have you, in your own experience, ever had a child who has died of a floppy larynx?

A. *Floppy larynx is a very common occurrence. Once in my life, came across a case of a child with a floppy larynx who died, but not of the opinion that the cause of death was a floppy larynx.*

Q. Have you, in all your reading of the medical literature, ever read of a child who has died of a floppy larynx?

A. *There is the one case, where a child with a floppy larynx died. It is extremely rare. There would have to be a significant history of a floppy larynx.*

Q. From your discussions with colleagues, both here in NSW and outside NSW, have you ever heard of a child that has died of a floppy larynx?

A. No

Q. If you, yourself, had conducted the post mortem examination of Caleb, without any knowledge of what happened to the other children subsequently, if you had conducted his post mortem, what would your diagnosis have been as the cause of death?

A. *The finding of haemosiderin would cause me not to call it SIDS but to ask further questions. Probably called it Unascertained.*

*haemosiderin  
→ birth?*

Q. And in your view were the findings on Caleb's post mortem examination consistent with him having been deliberately suffocated?

A. Yes

Q. Are you able to say whether or not Caleb died from a catastrophic asphyxiating event of unknown causes?

A. *Only to the extent that we all do, because we all stop breathing.*

Q. Thank you. I would now like to proceed to Patrick. If you were examining Patrick's ALTE on its own, without looking at any other children, are you able to say whether or not his ALTE was consistent with him having been deliberately smothered?

A. *I think that's really a question for a clinician. But the findings in the brain at post mortem were entirely consistent with that having been caused by an hypoxic episode.*

Q. Was it?

A. Yes

Q. And are you able to say whether or not Patrick's ALTE was a result of an acute catastrophic asphyxiating event?

A. *Findings at post mortem were entirely consistent with him having had an hypoxic event in the past and the timing is consistent.*

Q. Looking now at Patrick's death, again if you are looking only at Patrick on his own without any knowledge of what had happened to the other children, if you had conducted the post mortem examination on Patrick what would your diagnosis have been?

A. *In isolation, not ascertained - ascribing it to brain damage following an unexplained collapse, also noting that his mother found him on that occasion and also his earlier event.*

Q. And are you able to say whether Patrick's death was consistent with having been caused by deliberate smothering?

A. *Yes - but children with severe epilepsy do die suddenly.*

Q. What are you able to say about whether Patrick's death was a result of an acute catastrophic asphyxiating event of unknown causes?

A. *Whatever perspective you put on it, his death was probably attributable to the first event, but possibly a later asphyxiating event.*

Q. Moving now to Sarah. If you had conducted the post mortem examination of Sarah, again without any knowledge of what had happened to the other children, what in your view would have been the diagnosis of her cause of death?

A. *Taken in isolation, her death resembles SIDS. But at 10 months of age - need for caution - but Craig's account must be considered. Would probably say SIDS, but with misgivings.*

Q. In your view are the findings, as reported by Professor Hilton in relation to the post mortem examination of Sarah, consistent with her having been deliberately smothered?

A. *Yes. Never heard of a child dying of a swollen uvula.*

Q. Doctor, as a pathologist is one of the roles that a pathologist performs during the course of a post mortem examination to exclude death from unnatural causes?

A. *Yes - so far as one can.*

Q. Unnatural causes including both deliberate and accidental trauma?

A. *That's correct.*

Q. In your view did Professor Hilton, during his post mortem examination of Sarah, exclude deliberate or accidental trauma?

A. *He could not exclude deliberate or accidental suffocation.*

Q. In your view what was the most likely cause of Sarah's death, again looking at her death on its own?

A. *SIDS with misgivings - don't want to put family through police investigation - tend to err on the side of caution.*

Q. And looking at her death on its own, in your view was it appropriate to find her cause of death as being from SIDS?

A. *Yes - although there should have been an X-ray.*

Q. And again, looking at her case on its own, why was it not appropriate to find SIDS as her cause of death?

A. *Not applicable.*

Q. If you had been conducting Sarah's post mortem examination and you had seen two punctate abrasions under her lower lip, would you have taken photographs of them?

A. *Yes - I photograph all my SIDS cases. Finding minor marks on face of baby who has been resuscitated - it is neither here nor there.*

Q. And are there facilities in NSW that you are aware of for the taking of photographs during post mortem examinations?

A. *Not applicable.*

Q. Are those facilities that are only available to police officers or are they also available to the pathologists?

A. *Not applicable.*

Q. Why do you think it was of any importance to take photographs of these punctate abrasions?

A. *See above.*

Q. What do you say about whether or not Sarah, the third child, died of an acute and catastrophic asphyxiating event?

A. *She could have done.*

Q. In relation to Laura the diagnosis was that her cause of death was undetermined?

A. *Taken in isolation, myocarditis.*

Q. That it was consistent with smothering?

A. *Yes - couldn't exclude smothering.*

Q. Including deliberate smothering?

A. *Yes*

Q. And that she probably died from an acute catastrophic asphyxiating event of unknown causes?

A. *Probably. But in isolation - probably myocarditis.*



Q. Now, putting those four individual children together is this correct, that they all died from what in your view should have been diagnosed as undetermined causes?

A. *At child 2 - would have put not ascertained and advised Coroner to get police to carry out investigation.*

Q. That they all died in circumstances consistent with deliberate smothering?

A. *Yes*

Q. And that they all possibly died from an acute and catastrophic asphyxiating event of unknown causes?

A. *Yes*

Q. Is there any natural cause of death that could account for all those four deaths and the ALTE?

A. *Very rare causes run in families. Don't know of diseases where children who are well, suddenly drop dead in 100% of the children.*

*In respect of Laura - myocarditis could have made her easier to smother.*

R-v Kathleen FOLBIGG

Draft Supplementary Report

PJ Berry (Paediatric Pathologist)

I have been asked to provide a supplementary report describing the significance of Dr David Drucker's work on IL-10 in the Sudden Infant Death Syndrome, and his statement that:

"the test on Sarah [Folbigg] found she had two copies of the 'cot death gene' which would obviously increase her risk of SIDS". (Email 12<sup>th</sup> March 2003)

I have also been shown a separate undated communication from Dr Drucker in which he states:

"The samples we received have proved exceptionally difficult... we had to repeat the analyses five times until we had optimised conditions for the sample. Sarah is homozygous for the so-called cot death gene. She is at higher risk than even a baby with one copy would have been."

I have not been shown a formal report from Dr Drucker.

**Background**

In a paper submitted to the journal Human Immunology in May 2000 and published later that year, Dr Drucker and colleagues described an association between a particular naturally occurring variant of a gene and the Sudden Infant Death Syndrome. The gene in question encodes for IL-10, a substance that is important in inflammation and the response to infection. The particular variant (IL-10 S92A) implicated by Dr Drucker and colleagues is believed to result in lower production of IL-10. They hypothesised that this genetic variant might predispose to SIDS "by tardy initiation of protective antibody production [and hence susceptibility to infection] and a lower capacity to inhibit inflammatory cytokine production".

This, and other theories implicating novel mechanisms and infection have rightly attracted considerable interest among SIDS researchers.

This paper also received considerable attention in the media, which prematurely conferred the pejorative label "cot death gene". This label is misleading, not least because many cot death victims do not have this gene variant, and the vast majority of people with it lead healthy lives.

SIDS research is littered with abandoned theories so that most cot death researchers do not accept new findings until another independent group has confirmed them. For example, more than a dozen separate studies involving hundreds of SIDS victims have confirmed the risk of placing babies to sleep in the prone position, so that risk

IL-10 G

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noted

factor is accepted. Many other theories have not been independently confirmed, and so are not generally accepted.

The most common problems with SIDS studies involving statistics are small numbers of cases, case selection and inappropriateness of controls. The IL-10 study, while it involves very sophisticated laboratory methods, is essentially a statistical study comparing the frequency of the IL-10-592A variant in a group of SIDS cases and controls.

#### The size of the study

The study examined just 23 SIDS cases, and so cannot be regarded as anything more than an interesting preliminary study at this stage.

*more in second study w/ smaller results*

#### Case selection

The SIDS cases were included on the basis that they had been "subject to detailed postmortem examination in the North West Regional Perinatal Pathology Department in St Mary's Hospital (Manchester, United Kingdom)." The only way of being certain that a group of cases is unselected is to study all consecutive cases from a defined population over a defined period. If this is not done, then there is a possibility of selection bias, as for example if coroners directed "medical" sounding cases to the perinatal pathology department at St Mary's, and "legal" sounding cases to forensic pathologists elsewhere. Another possible source of bias arises if the diagnosis of SIDS is simply taken from the post-mortem report without further critical review; in this study no exclusions were made, and the authors state "it is possible that babies who died of other causes may have been included." From the paper alone, it is not possible to be confident that the SIDS group is free from selection bias.

#### Controls

The selection of appropriate controls depends on the question being asked in the particular study. In this case the question is; do SIDS babies carry the IL-10-592\*A gene variant more often than babies who do not die? The appropriate control group should therefore be made up of living babies carefully matched for possible confounding factors, and that matching should be tested as part of the analysis of the results of the study.

In this study the authors state that "The control group and their cytokine allele frequencies were those described by Perrey et al consisting of healthy, and sex- and ethnically-matched individuals from the North West Region." The study of Perrey et. al. uses as its control group "principally healthy volunteers or cadaveric renal transplant donors". Curiously, the figures for IL-10\*-A and IL-10\*-C among the 330 controls quoted in the Drucker paper (161 and 499 respectively) are not the same as those in the Perrey paper (151 and 509 respectively).

This control group is clearly prone to serious selection bias, and is inappropriate as a control group for a SIDS study. Indeed, Perrey et. al. warn that "Any study should have a set of matched controls, particularly in studies of other ethnic groups. We have reason to suggest that allele distribution may be quite different in other populations".

### The significance of the study

Assuming that the results are unaffected by selection bias, then the presence of the IL-10-592\*A gene confers about a 3-fold increased risk of SIDS. This is a relatively weak association, comparable to side sleeping, but considerably less than other established risk factors such as prone sleeping where the risk is increased by about 8-fold, head covering where the risk is increased about 30-fold, or sleeping with an adult on a sofa when the risk is increased about 50-fold.

not significant situation + Dr. 25 Fed a / Cal. Fam. 2-10-97

Using the SIDS rate quoted in the paper of 0.62 per 1000 live births, a 3-fold increase in risk would mean that an affected baby would have a risk of dying of SIDS of less than 1 in 500.

Dr Drucker says that Sarah Folbigg "is at higher risk than even a baby with one copy would have been". It may be that, if the association is substantiated, then babies with two copies will be at greater risk than babies with one copy of IL-10-592\*A. In their paper, the authors pooled the results from babies with one and two copies of IL-10-592\*A, so that the figure of 3 is an "average". It is therefore not possible to say from the data presented that the risk for babies with two copies is greater than that for babies with one copy of IL-10-592\*A.

### Is IL-10-592\*A a cause of death in SIDS?

Assuming that the results are unaffected by selection bias, then the authors have demonstrated an association between IL-10-592\*A and SIDS.

They have not presented data to support their theory that this gene variant predisposes to infection or an aberrant inflammatory response. For example, there are no data about antibody levels, nor are the presence of infection or inflammation in SIDS babies with and without this gene variant compared.

IL-10-592\*A therefore remains a possible *association* only, and cannot be invoked as a *cause* of death in SIDS. No pathologist to my knowledge has ever invoked IL-10-592\*A when certifying the cause of death of a baby who has died suddenly and unexpectedly.

### Independent confirmation

Dr Drucker and his colleagues conclude, "If the present study can be confirmed in a larger analysis, then IL-10 genotyping may provide a means to identify children at increased risk of SIDS..."

I am not aware of any other study confirming the association between IL-10-592\*A and SIDS. I have carried out a Medline search with negative results.

— Regional Overlays

I am aware that Dr Drucker has received funding to carry out a further study, but to my knowledge this has not yet been published.

### Conclusion

1. The work of Dr Drucker and his colleagues is of scientific interest

2. The published results of this group concerning IL-10-592\*A and SIDS can only be regarded as a preliminary study
3. There are several possible sources of selection bias in cases and controls which may make the conclusions unreliable
4. The results have not been confirmed in an independent study
5. If these findings are confirmed, then IL-10-592\*A confers a modest increase in the risk of SIDS
6. This is an association only, and not a cause of SIDS
7. The great majority of individuals with IL-10-592\*A do not die as cot deaths
8. The use of the term "cot death gene" in respect of IL-10 is pejorative and frankly misleading

Unsigned  
26/03/03

29/04/03

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## UNIFORM CIVIL PROCEDURE RULES 2005 - REG 31.23

## Code of conduct

## 31.23 Code of conduct

(cf SCR Part 39, rule 2; DCR Part 28A, rule 2; LCR Part 38B, rule 2)

(1) An expert witness must comply with the code of conduct set out in Schedule 7.

(2) As soon as practicable after an expert witness is engaged or appointed:

(a) in the case of an expert witness engaged by one or more parties, the engaging parties, or one of them as they may agree, or

(b) in the case of an expert witness appointed by the court, such of the affected parties as the court may direct,

must provide the expert witness with a copy of the code of conduct.

(3) Unless the court otherwise orders, an expert's report may not be admitted in evidence unless the report contains an acknowledgment by the expert witness by whom it was prepared that he or she has read the code of conduct and agrees to be bound by it.

(4) Unless the court otherwise orders, oral evidence may not be received from an expert witness unless the court is satisfied that the expert witness has acknowledged, whether in an expert's report prepared in relation to the proceedings or otherwise in relation to the proceedings, that he or she has read the code of conduct and agrees to be bound by it.

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### UNIFORM CIVIL PROCEDURE RULES 2005 - REG 31.27

#### Experts' reports

#### 31.27 Experts' reports

(cf SCR Part 36, rule 13C; DCR Part 28, rule 9C; LCR Part 23, rule 1D)

(1) An expert's report must (in the body of the report or in an annexure to it) include the following:

- (a) the expert's qualifications as an expert on the issue the subject of the report,
  - (b) the facts, and assumptions of fact, on which the opinions in the report are based (a letter of instructions may be annexed),
  - (c) the expert's reasons for each opinion expressed,
  - (d) if applicable, that a particular issue falls outside the expert's field of expertise,
  - (e) any literature or other materials utilised in support of the opinions,
  - (f) any examinations, tests or other investigations on which the expert has relied, including details of the qualifications of the person who carried them out,
  - (g) in the case of a report that is lengthy or complex, a brief summary of the report (to be located at the beginning of the report).
- (2) If an expert witness who prepares an expert's report believes that it may be incomplete or inaccurate without some qualification, the qualification must be stated in the report.
- (3) If an expert witness considers that his or her opinion is not a concluded opinion because of insufficient research or insufficient data or for any other reason, this must be stated when the opinion is expressed.
- (4) If an expert witness changes his or her opinion on a material matter after providing an expert's report to the party engaging him or her (or that party's legal representative), the expert witness must forthwith provide the engaging party (or that party's legal representative) with a supplementary report to that effect containing such of the information referred to in subrule (1) as is appropriate.



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### UNIFORM CIVIL PROCEDURE RULES 2005 - SCHEDULE 7

#### SCHEDULE 7 – Expert witness code of conduct

(Rule 31.23)

(cf SCR Schedule K)

##### 1 Application of code

This code of conduct applies to any expert witness engaged or appointed:

- (a) to provide an expert's report for use as evidence in proceedings or proposed proceedings, or
- (b) to give opinion evidence in proceedings or proposed proceedings.

##### 2 General duty to the court

- (1) An expert witness has an overriding duty to assist the court impartially on matters relevant to the expert witness's area of expertise.
- (2) An expert witness's paramount duty is to the court and not to any party to the proceedings (including the person retaining the expert witness).
- (3) An expert witness is not an advocate for a party.

##### 3 Duty to comply with court's directions

An expert witness must abide by any direction of the court.

##### 4 Duty to work co-operatively with other expert witnesses

An expert witness, when complying with any direction of the court to confer with another expert witness or to prepare a parties' expert's report with another expert witness in relation to any issue:

- (a) must exercise his or her independent, professional judgment in relation to that issue, and
- (b) must endeavour to reach agreement with the other expert witness on that issue, and
- (c) must not act on any instruction or request to withhold or avoid agreement with the other expert witness.

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## 5 Experts' reports

(1) An expert's report must (in the body of the report or in an annexure to it) include the following:

- (a) the expert's qualifications as an expert on the issue the subject of the report,
- (b) the facts, and assumptions of fact, on which the opinions in the report are based (a letter of instructions may be annexed),
- (c) the expert's reasons for each opinion expressed,
- (d) if applicable, that a particular issue falls outside the expert's field of expertise,
- (e) any literature or other materials utilised in support of the opinions,
- (f) any examinations, tests or other investigations on which the expert has relied, including details of the qualifications of the person who carried them out,
- (g) in the case of a report that is lengthy or complex, a brief summary of the report (to be located at the beginning of the report).

(2) If an expert witness who prepares an expert's report believes that it may be incomplete or inaccurate without some qualification, the qualification must be stated in the report.

(3) If an expert witness considers that his or her opinion is not a concluded opinion because of insufficient research or insufficient data or for any other reason, this must be stated when the opinion is expressed.

(4) If an expert witness changes his or her opinion on a material matter after providing an expert's report to the party engaging him or her (or that party's legal representative), the expert witness must forthwith provide the engaging party (or that party's legal representative) with a supplementary report to that effect containing such of the information referred to in subclause (1) as is appropriate.

## 6 Experts' conference

(1) Without limiting clause 3, an expert witness must abide by any direction of the court:

- (a) to confer with any other expert witness, or
- (b) to endeavour to reach agreement on any matters in issue, or
- (c) to prepare a joint report, specifying matters agreed and matters not agreed and reasons for any disagreement, or
- (d) to base any joint report on specified facts or assumptions of fact.

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(2) An expert witness must exercise his or her independent, professional judgment in relation to such a conference and joint report, and must not act on any instruction or request to withhold or avoid agreement.

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EXPERT CERTIFICATE

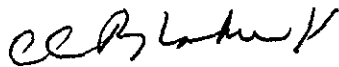
S177 EVIDENCE ACT 1995

The Expert Certificate is given by me pursuant to s177 of the Evidence Act that the defendant proposes to tender this Expert Certificate concerning my attached report dated , which is signed by me as an expert and:

- States my name and address;
- States that I have specialised knowledge based on my training, study or experience as specified in the report attached to this certificate; and,
- Set out an opinion that I hold, and which is wholly or substantially based on that knowledge.

Dated : 5 March 2019

Signed:



Name:

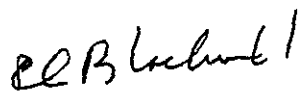
PROF. CECILIA CAROLINE BLACKWELL, PhD DSC FRCR Path FRCN

### CERTIFICATE – EXPERT REPORT

I refer to my report dated \_\_\_\_\_ which is attached to this certificate and certify as follows:

1. I was provided with a copy of the Uniform Civil Procedure Rules 2005 – Expert in Schedule 7 Witness Code of Conduct a copy of which is annexed to my report
2. I have read the Expert Witness Code of Conduct.
3. I agree to be bound by the Expert Witness Code of Conduct.

Dated: 5 March 2019

Sign: 

Name: PROF. CECILIA CAROLINE BLACKWELL, PhD DSc FRCPath FRSN

## Professional experience and qualifications

I am Professor Cecelia Caroline Blackwell. I am currently con-joint professor in Immunology and Microbiology, School of Health, University of Newcastle, NSW. I also hold professorial appointments in the meningitis reference laboratory which I helped to found at The National School of Public Health, Athens, Greece and in the Institute of Forensic Medicine, Semmelweis University School of Medicine, Budapest, Hungary. Until I took early retirement from the University of Edinburgh, Scotland in December 2001, I was Reader in Medical Microbiology. My previous appointments were in the United States: Assistant Professor, Department of Microbiology, Medical College of Ohio; Postdoctoral Fellow in Infectious Diseases and Associate in Medicine, Beth Israel Hospital and Harvard University School of Medicine.

My qualifications include the following degrees and fellowships: BS in Microbiology, Louisiana State University, USA; PhD in Medical Microbiology, Stanford University School of Medicine, USA; DSc in the Faculty of Medicine, University of Edinburgh; membership and fellowship of the Royal College of Pathologists, UK based on my research in susceptibility to infectious diseases. In 2014, I received the Distinguished Researcher Achievement Award from the International Society for Prevention of Infant Deaths for contributions to understanding of the physiology of inflammation and infection in sudden deaths in infancy. In 2016, I was awarded fellowship of the Royal Society of New South Wales (FRSN) for my research in infection and inflammation in sudden death in infancy and susceptibility to infection.

My research has been focussed on genetic, developmental and environmental factors that make individuals more susceptible to infectious diseases and conditions in which infection has been implicated such as sudden unexpected death in infancy. I have nearly 300 publications in refereed journals and refereed abstracts and am invited to present my research at national and international meetings on sudden infant death. I have been invited to contribute chapters on the role of infection and inflammation to major books on sudden infant death. I have edited two special issues of *FEMS Immunology and Medical Microbiology* and one issue for *Frontiers in Immunology* on the role of infection and inflammation in sudden infant deaths.

In 2000, I was asked to review the material relevant to the deaths of Christopher and Harry Clark whose mother, Sally Clark, had been convicted of their murder. It was my observation that the microbiology report was missing from Harry Clark's file that led to its recovery and to the evidence that he had suffered from a disseminated infection with *Staphylococcus aureus*. This and other reassessments of the medical evidence in relation to the infection led to the acquittal of Mrs. Clark at the High Court in London in January 2003.

## ***CURRICULUM VITAE***

**Name:** Professor Cecelia Caroline BLACKWELL

**Date of birth** 1 June 1946

### **Post-school education**

Louisiana State University  
Bachelor of Science.  
Distinguished Scholar, College of Arts and Sciences  
September 1964 - January 1967

Stanford University School of Medicine  
Doctor of Philosophy  
September 1967 - March 1972

The Royal College of Pathologists  
MRCPath (by publications) 1992  
FRCPath 1997

University of Edinburgh  
Doctor of Science 1993

Fellow of the Royal Society of New South Wales  
2015

### **Career: appointments held**

**April 1972 - September 1974**  
Postdoctoral Fellow, Department of Infectious Diseases,  
Beth Israel Hospital, Boston, Mass.  
Associate in Medicine, Harvard Medical School, Boston, Mass.

**September 1974 - June 1976**  
Instructor/Assistant Professor, Medical College of Ohio, Toledo, Ohio.

**October 1976 - March 1980**  
Research Fellow, Department of Bacteriology, University of Edinburgh.

**April 1980 - October 1989**

Lecturer, Department of Bacteriology, University of Edinburgh.

**October 1989 - October 1998**

Senior Lecturer, Department of Medical Microbiology,  
University of Edinburgh.

**May 1996 - present**

Visiting Professor National School of Public Health  
Honorary Consultant to Meningitis Reference Laboratory  
Athens  
Greece

**October 1998-December 2001** Reader, Department of Medical  
Microbiology, University of Edinburgh

**October 1999- present**

Visiting Professor, Faculty of Medicine and Health Sciences,  
University of Newcastle  
Newcastle  
Australia

**December 2000-present**

Co-joint Professor  
Immunology and Microbiology  
Faculty of Medicine and Health Sciences  
University of Newcastle  
Newcastle  
Australia

**Membership of Societies for which academic distinction is the criterion  
for membership:**

Alpha Epsilon Delta Honour Society (USA)  
Mu Sigma Rho Honour Society (USA)  
American Society for Microbiology  
British Society for the Study of Infection  
Royal College of Pathologists (by publications)  
Pathological Society of Great Britain and Ireland

**Prizes awarded and visiting professorships**

In 1993, I received the Schering Plough International Respiratory Infection Task Force Award (\$3,000) for research into paediatric medicine.

In recognition of my contribution to work on meningococcal disease in Greece, I was

appointed visiting professor and honorary consultant to the Meningitis Reference Laboratory at the National School of Public Health, Athens, Greece.

In recognition of my collaborative work on the role of infection in sudden unexpected death in infancy with colleagues at Semmelweis University Medical School in Budapest, I have been appointed to a visiting research professorship.

In 2012, I gave the John Lewis Emory Memorial address sponsored by the Royal College of Pathologists, UK

In 2014, I was given an excellence award by the International Society for the Prevention of Infant Deaths for my work on the role of infection and inflammation in sudden death in infancy.

### **Contributions to profession**

I am invited to referee papers submitted to journals in a variety of disciplines - microbiology, immunology, gastroenterology, infectious diseases, paediatrics. I also referee grant applications dealing with meningitis, *H pylori* infections and Sudden Infant Death Syndrome for major research charities and the Medical Research Council of Canada, the Medical Research Council of The Czech Republic, Foundation for the Study of Infant Deaths (UK) and the Child Health Research Foundation of New Zealand. I have been invited to contribute the chapter on infection in the most recent books published on sudden death in infancy.

### **Papers invited at symposia and conferences**

1986 - IV International Symposium on Pyelonephritis, Goteborg, Sweden.

IV International Symposium on Infections in the Compromised Host, Sweden.

1987 - NATO Advanced Workshop - Complement, Phagocytes and Bacteria, Maratea, Italy.

Medical Research Council Workshop on Bacterial Meningitis, Oxford.

Innate and specific immunity to bacterial meningitis.  
British Society for the Study of Infections, London.

1989 - Arthritis and sexually transmitted diseases. Scottish Rheumatology Society, Pitlochry.

International Symposium on the role of bacterial antigens in organ



specific autoimmunity. Lübeck, FRG.

West Midlands Blood Transfusion Service - Applications of Flow Cytometry in Blood Transfusion, Birmingham.

1990 - V International Symposium on Infections in Immunocompromised Host, Peebles, Scotland.

Symposium on Sudden Infant Death Syndrome, Scottish Cot Death Trust, Glasgow.

Royal College of Physicians (London) and Royal College of Pathologists, Joint Conference on Infectious Diseases.

International Symposium for the Directors of the Institutes Pasteur (world-wide), Institute Francais, Athens, Greece.

1992 - Symposium on Sudden Infant Death Syndrome - Royal College of Pathologists, London.

Meningitis Research Symposium - Dundee University

8<sup>th</sup> International Pathogenic Neisseria Conference - Mexico

Scottish Clinical Microbiologists - Dundee

Symposium on Sudden Infant Death Syndrome, Scottish Cot Death Trust, Royal College of Physicians and Surgeons - Glasgow.

1993 - Joint Meeting, Foundation for the Study of Infant Deaths and Scottish Cot Death Trust - Cambridge.

Sudden Infant Death Syndrome (SIDS) Update. Institute of Medical Laboratory Scientists Cellular Pathology Discussion Group - University of Edinburgh

1993 - Developmental Stages of the Infant in Relation to Sudden Infant Death syndrome (SIDS) - Leicester University

Meningitis Discussion Group - Dundee University

1994 - International Symposium on Sudden Infant Death Syndrome (SIDS) - Stavanger, Norway. [member of expert review panel for case reviews]

9<sup>th</sup> International Pathogenic Neisseria Conference - Winchester UK

Developmental Physiology and SIDS – University of Leicester

1995 - European Monitoring Group for Meningococci - Bad Gleichenberg, Austria

Developmental Physiology and SIDS – University of Leicester  
Scottish Cot Death Trust Research Conference - University of Aberdeen

1996 - Bat Sheeva Seminar, Towards Anti-Adhesin Therapy Zichron Yaakov, Israel

Developmental Physiology and SIDS – University of Leicester

Fourth SIDS International Congress, Washington, D.C.

Workshop on Diagnosis of SIDS, Paris, France

Leopoldina Symposium “Specific Adherence Mechanisms in Microbiology and Immunology, Deutsche Akademie der Naturforscher Cologne, Germany

1997 - International Conference on Sudden Infant Death Syndrome- Dublin, Ireland

European Society for the Study and Prevention of Infant Death - Risk factors to biological causes: the role of infection in SIDS. Barcelona, Spain.

European Monitoring Group on Meningitis - Carriage rates for different populations in Greece and characteristics of meningococcal isolates. Institute Pasteur, Paris, France

Australian SIDS Foundation Annual Meeting– keynote addresses

- 1) Infectious Agents and SIDS
- 2) Why is smoking a risk factor for SIDS?, Melbourne, Australia

1998 - Fifth SIDS International Congress, Rouen, France

10th International Pathogenic Neisseria Conference, Nice, France

Developmental Physiology and SIDS – University of Leicester

1999 - Edinburgh International Science Festival - "Prone to Infection"

European Monitoring Group on Meningitis, Heraklion, Crete  
Risk factors for meningococcal disease and carriage.

European Society for the Study and Prevention of Infant Death -  
Infection, Inflammation, Sleep and SIDS: More pieces to the puzzle,  
Jerusalem, Israel

Developmental Physiology and SIDS – University of Leicester

Scottish Cot Death Trust Conference on Sudden Infant Death Syndrome

2000- 6<sup>th</sup> SIDS International Conference, Auckland, New Zealand

Risk factors and mechanisms of death in Sudden Infant Death Syndrome  
(SIDS Research Institute) Newcastle, Australia

Susceptibility to meningococcal disease - Stobhill Hospital, Glasgow

Developmental Physiology and SIDS – University of Leicester

Susceptibility to infection - Leopoldina Symposium "Implant Materials -  
Infection, Tissue Integration, Advances in New Materials."  
Koln, Germany

12<sup>th</sup> International Neisseria Meeting , Galveston, Texas, USA

Symposium on SIDS – Manchester Medical Society, Manchester

2001- European Workshop on Bacterial Protein Toxins (Etox 10, Bohon,  
Belgium

SIDS Australia National Meeting – Why is smoking a risk factor for SIDS

Developmental Physiology and SIDS – University of Leicester

European Society for the Study and Prevention of Infant Death, Istanbul,  
Turkey

2002 - Second Conference on Meningococcal Disease - Meningitis Association  
of Scotland, Edinburgh

Developmental Physiology and SIDS – University of Leicester

2003 - European Society for the Study and Prevention of Infant Death, Oslo, Norway, "From risk factors to death mechanisms (in SIDS) - the role of microbiological factors" .

Developmental Physiology and SIDS – University of Leicester

2004 - Developmental Physiology and SIDS – University of Leicester

Infection and SIDS, 8<sup>th</sup> SIDS International, Edmonton, Canada

Smoking, infection and SIDS, 8<sup>th</sup> SIDS International, Edmonton, Canada

2005 - Ethnicity, inflammation, stillbirths and SIDS. . 13<sup>th</sup> Annual conference, National SIDS Council of Australia. Adelaide, Australia

Developmental Physiology and SIDS – University of Leicester

Infection ethnic groups and SIDS. First Candle Conference, Washington D.C.

2006- Ethnicity, smoking, inflammation and sudden death in infancy 9<sup>th</sup> SIDS International, Yokohama, Japan

The role of infection in SIDS, Forensic Paediatric Medicine Institute of Forensic Medicine, Oslo, Norway

Developmental Physiology and SIDS – University of Leicester

Ethnicity, infection and inflammation in sudden infant deaths - Manchester Medical Society, Manchester, UK

Ethnicity, smoking, cytokine responses and sudden unexpected death in infancy, Joint meeting Scottish Cot Death Trust and Foundation for the Study of Infant Deaths, Stirling, Scotland

2007- Developmental Physiology and SIDS – University of Leicester

Smoking as a risk factor for SIDS – Foundation for the Study of Infant Deaths, London, UK

Parallels between SIDS and Stillbirths: a role for inflammation. International Stillbirths Association, Birmingham, UK

2008 - Developmental Physiology and SIDS – University of Leicester

Ethnicity and infant deaths– Foundation for the Study of Infant Deaths, London, UK

2009 – Developmental Physiology and SIDS – University of Leicester

Inflammation, infection and SIDS: explaining the risk factors. Kaaren Fitzgerald Memorial Lecture, Ritchie Institute for Child Health, Monash University, Melbourne

Cytokine responses and Sudden Infant Death Syndrome. - Foundation for the Study of Infant Deaths, London, UK

Genetics and inflammatory responses - Foundation for the Study of Infant Deaths, London, UK

Cytokines, Ethnicity and SIDS – Sudden Infant Death and Child Maltreatment - Norwegian Forensic Society

2010 - Infection and SUDI/SIDS: possible links to stillbirths. ISA and ISPD Joint Conference, Sydney Australia 8-10 October

Developmental Physiology and SIDS – University of Leicester

2011 - Developmental Physiology and SIDS – University of Leicester

The weaker sex? Why is there an excess of males among SIDS/SUDI Soria Moria Conference on Paediatric Forensic Medicine, Oslo, Norway

2012 – The John Leiw Emery Memorial Lecture - Developmental Physiology and SIDS – University of Leicester

### **Invited Lectures**

1986 - Blood groups, secretor status and susceptibility to infection. Blood Transfusion Service, Edinburgh.

Blood Groups and Disease. Department of Pathology, University of Edinburgh.

1987 - ABO blood groups, secretor status and susceptibility to infection. Department of Microbiology, Glasgow University

1988 - Secretor status and susceptibility to infectious agents -

Department of Microbiology, University of Dundee.

1989 - Genetic susceptibility to infection - Hellenic Institute Pasteur, Athens.

Genetic Susceptibility to infectious and autoimmune diseases in different ethnic groups. Institute for Human Genetics, Free University, Amsterdam.

1990 - Non-secretion of ABO blood group antigens and susceptibility to bacterial meningitis. Blood Transfusion Service, Edinburgh.

The biological significance of the secretor gene,  
Department of Human Genetics, University of Newcastle upon Tyne.

1991 - Infectious Agents and the Aetiology of Autoimmune Thyroid disease - Endocrine Unit, Department of Medicine, Royal Infirmary of Edinburgh

The role of the secretor gene in HIV infection - Infectious Diseases Unit, City Hospital, Edinburgh

Meningitis research in Scotland - The Meningitis Association (Scotland) Glasgow.

1993 - A new approach to meningococcal vaccines - Pasteur/Merieux, Lyons, France

The role of toxigenic bacteria in Sudden Unexpected Nocturnal Deaths World Health Organisation, Field Epidemiology Training Programme, Bangkok, Thailand

1994 - Toxins and Tragedies - Edinburgh and East of Scotland Society of Anaesthetists

NATO Intensive Course - Pathogenesis and Prevention of Bacterial Diseases, Cantacuzino Institute, Bucharest, Romania.

Infection, inflammation and SIDS - synergy between developmental and environmental factors - University of Southampton

SIDS- Department of Clinical Biochemistry, Royal Infirmary Edinburgh

1995 - Genetic and environmental factors affecting susceptibility to *Helicobacter pylori* Department of Medicine and Therapeutics, University of Glasgow.

- 1997 - Bacterial toxins in Sudden Infant Death Syndrome -PLSD. Medical Countermeasurements, CBD, Ministry of Defence, Porton Down
- Keynote address Australian SIDS Conference, Melbourne, Australia
- Blood group phenotypes, microbial receptors and susceptibility to infection - School of Microbiology, La Trobe University, Australia
- Smoking, viruses and bacterial infections - MacFarlane Burnet Centre for Medical Research, Melbourne, Australia
- 1999 - Genetic, developmental and environmental factors contributing to susceptibility to bacterial infections Department of Microbiology, Tel Aviv University.
- 2000 - Risk factors for SIDS in relation to infection and inflammatory responses Institute for Child Health Research, Perth, Australia
- Infection, inflammation and sleep: key pieces to the SIDS puzzle  
Department of Infectious Disease and Microbiology, Women's and Children's Hospital, Adelaide, Australia
- Susceptibility to meningococcal infection, Hunter Area Pathology Service, Newcastle, Australia
- Ethnic groups and SIDS, Hunter Immunology Unit, Newcastle, Australia
- Royal Medical Society - Susceptibility to meningitis
- Department of Child Health, Royal Hospital for Sick Children, Glasgow –  
Infectious agents and SIDS: explaining the risk factors
- 2001 - Menzies Research Institute, NT, Australia - Ethnic groups and SIDS:  
the  
need for comparative studies
- Princess Margaret Children's Hospital, Perth, WA, Australia - SIDS:  
from epidemiology to mechanisms of death
- Department of Forensic Medicine, Semmelweis University, Budapest  
Sudden Infant Death Syndrome: how the risk factors increase the  
“dangerousness” of infection.
- University of St. Andrews - Biological Society - Sudden Infant Death  
Syndrome: the role of infection and inflammation

2009 - Inflammation, infection and SIDS: explaining the risk factors. Kaaren Fitzgerald Memorial Lecture, Ritchie Institute for Child Health, Monash University, Melbourne.

2012 - Pieces of the puzzle - exploring the risk factors for SIDS. John Lewis Emory Memorial Lecture – Royal College of Pathologists, UK

### **Consultations**

Advisor, The Meningitis Association (Scotland)

Consultant to CIBA Information Service on Bacterial Meningitis

Consultant for Consumer's Association, Which Health.

Consultant to Department of Bacteriology, Hellenic Institute, Pasteur, Athens, Greece (1990-1992)

Consultant to the National Meningococcal Reference Laboratory, National School of Public Health, Athens Greece (1993-present)

Burton Copland Solicitors, Manchester, UK (Legal Aid) 2002 – present

New South Wales Legal Aid (2005 – 2006)

Horowitz and Bilinsky, Sydney, Australia (Legal Aid) (2005)

University of Newcastle Legal Centre (2013- present)



## Research grants

Scottish Home and Health Department - Methods for typing strains of *Neisseria gonorrhoeae* with reference to the development of serological screening tests and epidemiological surveillance (1976-1981) - £46,420 (\$116,050)

Medical Research Council - Host-parasite interactions affecting infection by *Neisseria gonorrhoeae* and *Neisseria meningitidis* (1981-1984) - £30,259 (\$75,647)

Scottish Home and Health Department - Blood group, secretor status and susceptibility to infection (1982-1985) - £37,476 (\$94,117)

Glaxo Group Research Limited - Blood group, secretor state and susceptibility to *Candida albicans* infection (1983-1985) - £18,849.(\$47,118)

Arthritis and Rheumatism Council - Blood group, secretor state and susceptibility to reactive arthritis following infection with Gram-negative bacteria (1985-1988) - £36,515 (\$91,287)

The Wellcome Trust - Susceptibility to infection by *Candida albicans*: genetic and environmental influences (1985-1987) - £35,526. (\$91,315)

Sir Stanley and Lady Davidson Research Fund - Characterization of *Escherichia coli* isolated from urinary tract infections (1986-1987) - £7,538 (\$18,845)

The Meningitis Trust - Secretor state and carriage of *Neisseria meningitidis* (1986-87) - £27,284. (\$68,210)

Scottish Hospital Endowments Research Trust - Non-secretion of blood group antigens and susceptibility to bacterial meningitis (1987-89) - £13,840 (\$34,600)

Tenovus-Scotland - Non-secretion of blood group antigens and susceptibility to bacterial meningitis (1987-1990) - £28,000 (\$70,000)

The Meningitis Trust - supplemental grant for Tenovus award (1987-1990) - £3,000.

The Medical Research Council - Secretor state and susceptibility to bacterial meningitis (1987-1989) - £13,284 (\$33,210)

Sir Samuel Scott of Yews Trust - Genitourinary tract infections during pregnancy (1987-1988) - £3,000 (\$7,300)

The Cunningham Trust - Clinical and microbial features of secretory immune responses in HIV positive homosexual males and in drug abusers in Lothian Region (1987-1988) - £15,326 (\$38,315)

The Hoyes Bequest - Susceptibility to fungal infection (1988) - £1,500 (\$3,750).

The Wellcome Trust - Fungal infections in immunocompromised patients (1988-1990) - £53,416 (\$133,540)

The Meningitis Trust - Secretor status and susceptibility to bacterial meningitis (1989) £3,448 (\$8,620)

The Department of Medicine, The Royal Infirmary, Edinburgh, Endocrinology projects (1989-1990) - £2,000 (\$5,000)

Lothian Health Board - The role of secretor status in heterosexual transmission of HIV (1990) - £2,000 (\$5,000)

Lanarkshire Health Board - Serological responses to carriage of *Neisseria meningitidis* (1990) - £2,000 (\$5,000)

Sir Samuel Scott of Yews Trust - The role of bacteria in spondylarthropathies (1991) - £5,800 (\$14,500)

Ministry of Health, Welfare and Social Security, Athens - Genetic and environmental factors associated with *Neisseria meningitidis* (1991) - DR 2,500,000.

The Meningitis Trust - The role of viruses as predisposing factors for meningococcal carriage and disease (1991-1994) - £47,067 (\$117,668)

The Meningitis Association (Scotland) - Genetic and environmental factors associated with susceptibility to bacterial meningitis (1991) - £10,000 (\$25,000)

European Economic Community - Blood group and secretor status in heterosexual transmission of HIV in Greece (1992) - £2,000 (\$5,000)

The Scottish Cot Death Trust - Factors affecting colonization by toxigenic bacteria with reference to sudden infant death syndrome (SIDS) (1992-1995) - £85,655 (£214,137)

Sir Samuel Scott of Yews - Support for research on infections in infancy (1992) - £5,000 (\$12,500)

The Scottish Cot Death Trust - Susceptibility to infection in relation to sudden infant death syndrome (SIDS) (1993-1996) - £89,931 (\$244,828)

The Scottish Cot Death Trust - Toxigenic bacteria and sudden infant death syndrome. (1993-1996)- £ 68,532 (\$171,330)

Meningitis Association of Scotland - Susceptibility to meningococcal disease (1993) - £4284 (\$10,705)

Overseas Development Administration - The role of toxigenic bacteria in Sudden Unexpected Nocturnal Deaths (SUND) (1993) - £3,500 (\$8,750)

Babes in Arms - Detection of Lewis antigens during infant development (1994) - £15,000 (\$37,500)

Meningitis Association of Scotland - Meningococcal disease in Scotland (1994-1995)- £4415 (\$11,036)

NATO- Pathogenesis and prevention of infectious diseases (1994) - £5,800 (\$14,500)

Meningitis Research Foundation- Small equipment grant (1994)- £300. (\$750)

Babes in Arms - major equipment grant (1994) - £40,000 (\$100,000)

Meningitis Research Foundation - Isolation and characterization of surface components of *Neisseria meningitidis* that enhance bacterial binding to virus infected cells (1995)- £15,000 (\$37,500)

Foundation for the Study of Infant Deaths - The role of glycoprotein G of respiratory syncytial virus in binding of toxigenic bacteria to epithelial cells (1994-1995) .£4,100 (\$10,250)

Scottish Cot Death Trust - Investigation of the change in infant immunization schedules and the decline of sudden infant death syndrome (1995-1996) - £29, 660 (\$74,150)

Meningitis Association of Scotland - supplement to studies on meningococcal disease in Scotland (1995)- £3050 (\$7,626)

Meningitis Association of Scotland and Trustee Savings Bank -- equipment grant (1995) - £3,500 (\$8,750)

Chest Heart and Stroke Scotland -- Smoking and viral infection in exacerbations of chronic bronchitis (1996-1997) £58,701.(\$146,753)

Chest Heart and Stroke Scotland -- Incidence of antibodies to *Helicobacter pylori* among patients who died of coronary heart disease or thrombosis (with Prof. A. Busuttil) (1996) £2,900 (\$7,250)

Sir Samuel Scott of Yews -- Preliminary investigations on the role of infectious agents and inflammatory responses in cot deaths and apparent life-threatening events (ALTE)- £5,000 (\$12,500)

Meningitis Association of Scotland – supplement to studies on meningococcal disease in

Scotland (1996) -- £7,600 (\$19,000)

Chest Heart and Stroke Scotland -- Chronic infection and coronary heart disease (with Prof. A. Busuttil and Dr. S. Sutherland) (1997-1999) £58,492 (\$146,230)

Babes in Arms -- The protective effect of breast feeding in relation to Sudden Infant Death Syndrome (1996-1999) £40,700 (\$101,750)

Sir Jules Thorn Trust -- The potential use of receptor analogues in prevention of superficial fungal infections £55,974 (\$139,935)

Chief Scientists Office-- Epidemiological and laboratory studies of *Escherichia coli* O157 (1997-1999) £92,177 (\$230,443)

Britannic Assurance -- Studies on nasopharyngeal flora of Asian infants in relation to maternal smoking £500 (\$1,250)

Chest Heart and Stroke Scotland -- Detection of antibodies to *Helicobacter pylori* in patients following their first heart attack. £3,000 (\$7,500)

Meningitis Association of Scotland and Trustee Savings Bank Foundation -- (1997-2000) £40,500 (\$101,250)

Scottish Cot Death Trust -- The protective effect of immunisation in relation to SIDS (1998-2000) (£71,441) (\$178,603)

Scottish Cot Death Trust -- Genetic control of inflammatory responses in relation to Sudden Infant Death Syndrome (SIDS) (1999-2001) (£74,183) (\$184,458)

Sir Samuel Scott of Yew Trust - Studies on susceptibility to *Escherichia coli* O157 (1999) £5,000 (\$12,500)

Wellcome Trust- Effects of antibiotic selection on virulence properties of *Moraxella catarrhalis* (with Prof. S.G.B. Amyes) (1999-2001) £95,509 (\$238,772)

Meningitis Association of Scotland - equipment grant for studies on susceptibility to meningococcal disease £22,000 (\$55,000)

Scottish Cot Death Trust and Babes in Arms - The effects of developmental stage and environmental risk factors for Sudden Infant Death Syndrome on the nasopharyngeal flora of infants from different ethnic groups £ 30,000 (\$75,000)

Babes in Arms - Cytokine polymorphisms in Sudden Infant Death Syndrome £12,000 (\$30,000)

John Hunter Childrens Hospital Research Fund: The effects of cigarette smoke on immune and inflammatory responses in Aboriginal and non-Aboriginal children with middle ear infections \$ 24,700

New Staff Research Funds - Cytokine polymorphisms in Sudden Infant Death Syndrome \$ 10,000

Babes in Arms - Cytokine polymorphisms in Sudden Infant Death Syndrome (2002-2003) A\$33,600

New Staff Research Funds - Cytokine polymorphisms in Sudden Infant Death Syndrome (2002-2004) \$ 10,000

Babes in Arms - Passive exposure to cigarette smoke, inflammatory and immune responses in young children (2005-2006) \$41,070

Hunter Children's Research Foundation - Assessment of *Alloiococcus otitis* for its potential as a pathogen in otitis media (2005-2006) (with A/Prof. J. Stuart) \$19,000.

Foundation for the Study of Infant Deaths (UK) - The effects of genetic background and cigarette smoke on inflammatory responses implicated in Sudden Infant Death Syndrome (2006-2008) \$ 295,000

Hunter Medical Research Institute - *In vitro* assessment of genetic and environmental risk factors for SIDS (2006) \$14,000

NHMRC – Stress during pregnancy and the developmental origins of renal disease in Aboriginal Australians. With Prof. R. Smith, Prof. E. Lumbers, Dr. E. Toussaint D'Espignet, A/Prof. P. Wadhwa, A/Prof. A. Bisitis (2009-2011, \$832,535).

Stillbirth Association of Australia - The role of infection and inflammation in stillbirths (2010) \$25,000

John Hunter Hospital Charitable Trust - Examination of a new species (*Alloiococcus otitidis*) implicated in ear infections (2014) \$28,000

Hunter Medical Research Institute - Describing the bacterial flora of the middle ear in health and disease (2015) \$20,000.

## Research supervision

- PhD supervision:
- D.F. Kinane, BDS, **PhD** (1983)
  - F.P. Winstanley, BSc, **PhD** (1984)
  - A. Rahat BA, **PhD** (1990)
  - F.Z.M. Aly, BSc (Hons), BDS, MBChB, FDS, **PhD** (1992)
  - M.W. Raza, MB, BS, (1992), **PhD**
  - A.A. Zorgani BSc, MSc, Dip.Bact (1993), **PhD**
  - A.T. Saadi, MBChB, **PhD** (1994)
  - G. Tzanakaki, BSc **PhD** (1996)
  - S.D. Essery, BSc, **PhD** (1997)
  - O.R. El Ahmer, BSc, **PhD** (1997)
  - A.M Al Kout, BSc, **PhD** (1997)
  - A.E. Gordon, BSc, **PhD** (1999)
  - O. Al Madani, MD, **PhD** (1999)
  - J.M. Braun, BSc **PhD** (2001)
  - S.M. Moscovis BSc (hons) **PhD** (2010)
  - C.I.J. Ashhurst-Smith **PhD** (2011)
- :
- MD supervision:
- G. W. Smith, MBChB, BSc, MRCP (1993), **MD**
  - S. Dundas, MBChB **MD** (2002)
  - M.W. Raza, MBChB, PhD **MD** (2006)

## **Publications**

### **Books published**

Blackwell, C.C. 1972. Unusual antibiotic resistance associated with the resistance transfer factor R1 in certain rough classes of *Salmonella typhimurium*. Ph.D. Thesis Stanford University School of Medicine.

Blackwell, C.C. and Weir, D.M. 1981. Principles of Infection and Immunity in Patient Care. Churchill Livingstone.

Blackwell, C.C. 1993. Genetic and environmental factors influencing susceptibility to infectious agents. DSc Thesis, Faculty of Medicine, University of Edinburgh.

### **Books edited**

Weir, D.M., Blackwell, C.C., Herzenberg, L.A. and Herzenberg, L.A. 1986. Handbook of Experimental Immunology (4th ed.). Blackwell Scientific Publications, Oxford

- Vol. 1 Immunochemistry
- Vol. 2. Cellular Immunology
- Vol. 3. Genetics and Molecular Immunology
- Vol. 4. Applications of Immunological Methods

Weir, D.M., Herzenberg, L.A., Blackwell, C.C., and Herzenberg, L.A. 1997 Weir's Handbook of Experimental Immunology (5th ed.) Blackwell Science

- Vol. 1 Immunochemistry and Molecular Immunology
- Vol. 2. Cell Surface and Messenger Molecules of the Immune System
- Vol. 3. The Lymphoid System
- Vol. 4. The Integrated Immune System

Blackwell, C.C. Blood groups and diseases. 1989. FEMS Immunology Microbiology (special issue on blood groups and diseases; festschrift for the 85<sup>th</sup> birthday of A.E. Mourant, FRS)

Blackwell, C.C. Sudden Infant Death Syndrome 1999. FEMS Immunology Medical Microbiology (special issue on infection and cot deaths)

Infection, Inflammation and SIDS (Thematic Issue). (2004) FEMS Immunology and Microbiology Ed. C.C. Blackwell. 42: 1-145.

### Papers in refereed journals

Blackwell, C.C. and Feingold, D.S. 1975. Frequency and some properties of clinical isolates of methicillin resistant *Staphylococcus aureus*. American Journal of Clinical Pathology, 64: 372-377.

Blackwell, C.C., Freimer, E.F. and Tuke, G.C. 1976. Cephatrozone (BL-S640): In vitro comparison with other cephalosporins. Antimicrobial Agents and Chemotherapy, 10: 288-292.

Blackwell, C.C., Young, H. and Bain, S.S.R. 1978. Isolation of *Neisseria meningitidis* and *Neisseria catarrhalis* from the genito-urinary tract and anal canal. British Journal of Venereal Diseases, 54: 41-44.

Freimer, N.B. Ogmundsdottir, H.M., Blackwell, C.C., Sutherland, I.W. Grahame, L. and Weir, D.M. 1978. The role of cell wall carbohydrate in binding of microorganisms to mouse peritoneal exudate macrophages. Acta Pathologica et Microbiologica Scandinavia Section B. 86: 53-57.

Blackwell, C.C., Young, H. and Anderson, I.S. 1979. Sensitivity of *Neisseria gonorrhoeae* to partially purified pyocines of *Pseudomonas aeruginosa* and a possible approach to epidemiological typing. Journal of Medical Microbiology, 12: 321-335.

Blackwell, C.C. and Law, J.A. 1981. Typing of non-serogroupable *Neisseria meningitidis* by means of sensitivity to R-type pyocines of *Pseudomonas aeruginosa*. Journal of Infection, 3: 370-378.

Blackwell, C.C., Winstanley, F.P. and Telfer Brunton, W.A. 1981. Sensitivity of thermophilic bacteria campylobacters to R-type pyocines of *Pseudomonas aeruginosa*. Journal of Medical Microbiology, 15: 247-251.

Weir, D.M., Blackwell, C.C. and McLean, C.M. 1981. Impaired bacterial binding to peritoneal exudate cells from mice with alloxan induced diabetes. Journal of Clinical and Laboratory Immunology, 5: 37-40.

Weir, D.M., Stewart, J., Glass, E.J., Oliver, A., Blackwell, C.C. and Doyle, J. 1981. Macrophage lectin receptors and bacterial recognition. Proceedings of the International Workshop on the Heterogeneity of Mononuclear Phagocytes. Ed. O. Forster. Academic Press.

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Kinane, D.F., Blackwell, C.C., Winstanley, F.P. and Weir, D.M. 1983. Blood group, secretor status and susceptibility to infection by *Neisseria gonorrhoeae*. *British Journal of Venereal Diseases*, 59: 44-46.

Blackwell, C.C., Kowolik, M.J., Winstanley, F.P., Kinane, D.F. Weir, D. M., Law, J.A. and Ho Wang Chok, Y.L. 1983. ABO blood groups and susceptibility to gonococcal infection I. Factors affecting phagocytosis of *Neisseria gonorrhoeae*. *Journal of Clinical and Laboratory Immunology*, 10: 173-178.

Winstanley, F.P., Blackwell, C.C., Weir, D.M. and Kinane, D.F. 1983. ABO blood groups and susceptibility to gonococcal infection. II. The relationship of lipopolysaccharide type to gonococcal sensitivity to the bactericidal activity of normal human serum. *Journal of Clinical and Laboratory Immunology*, 11: 27-32.

Kinane, D.F., Blackwell, C.C., Winstanley, F.P., Weir, D.M. and Elton, R.A. 1983. ABO blood groups and susceptibility to gonococcal infection. III. role of isohaemagglutinins in increased association of *Neisseria gonorrhoeae* to monocytes from blood group B individuals. *Journal of Clinical and Laboratory Immunology*, 12: 83-86.

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Weir, D.M., Blackwell, C.C., Stewart, J., Glass, E.J. and Oliver, A. 1983. Macrophage receptors for bacterial cell wall sugars and immune response genes. Possible determinants of susceptibility to infection. *British Journal of Rheumatology*, 11: (suppl. 2) 161-167.

Winstanley, F.P., Blackwell, C.C., Weir, D.M. and Kinane, D.F. 1983. Gonorrhoea: a predisposing factor for meningococcal disease? *Lancet* ii: 1134-1135.

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Winstanley, F.P., Blackwell, C.C., Tan, E.L., Patel, P.V., Parsons, N.J., Martin, P.V.M. and Smith, H. 1984. Alteration of pyocin-sensitivity pattern of *Neisseria gonorrhoeae* is associated with induced resistance to killing by human serum. Journal of General Microbiology, 130: 1303-1306.

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susceptibility to infections by *Candida albicans*. Protein-Carbohydrate Interactions in Biological Systems, ed. D. Lark, London: Academic Press, pp.231-233.

Blackwell, C.C., Jonsdottir, K., Hanson, M.F., Weir, D.M., Brettle, R.P. Todd, W.T.A., Chaudhuri, A.K.R. and Mathew, B. 1986. Non-secretion of ABO blood group antigens and susceptibility to infection with *Neisseria meningitidis* and *Streptococcus pneumoniae*. *Lancet*, ii: 284-285.

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Blackwell, C.C., James, V.S. and Weir, D.M. Gemmill, J.D., Patrick, A.W., Collier, A., Clarke, B.F. 1987. Secretor state of patients with type 1 or type 2 diabetes mellitus. *British Medical Journal* 295: 1024-1025.

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Shinebaum, R., Blackwell, C.C., Forster, P.J.G., Abernethy, V.E., Weir, D.M. and Nuki, G. 1987. Secretor state, Lewis blood group antigens and the spondylarthropathies. *Clinical Rheumatology* 6: 113-114.

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May, S.J., Blackwell, C.C., Brettle, R.P., MacCallum, C.J. and Weir, D.M. 1989. Non-secretion of ABO blood group antigens: a host factor predisposing to recurrent urinary tract infections and renal scarring. *FEMS Microbiology Immunology* 47: 383-388.

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Thom, S.M., Blackwell, C.C., McCallum C.J., Weir, D.M., Brettle, R.P. Kinane, D.F. and Wray, D. 1989. Non-secretion of blood group antigens and susceptibility to infection by *Candida* species. *FEMS Microbiology Immunology* 47: 401-406.

May, S.J., Blackwell, C.C. and Weir, D.M 1989. Lewis<sup>a</sup> blood group antigen as a receptor for *Candida* species. *FEMS Microbiology Immunology* 47: 407-410.

Holbrook, W.P. and Blackwell, C.C. 1989. Secretor state and dental caries in Iceland. *FEMS Microbiology Immunology* 47: 397-400.

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Blackwell, C.C., Aly, F.Z.M., James, V.S., Weir, D.M., Collier, A. Patrick, A.W., Cumming, C.G. and Clarke, B.F. 1989. Blood group, secretor status and oral carriage of *Candida* and other fungal species among patients with diabetes mellitus. *Diabetes Research*, 12: 101-104.

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carriage of *Neisseria meningitidis*. *Epidemiology and Infection* 104: 203-209.

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Rahat, A., Stewart, J., Blackwell, C.C. and Weir, D.M. 1990. A semi-quantitative method for measurement of H type 1 and H type 2 on buccal cells and in saliva of secretors and non-secretors. *Vox Sanguinis*, 59: 101-105.

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**EXPERT CERTIFICATE**

**In the matter of:**

**Police -v-**

**Name of expert:**

**Date: October 6, 2000**

**Page No.:** 1

**Name: Janice Ophoven, M.D.**

**Address: 6494 Crackleberry Trail, Woodbury, MN 55129 U.S.A.**

**Occupation: Pediatric Forensic Pathologist Telephone No.: 651-458-0541**

**STATES: -**

**EXPERT CERTIFICATE**

**Section 177, Evidence Act 1995 No. 25**

1. This Statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The Statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything which I know to be false or do not believe to be true.

2. I am 53 years of age.

3. I hereby certify:

My full name is: Janice Jean Ophoven, M.D.

My contact address is:

6494 Crackleberry Trail

Woodbury, Minnesota 55125

USA

I have a specialised knowledge based on the following training, study and experience:

I received my medical degree from the University of Minnesota in 1971

I completed residency training in Paediatrics at the University of Minnesota

I completed residency training in Anatomic Pathology at the University of Minnesota

I received specialty training in Paediatric Pathology at the University of Minnesota and Minneapolis Children's Hospital

I completed a fellowship in Forensic Pathology at the Hennepin County Medical Examiner's Office in 1980

I was the Associate Director and Director of Laboratories at the St. Paul Children's Hospital 1980 - 1988

**Witness:** \_\_\_\_\_

**Signature:** \_\_\_\_\_

**EXPERT CERTIFICATE****Page No.:2****In the matter of:****Police -v-****Name of expert:****Date: October 6, 2000**

I have maintained a practice in Paediatric Forensic Pathology for 20 years and have participated in the investigation of deaths and injuries in childhood in Canada and across the U.S.

I have provided courtroom testimony in deaths and injuries to children on numerous occasions.

I have been published in textbooks and peer-reviewed journals and have given educational seminars and workshops on issues pertaining to pediatric forensic pathology

4. From June 15 – August 13, 2000 I examined:

- Medical records of Kathleen Folbigg
  - Health Insurance Commission Records
  - Expert Certificate by Dr. Innis
  - Medical records supplied by Dr. Marley
  - Medical records supplied by Dr. Cash
- Medical records of Caleb Folbigg
  - Statement from Dr. Bridget Wilcken
  - Newborn Screening Blood results
  - Newcastle Western Suburbs Hospital records
  - Coroners Brief
  - Ambulance Records
- Medical records of Patrick Folbigg
  - Statement of Dr. Bridget Wilcken
  - Newborn screening blood results
  - Medical records from Newcastle Western Suburbs Hosp
  - Statement from Dr. Wilkinson
  - Medical Certificate of cause of death
  - Cause of death certificate (hand written)
  - History, examination and progress notes
  - Report by Dr. Wilkinson to Marley
  - Report by Dr. Wilkinson to Dr. Morris
  - Adelaide Children's Hospital Pathology Report
  - Mater Hospital Pathology reports
  - Report by Dr. Challinor to Dr. Wilkinson
  - Biochemistry reports
  - Report by Dr. Wilkinson to Dr. Thomas
  - Physiotherapy report
  - Autopsy report
  - Report by Dr. Wilkinson to Dr. Bale
  - HAPS reports
  - Histopathology Dept Report
  - Report by Dr. Wilkinson to Folbiggs
  - Report by Dr. Colley to Dr. Wilkinson
  - Report by Dr. Marley to Dr. Holland
  - Dr. Colley to Dr. Wilcken
  - Dr. Wilkinson Dr. Colley
  - Dr. Edwards to Dr. Hardacre
  - Newcastle Mater Hospital Records June 14, 1990
  - Newcastle Mater Hospital Records October 18, 1990
  - Newcastle Mater Hospital Records November 4, 1990

**Witness:** \_\_\_\_\_**Signature:** \_\_\_\_\_

**EXPERT CERTIFICATE**

Page No.:3

In the matter of:

Police -v-

Name of expert:

Date: October 6, 2000

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- Newcastle Mater Hospital Records November 14, 1990
  - Newcastle Mater Hospital Records December 22, 1990
  - Statement by Dr. Marley
  - Pediatric Summary
  - Ambulance Records
  - Beresfield Crematorium record
  - Medical records of Sarah Folbigg
    - Statement by Dr. Wilcken
    - Newborn Screening Blood Results
    - John Hunter Hospital Records
    - Statement by Dr. Marley
    - Pediatric discharge
    - Perinatal database
    - Reports: Dr. Hardacre to Dr. Marley
    - Buckner to Holland
    - Hardacre to Marley
    - Hardacre to Holland
    - Pickford to Marley
    - Edwards to Hardacre
    - Handwritten notes
    - Ambulance Records
    - Coroners Brief
  - Medical records of Laura Folbigg
    - Statement of Dr. Wilcken (1.14.00)
    - Newborn Screening Blood Results
    - Statement of Christopher Seton
    - Handwritten sleep notes by Kathleen Folbigg
    - Report by Dr. Seton to Det. Ryan
    - Referral by Dr. Seton to Dr. King
    - Letter by Mr. Folbigg to Dr. Seton
    - Report by Dr. Seton to Mr. Folbigg
    - Newborn discharge summary
    - Report by Dr. Seton to Dr. King
    - Corometrics monitor supply record
    - Urine metabolic profile
    - Sleep study report (10.7.97)
    - Royal Alexandra Hospital for Children Medical History
    - Sleep study report by Seton to Sanders
    - Letter by Craig Folbigg to Margaret Tanner
    - Report by Seton to Craig Folbigg
    - Report by Seton to Dr. Sanders
    - Patient alarm traces (Corometric monitor print outs)
    - Statement of Dr. Innis
    - Information sheet
    - Progress Notes
    - Singleton Hospital Records
    - Ambulance report
    - Fairholme Surgery Records
    - Statement of Dr. Cash
    - Newborn discharge summary
    - Report by Dr. Seton to Dr. King
    - Sleep study reports 10.7.97 and 2.3.98
    - Report by Dr. Seton to Craig Folbigg
    - Report by Dr. Seton to Dr. Sanders
    - Ambulance records
  - Transcript of interview with Kathleen Folbigg
  - Psychological report by Roz Garbutt

Witness: \_\_\_\_\_

Signature: \_\_\_\_\_

**EXPERT CERTIFICATE**

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In the matter of:

Police -v-

Name of expert:

Date: October 6, 2000

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- Diary entry (19.2.89)
  - Diary entries (June 4, 1996 – June 5, 1997)
  - May 1992 Gibbs personal diary entry
  - Diary entry July 1999
  - Diary Entry June 6, 1997 – April 10, 1998
  - Diary Entries January 1 – 3 1999
  - Diary entry June 19, 1999
  - Telephone Interception transcripts
  - Listening Device Transcripts
  - 26 autopsy photos of Laura Folbigg
  - 53 Microscopic Slides-Laura
  - 40 Microscopic Slides-Sarah
  - 21 Microscopic Slides-Patrick
  - 14 Microscopic Slides – Caleb
  - Meeting with Constable B.Ryan on June 19, 2000

**5. The following is a list of my findings**Caleb Gibson Folbigg

- Caleb was the product of a fullterm pregnancy and his 21-year-old married mother, Kathleen Folbigg, received adequate prenatal care. A “fainting” episode and a bout with the chicken pox complicated the pregnancy.
- Caleb was delivered vaginally with forceps assistance following an essentially uncomplicated labor on February 1, 1989. The baby’s birth weight was 3230 grams and apgars were 9 at 1 and 9 at 5 minutes. His newborn course was complicated by a brief bout with transient tachypnea [mild respiratory distress] that resolved without difficulty. He was discharged home with his mother.
- His pediatrician, B.J. Springthorpe, at well child evaluation noted inspiratory stridor when the child was placed supine or agitated. The problem was characterised as mild laryngomalacia and no further followup was recommended.
- In the early morning of February 20, 1989, Kathleen fed Caleb [approximately 0100 hours]. Kathy checked on the baby again at 0250 hours and found him “cold” with bloody froth in his nose and mouth. Emergency medical services were called and they found the child in full cardiopulmonary arrest [essentially DOA], his skin warm to the touch, pale and cyanotic. The child was pronounced dead around 0300 hours on February 20, 1989.
- Autopsy examination was performed by Dr. R. Cummings at 1145 hours on February 20, 1989 in the City Morgue, Newcastle, New South Wales. His findings included:
  - A well developed, well nourished male infant, weight 3970 g.

Witness: \_\_\_\_\_

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- The stomach contained curdled milk.
  - The lungs appeared congested and there were extravasated red blood cells in the tissue.
  - There was no mention of petechial hemorrhages, specifically in the thymus.
  - Routine toxicological analysis was negative.
  - Cause of death: SIDS [Sudden Infant Death Syndrome]
  - Bridget Widcken performed complete biochemical profile on blood samples from Caleb. The results were entirely normal.

**Patrick Folbigg**

- Patrick was the product of a fullterm pregnancy and his mother, Kathleen Folbigg, received adequate prenatal care. The pregnancy was uncomplicated.
- Patrick was delivered vaginally following an essentially uncomplicated labor on June 3, 1990. The baby's birth weight was 3410 grams and apgar was 8 at 5 minutes. His newborn course was uncomplicated and he was discharged home with his mother.
- His pediatricians were Richard Henry and Barry Springthorpe. He was scheduled for a sleep study for one week after his discharge. The examinations showed no GE reflux and the sleep study was normal.
- In the early morning of October 18 1990, Patrick's mother reports that she heard him coughing at approximately 0300 hours. At 0430 she was up and heard him "gasping" in his room and found him cyanotic, lifeless and making minimal respiratory effort. Emergency responders arrived around 0500 hours and provided oxygen and respiratory support. He improved spontaneously and was admitted to the hospital through the emergency department. During hospitalisation the child developed right-sided seizures that proved over time to be difficult to control and required multiple subsequent hospitalisations. Normal EEG is present in the record from October 18, 1990.
- CT scans revealed bilateral abnormalities of the brain specifically in the occipital lobes of the brain. The child also presented with severe visual deficits. The attending physicians evaluated a multitude of possible etiologies including herpes encephalitis, but eventually concluded that he suffered from encephalopathic

**Witness:\_\_\_\_\_****Signature:\_\_\_\_\_**

**In the matter of:****Police -v-****Name of expert:****Date: October 6, 2000**

disorder, cause unknown. The findings were consistent with a severe hypoxic event. Despite these physical setbacks the baby continued to show satisfactory growth and development.

- On the morning of February 13, 1991, Patrick's mother put him down for a nap at ~ 0730 hours and she found him lifeless at 0930-1000 hours. Emergency responders arrived at ~1020. Emergency medical services found the child in full cardiopulmonary arrest [essentially DOA], his skin warm to the touch, pale and cyanotic. The child was pronounced dead at 1040 hours. The baby's father was apparently not present in the household at the time of his death.
- Dr. J. Bishop and Dr. G. Singh-Khaira performed autopsy examination at 1230 hours on February 13, 1991 in the Pathology Department of Newcastle Mater Hospital, Waratah. Their findings included:
  - A well developed, well nourished male infant, weight 8.57 kg.
  - The lungs showed posterior dependant congestion.
  - There was no mention of petechial hemorrhages, specifically in the thymus. The thymus was described as large.
  - Routine and special analyses were negative.
  - Neuropathology examination revealed laminar cortical necrosis of the brain with cystic degeneration in the visual cortex. This is most consistent with old infarcts occurring at the time of his arrest at age 5 months. No evidence of congenital abnormalities was present.
  - Cause of death: SIDS [Sudden Infant Death Syndrome]
  - Note: Dr. Wilkinson, the baby's paediatrician, noticed petechial hemorrhages that were interpreted as agonal. No note in the autopsy report is present.
- Bridget Widcken performed complete biochemical profile on blood samples from Patrick. The results were entirely normal.

**Sarah Folbigg**

- Sarah was the product of a fullterm pregnancy and her mother, Kathleen Folbigg, received adequate prenatal care.
- Sarah was delivered vaginally following an essentially uncomplicated labor on October 14, 1992. The baby's birth weight was 3020 grams and apgars were 9 at

**Witness: \_\_\_\_\_****Signature: \_\_\_\_\_**

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- 1 and 10 at 5 minutes. The parents elected to take the baby home with apnea monitoring. She was discharged home with her mother.
- She underwent sleep studies November 15, 1992 and the results were interpreted as within normal limits. Overall well child visits did not indicate any reason for concern; the baby's growth and development were good. The baby had a history of snoring at sleep.
  - The father reported increasing tension between the mother and Sarah.
  - The baby was put to sleep in a single bed in the parent's room at about 2100 hours on August 29, 1993 without the monitor. The mother reports hearing the child "turn over" at about midnight. She got up to go to the bathroom at 0130 hours on August 30, 1993, did not hear her breathing and found her lifeless. Emergency services were summoned. Emergency medical services were called and they found the child in full cardiopulmonary arrest [essentially DOA], skin warm to the touch, pale and cyanotic. The child was pronounced dead at 0130 August 30, 1993.
  - Dr. John Miller Napier Hilton performed autopsy examination at 0800 hours on August 31, 1993 in Sidney. His findings included:
    - A well developed, well nourished female, weight 9.44 kg.
    - Small scratches on the right upper arm, below the lower lip on the left and on the chin.
    - The stomach contained curdled milk.
    - The lungs showed pulmonary edema and congestion.
    - There were petechial hemorrhages present, specifically in the thymus, heart and lung.
    - Routine toxicological analysis was negative.
    - Cause of death: SIDS [Sudden Infant Death Syndrome]
  - Bridget Widcken performed complete biochemical profile on blood samples from Sarah. The results were entirely normal.

**Laura Folbigg**

- Laura was the product of a fullterm pregnancy and her mother, Kathleen Folbigg, received adequate prenatal care.

**Witness:\_\_\_\_\_****Signature:\_\_\_\_\_**

**EXPERT CERTIFICATE****Page No.:8****In the matter of:****Police -v-****Name of expert:****Date: October 6, 2000**

- 
- Laura was delivered vaginally following an essentially uncomplicated labor on August 7, 1997. The baby's birth weight was 3260 grams and apgars were 9 at 1 and 10 at 5 minutes. The parents elected to take the baby home with apnea monitoring. She was discharged home with her mother.
  - She underwent sleep studies under the care of Dr. Chris Seton. His impression was that the child had a mild central apnea that resolved over time and were interpreted as of no medical significance. At no time did her clinical picture or studies show evidence of obstructive apnea. Her care was monitored by the medical staff at New Children's Hospital, Westmead [Ms Margaret Tanner].
  - The father reported concerns about Kathleen's use of the monitor during the day when he was not present.
  - Laura received her family medical care from Doctor Sanders of singleton and Dr. Innis of Singleton Heights Medical Practice.
  - Laura had recently seen Dr. Innis for her 18-month routine well child visit and vaccination.
  - Laura had a history of one week of cold and flu-like syndrome, and she had been administered Demazin for treatment of symptoms. She received her last dose of the medication on February 27, 1999.
  - On March 1, 1999 Kathleen took the child to the gym and to her father's place of work to "visit ". Kathleen reported that she fell asleep in the car and she put her to bed upon arrival home at ~ 1100hours. Approximately 30-60 minutes later Kathleen reported hearing the child "coughing in the bedroom." She checked on her ~ 5 minutes later and found her supine and lifeless. She started CPR, emergency medical services were called, and they arrived at 1214 hours finding the child in full cardiopulmonary arrest [essentially DOA], skin warm to the touch. The child was pronounced dead at Singleton hospital at 1245 hours March 1, 1999. SIDS Death Scene Investigation Checklist was completed.
  - Autopsy examination was performed by Dr. Allan David Cala at ~2100 hours March 1, 1999 at NSW Institute of Forensic Medicine, Glebe. His findings included:
    - A well developed, well nourished 20-month-old female, weight 11.52 kg.
    - There was lividity on the left side of the face and posteriorly.

**Witness:\_\_\_\_\_****Signature:\_\_\_\_\_**



**EXPERT CERTIFICATE****Page No.:9****In the matter of:****Police -v-****Name of expert:****Date: October 6, 2000**

- 
- No significant physical injuries were identified on physical examination.
  - The lungs showed focal hemorrhage and collapse.
  - Examination of the heart showed no gross abnormalities. Microscopic examination of the tissues from the heart revealed inflammatory infiltrate in the heart, consistent with viral myocarditis.
  - Toxicological analysis was noncontributory.
  - There were petechial hemorrhages present in the thymus.
  - Routine toxicological analysis was negative
  - Cause of death: Undetermined
  - My review of the autopsy materials reveals the presence of myocarditis, most probably viral in origin. Dr. Cala states in his report that his finding of myocarditis is consistent with Laura's recent illness and is probably incidental. I concur with this conclusion.
  - Bridget Widcken performed complete biochemical profile on blood samples from Laura. The results were entirely normal.

**6. Conclusions:**

- In forming my conclusions, I have utilized all of the materials made available to me including the medical history and records, the autopsy reports and materials, the police investigative documents, the interview transcripts from Kathleen Folbigg, diary entries, witness statements, and listening device materials.
- The materials and investigative information provided in this case are of excellent quality and are sufficient for me to render an opinion to a reasonable degree of medical certainty.
- It is my opinion that these four children were all the victims of homicidal assaults that resulted in their suffocations. Suffocation is the interference with breathing by external obstruction of the nose and mouth. This process will take approximately 4 to 5 minutes to complete. During the first 1 and 1/2 to 2 minutes, while they are still fully conscious, the child will fight aggressively for their life. In small infants, this typically does not result in any external signs or physical evidence.

**Witness:** \_\_\_\_\_**Signature:** \_\_\_\_\_

**EXPERT CERTIFICATE**

Page No.:10

In the matter of:

Police -v-

Name of expert:

Date: October 6, 2000

- 
- I have participated in the investigation of both accidental and homicidal suffocation in children in over the course of my 20 years as a practicing pediatric forensic pathologist. Unfortunately multiple infant homicides within one family are now well documented in the literature and in forensic experience. Typically the perpetrator does not confess to the crimes but in many cases such as this the facts of the case make the diagnosis. Important facts in this case that lead to the conclusion of homicidal suffocation include:
- The autopsy fails to identify any known natural disease or disease process that could explain the sudden deaths of these infants. All four children were growing and developing normally for their age and circumstance. Despite Patrick's handicaps he was advancing well.
  - The autopsy findings in these babies are all consistent with death by suffocation.
  - The infants were all in the care of the same person at the time of their death, their mother, and she was the last person to see each of them alive.
  - None of the deaths in this case can be attributed to SIDS [Sudden Infant Death syndrome]. It is well recognized that the SIDS process is not a hereditary problem and the statistical likelihood that 4 children could die from SIDS is in excess of 1 in a trillion.
  - The diagnosis of SIDS requires that following a complete investigation and autopsy no other cause of death is identified. Forensic standards of practice would not allow for consideration of a second diagnosis of SIDS after a second sudden death and by the time a third child has died, the death must be investigated as a homicide.
  - Patrick's sudden, profound and irreversible brain damage is consistent with and diagnosed as a hypoxic episode. Hypoxia in this case is synonymous with asphyxia and unfortunately heralds the fatal event in retrospect. No natural disease or process has been identified to explain this event. In my opinion, the cause of Patrick's cardio-respiratory arrest is the same process that killed him and his siblings.
7. In my opinion the cause of death and manner of death should be listed as follows:

Witness: \_\_\_\_\_

Signature: \_\_\_\_\_

**EXPERT CERTIFICATE**

**Page No.:11**

**In the matter of:**

**Police -v-**

**Name of expert:**

**Date: October 6, 2000**

---

Caleb Folbigg-	<b>Cause of Death:</b> Undetermined
	<b>Manner of Death:</b> Undetermined
Patrick Folbigg -	<b>Cause of Death:</b> Suffocation
	<b>Manner of Death:</b> Homicide
Sarah Folbigg -	<b>Cause of Death:</b> Suffocation
	<b>Manner of Death:</b> Homicide
Laura Folbigg -	<b>Cause of Death:</b> Suffocation
	<b>Manner of Death:</b> Homicide

Witness: \_\_\_\_\_

Signature: \_\_\_\_\_

**Witness:** \_\_\_\_\_

**Signature:** \_\_\_\_\_

THE REGIONAL MEDICAL GENETICS UNIT  
for NEWCASTLE AND NORTHERN NEW SOUTH WALES

CASE RECORD

Reference No. 1564 Cross to \_\_\_\_\_

Name of Patient/Consultand Kathleen Megan FOLBIGG DOB 14.6.67 Sex F

Address 9 Dower Close Thornton Postcode 2322

Telephone: Home 66 4489 Work 57 1099

Referred by Dr Chris Marley Family Doctor Same  
Geoff Hardacre 343534

Date	Notes
12/11/91	Craig Gibson 25-11-61. MR + MRS Folbigg S/B AC. To collect information & send for another appt. A Colley.
1/12/91	Olw Dr B Wilkin - to write letter re: blood spots for mCAD. Make app early Feb. A.C. to contact B. Wilkin A Colley. 18/2/92
18/2/92	MR + MRS Folbigg + MR's sister. S/B AC. <del>Pregnancy counselling.</del> <del>See baby.</del> A Colley.
30/10/92	13 DEC 1992 New baby born ♀ Sarah 14/10/92 For <del>single</del> glycone to Olive Latham in line of mCAD. On sleep monitor & to have a sleep study 5/11/92 A Colley.
5/11/93	Kathleen, Craig & Craig's sister Carol + Robert S/B AC at WSH. Dlw J Chintodoulen - No new ideas, no specimens needed from parents. For ① crisis phone No ② counselling pamphlet ③ Henry Wellmore phone No. ④ Adoption <del>YES</del> /DOCS phone No. A Colley.

PROGRESS NOTES:

27/11/91

From reading mater Notes + reports its clear I think, that Patrick suffered cerebral infarcts in a near-miss SIDS at  $\frac{5}{12}$  + subsequent problems were due to this.

Question - why the near-miss at  $\frac{5}{12}$ ?

? Same as brother had.

A Colley.

Pregnant Dr M. Holland Otago.

check up today. weeks 8/40.

Scan 9/40

30/10/92

Baby Folbigg  
16 days. baby (girl)

Sarah

DOB: 14/10/92 JHM.

SBCus Cooper - on monitor

sleep study 5<sup>th</sup>/11/92 at JHM.

Suggest urine for acyl glycine → O.C. Lab.

A Colley.

30/8/93

Sarah (Mrs C) continue MCAD test (✓)

Died at 10 1/2 age last night

on monitor till 1 1/2 age

→ would have kept this on

Dr. A. Butler - suggest Globe Coroner visit take skin for fingerprint culture, spleen for DNA extraction - banking, and freeze whole to -70°C for transport to Chris Lutton in dry ice.

Sh Chris Lawrence Globe Coroner (ant Pathologist)

02 660 5977 - hell take skin. Want be able to collect

muscle till tomorrow

Called Meredith Wilson 02 692 6273 - (F&V Clinic) 516 7101

she'll call back at 12.45pm

Called Eddie Smith - she's happy to culture

1910

Individent  
12/11 had been last 1/2 to see ... on X 10 and felt ... JR

Caleb - <sup>pregnancy</sup> well, fainted - low BP. chicken pox.  
no H/T, UTI.

- term preps Bwt 7lb 7 3/4oz. 19 1/2" long
- humidifier over night, brined from forepots
- bottle - "very larynx" needed encouragement home day 5. gaining weight.
- no abn noted at all.

Routine check = Springfield - feeding - strider!

P.M. SIDS

phototherapy for jaundice.

Patrick

pregnancy OK, UTI

39/40 Natural Cephalic

7lb 1 3/4oz. 22" long.

fairer than Caleb but same features.

No humidifier.

No strider, no floppy larynx.

Bottle well. Good appetite.

no jaundice.

Day 6 home

No abn noted

Dr Morris - N checks.

Sat up alone 6/12.

3/12 - unconscious in bed  
3am

limp, gasping, pallor.  
breathing deeply.

Mater

↑ Post ictal ??

fit - status on & off 8-12hrs

Continued to have focal fits after  
this

result fits - cortical blindness.

Royal Blind Society.

22/12/91 → long 1 1/2 hr fit.

physically N

night before 11.30pm vomit +

clinging.  
empt, sweating

up OK 8am → down to sleep.

10am → dead.

( 8/8 Jan Wilkinson

he had breakfast.

P.M. SIDS.

OUR REFERENCE

YOUR REFERENCE  
2 APRIL 2003

DATE



OFFICE OF THE DIRECTOR  
OF PUBLIC PROSECUTIONS

265 Castlereagh Street  
Locked Bag A8  
Sydney South NSW 1232  
DX 11525 Sydney Downtown  
Telephone (02) 9285 8611  
Facsimile (02) 9285 8600

H

Legal Aid  
FAX:  
Attn: Peter Krisenthal

Dear Mr Krisenthal,

**R-v-Kathleen FOLBIGG  
IgG TESTING**

We refer to your letter dated 28 March 2003.

We note that, on 1 April 2003, we handed to you the attached e-mail from Dr Charles Hii dated 31 March 2003.

It is our understanding from Dr Hii's e-mail and from speaking with Dr Hii that he cannot give an assurance that the results of any testing which he might conduct would be accurate. This is because the proteins may have been affected by the way in which the blood was stored. If the protein molecules were relevantly affected, then it would appear in the test results that there was an IgG deficiency even though that result may only have arisen as a result of the storage of the blood as opposed to any condition of the child. The test could not distinguish between these 2 potential causes of small levels of IgG molecules.

We advise that Detective Ryan spoke with Dr Charles Hii on 2 April 2003. Dr Hii asked Detective Ryan whether the 4 Folbigg children had presented with infections or a failure to thrive. Dr Hii said,

"If any of these children died from IgG deficiency, they surely would have been hospitalised on a number of occasions with recurrent infections".

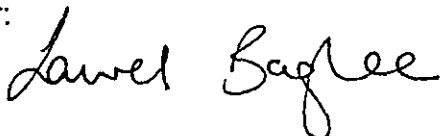
We note our advice to you today that, as suggested by Dr Charles Hii, Detective Ryan is making enquiries to locate a protein biochemist who can indicate whether or not the preservative used to store the blood sample of Laura Folbigg would have corrupted the protein molecules for the purpose of this testing.

We confirm our earlier advice that we are prepared to have the blood sample tested by a suitably qualified person nominated by you, provided that person is able to certify that the results will be reliable and accurate. We are concerned to obtain this assurance because, once the blood sample is thawed and processed for testing, the blood sample is corrupted and rendered unsuitable for any future analysis.

Yours faithfully,

SE O'Connor  
Solicitor for Public Prosecutions

Per:

A handwritten signature in black ink, appearing to read "Laurel Baglee". The signature is written in a cursive, flowing style.



ver, Jane

From: RYAN, BERNARD [RYAN1BER@POLICE.NSW.GOV.AU]  
Sent: Monday, 31 March 2003 7:24 PM  
To: Culver, Jane  
Subject: IgG

--- Received from NSWP.RYAN1BER 0248232008  
18:24 -----

31/03/03

Jane,

Email from Dr Hii.

Bernie

-----  
-----  
From: charles.hii@adelaide.edu.au  
To: ryanlber@police.nsw.gov.au  
Date: Mon, 31 Mar 2003 16:35:48 +0930  
Subject: IgG

Dear Bernie,

Following our conversation this morning, here is a summary of the issues covered.

In theory, our ELISA should be able to detect IgG in any sample, provided it is in liquid form or can be dissolved if it is solid. Any colouration in the sample due to haemolysis is of little or no concern as there are a number of washing steps in the procedure. However, the ELISA will not be able to determine the degree of IgG degradation if there is any. We are of the opinion that immunoglobulins are fairly stable, especially if the sample has been stored properly. The presence of other serum proteins in whole blood will protect IgG from being degraded by proteases and the activity of proteases will be minimal at -4 or -20oC. According to Tanya, the sample has been stored at -4oC for up to 5 weeks after collection and at -20oC until a month ago when it was brought back to -4oC. We do not see any problem in this, especially when bacterial growth is stopped by preservatives in the tube. However, Tanya told me that oxalate was used as a preservative and I am not sure whether this affects protein structure/shape. A protein biochemist will be able to confirm this and I am not one. If it does, then the ELISA will not detect the IgG.

Tanya also said that she was not able to get any liquid out even after centrifugation. However, she did not use an ultracentrifuge for this. Although we have not tried this on frozen blood before, my colleagues and I are of the opinion that it should be possible to get some "serum-like" liquid out if the sample is subjected to ultracentrifugation. I can get the lab to test on a sample tomorrow.

If we manage to get a value for the IgG and if it is not considered to be deficient for the age group of the child, won't this be of use? You can

Discuss this with Peter Krisenthal. If you decide to send the sample,  
please allow for up to 2 weeks to get the results.

Regards,  
Charles

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-----  
All mail is subject to content scanning for possible violation  
of New South Wales Police Service Electronic Mail Policy.  
All persons are required to familiarise themselves with the  
content of the policy located on the MEMO Bulletin Board  
and on the NSWPS Intranet.  
-----  
-----

----- 31/03/03 18:24 ----- Sent to

-----  
-> JCulver@odpp.nsw.gov.au

\*\*\*\*\*  
This message and any attachment is confidential and may  
be privileged or otherwise protected from disclosure. If you  
have received it by mistake, please let us know by reply  
and then delete it from your system; you should not copy  
the message or disclose its contents to anyone.  
\*\*\*\*\*

R-v Kathleen FOLBIGG

Draft Supplementary Report

PJ Berry (Paediatric Pathologist)

I have been asked to provide a supplementary report describing the significance of Dr David Drucker's work on IL-10 in the Sudden Infant Death Syndrome, and his statement that:

"the test on Sarah [Folbigg] found she had two copies of the 'cot death gene' which would obviously increase her risk of SIDS". (Email 12<sup>th</sup> March 2003)

I have also been shown a separate undated communication from Dr Drucker in which he states:

"The samples we received have proved exceptionally difficult... we had to repeat the analyses five times until we had optimised conditions for the sample. Sarah is homozygous for the so-called cot death gene. She is at higher risk than even a baby with one copy would have been."

I have not been shown a formal report from Dr Drucker.

**Background**

In a paper submitted to the journal Human Immunology in May 2000 and published later that year, Dr Drucker and colleagues described an association between a particular naturally occurring variant of a gene and the Sudden Infant Death Syndrome. The gene in question encodes for IL-10, a substance that is important in inflammation and the response to infection. The particular variant (IL-10 592A) implicated by Dr Drucker and colleagues is believed to result in lower production of IL-10. They hypothesised that this genetic variant might predispose to SIDS "by tardy initiation of protective antibody production [and hence susceptibility to infection] and a lower capacity to inhibit inflammatory cytokine production".

This, and other theories implicating novel mechanisms and infection have rightly attracted considerable interest among SIDS researchers.

This paper also received considerable attention in the media, which prematurely conferred the pejorative label "cot death gene". This label is misleading, not least because many cot death victims do not have this gene variant, and the vast majority of people with it lead healthy lives.

SIDS research is littered with abandoned theories so that most cot death researchers do not accept new findings until another independent group has confirmed them. For example, more than a dozen separate studies involving hundreds of SIDS victims have confirmed the risk of placing babies to sleep in the prone position, so that risk

*noted*

*I*  
IL-10 G

*Received*  
*29/4/03.*

factor is accepted. Many other theories have not been independently confirmed, and so are not generally accepted.

The most common problems with SIDS studies involving statistics are small numbers of cases, case selection and inappropriateness of controls. The IL-10 study, while it involves very sophisticated laboratory methods, is essentially a statistical study comparing the frequency of the IL-10-592A variant in a group of SIDS cases and controls.

#### The size of the study

The study examined just 23 SIDS cases, and so cannot be regarded as anything more than an interesting preliminary study at this stage.

*more on second study w/ similar results*

#### Case selection

The SIDS cases were included on the basis that they had been "subject to detailed postmortem examination in the North West Regional Perinatal Pathology Department in St Mary's Hospital (Manchester, United Kingdom)." The only way of being certain that a group of cases is unselected is to study all consecutive cases from a defined population over a defined period. If this is not done, then there is a possibility of selection bias, as for example if coroners directed "medical" sounding cases to the perinatal pathology department at St Mary's, and "legal" sounding cases to forensic pathologists elsewhere. Another possible source of bias arises if the diagnosis of SIDS is simply taken from the post-mortem report without further critical review; in this study no exclusions were made, and the authors state "it is possible that babies who died of other causes may have been included." From the paper alone, it is not possible to be confident that the SIDS group is free from selection bias.

#### Controls

The selection of appropriate controls depends on the question being asked in the particular study. In this case the question is; do SIDS babies carry the IL-10-592\*A gene variant more often than babies who do not die? The appropriate control group should therefore be made up of living babies carefully matched for possible confounding factors, and that matching should be tested as part of the analysis of the results of the study.

In this study the authors state that "The control group and their cytokine allele frequencies were those described by Perrey et al consisting of healthy, and sex- and ethnically-matched individuals from the North West Region." The study of Perrey et. al. uses as its control group "principally healthy volunteers or cadaveric renal transplant donors". Curiously, the figures for IL-10\*-A and IL-10\*-C among the 330 controls quoted in the Drucker paper (161 and 499 respectively) are not the same as those in the Perrey paper (151 and 509 respectively).

This control group is clearly prone to serious selection bias, and is inappropriate as a control group for a SIDS study. Indeed, Perrey et. al. warn that "Any study should have a set of matched controls, particularly in studies of other ethnic groups. We have reason to suggest that allele distribution may be quite different in other populations".

### The significance of the study

Assuming that the results are unaffected by selection bias, then the presence of the IL-10-592\*A gene confers about a 3-fold increased risk of SIDS. This is a relatively weak association, comparable to side sleeping, but considerably less than other established risk factors such as prone sleeping where the risk is increased by about 8-fold, head covering where the risk is increased about 30-fold, or sleeping with an adult on a sofa when the risk is increased about 50-fold.

not significant situation  
25 fold increase  
to the fatal  
cause  
in my  
view

Using the SIDS rate quoted in the paper of 0.62 per 1000 live births, a 3-fold increase in risk would mean that an affected baby would have a risk of dying of SIDS of less than 1 in 500.

Dr Drucker says that Sarah Folbigg "is at higher risk than even a baby with one copy would have been". It may be that, if the association is substantiated, then babies with two copies will be at greater risk than babies with one copy of IL-10-592\*A. In their paper, the authors pooled the results from babies with one and two copies of IL-10-592\*A, so that the figure of 3 is an "average". It is therefore not possible to say from the data presented that the risk for babies with two copies is greater than that for babies with one copy of IL-10-592\*A.

### Is IL-10-592\*A a cause of death in SIDS?

Assuming that the results are unaffected by selection bias, then the authors have demonstrated an association between IL-10-592\*A and SIDS.

They have not presented data to support their theory that this gene variant predisposes to infection or an aberrant inflammatory response. For example, there are no data about antibody levels, nor are the presence of infection or inflammation in SIDS babies with and without this gene variant compared.

IL-10-592\*A therefore remains a possible *association* only, and cannot be invoked as a *cause* of death in SIDS. No pathologist to my knowledge has ever invoked IL-10-592\*A when certifying the cause of death of a baby who has died suddenly and unexpectedly.

### Independent confirmation

Dr Drucker and his colleagues conclude, "If the present study can be confirmed in a larger analysis, then IL-10 genotyping may provide a means to identify children at increased risk of SIDS..."

I am not aware of any other study confirming the association between IL-10-592\*A and SIDS. I have carried out a Medline search with negative results.

Regional studies

I am aware that Dr Drucker has received funding to carry out a further study, but to my knowledge this has not yet been published.

### Conclusion

1. The work of Dr Drucker and his colleagues is of scientific interest

2. The published results of this group concerning IL-10-592\*A and SIDS can only be regarded as a preliminary study
3. There are several possible sources of selection bias in cases and controls which may make the conclusions unreliable
4. The results have not been confirmed in an independent study
5. If these findings are confirmed, then IL-10-592\*A confers a modest increase in the risk of SIDS
6. This is an association only, and not a cause of SIDS
7. The great majority of individuals with IL-10-592\*A do not die as cot deaths
8. The use of the term "cot death gene" in respect of IL-10 is pejorative and frankly misleading

Unsigned  
26/03/03

29/04/03



**Department for Administrative  
and Information Services**

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Fax +61 8 8226 7777  
DX 510 Adelaide  
Email [admin@forensic.sa.gov.au](mailto:admin@forensic.sa.gov.au)

18/10/02

To: Mr Peter Krisenthal,  
Solicitor,  
Legal Aid NSW,  
Central Square Building,  
Cnr. Castlereagh & Hay Sts.  
Sydney, NSW 2000,  
PO Box K847,  
Haymarket, NSW, 2000

**Re: the deaths of Caleb, Patrick, Sarah and Laura Folbigg**

I have been asked by Mr. Peter Krisenthal in two letters dated 8/8/02 and 16/9/02 to provide an opinion as to the causes of death of these infants. In preparing this report I have based my opinions on:

- 1) An autopsy report on Caleb Gibson Folbigg by Dr. R. Cummings dated 9/5/89;
- 2) An autopsy report on Patrick Allan Folbigg by Dr. J. Bishop and Dr. G. Singh-Khaira dated 14/2/91
- 3) A neuropathology report on Patrick Allan Folbigg by Dr. A. Kan dated 24/6/91;
- 4) An autopsy report with associated ancilliary testing on Sarah Kathleen Folbigg by Assoc. Prof. J.M.N. Hilton dated 25/11/93;
- 5) A neuropathology report on Sarah Kathleen Folbigg by Dr. R. Pamphlett undated;
- 6) An autopsy report with associated ancilliary testing on Laura Elizabeth Folbigg by Dr A.D. Cala dated 26/7/99;
- 7) A series of 48 glass slides from Laura Elizabeth Folbigg;

- 8) A series of 29 colour autopsy photographs of Laura Elizabeth Folbigg;
- 9) A blue folder of medical records of Caleb Folbigg;
- 10) A black folder of medical records of Patrick Folbigg;
- 11) A blue folder of medical records of Sarah Folbigg;
- 12) A blue folder of medical records of Laura Folbigg;

I have also received reports and statements of expert opinions of:

- 1) Dr B. Wilcken dated 10/12/91 & 14/1/00;
- 2) Dr. A. Colley dated 4/12/91 & 27/2/92;
- 3) Dr. S.M. Beal dated 8/12/99;
- 4) R. Garbutt dated 4/2/00;
- 5) Prof. J. Berry dated November 2000;
- 6) Dr. J. Ophoven dated 6/10/00 & 1/12/01;
- 7) Prof. P. Herdson dated 17/1/02.

### **BACKGROUND:**

I am currently employed by the Forensic Science Centre in Adelaide as a Specialist Forensic Pathologist and have been there since May 1999; prior to that I was a Senior Consultant Histopathologist at the Women's and Children's Hospital, with a position of Visiting Consultant Pathologist at the Forensic Science Centre. I hold Clinical Professorships with the Departments of Pathology and Paediatrics at the University of Adelaide. I am also a Consultant Paediatric Forensic Pathologist to the Child Protection Unit at the Women's and Children's Hospital, Adelaide.

I qualified in medicine in Australia in 1978 (University of Tasmania (MBBS) and in Canada in 1982 (LMCC). I hold fellowships in Anatomical Pathology in three countries: Canada (FRCPC), the United Kingdom (FRCPath) and the United States (FCAP). I also hold a fellowship in Family Medicine with the Canadian College of Family Physicians (CCFP). I have a specific interest in sudden infant and childhood death and have published or have in press over 270 papers in peer-reviewed journals, many of which deal with natural, accidental and homicidal causes of sudden infant death. I have also presented or coauthored over 200 papers that have been presented at national and international meetings. I regularly direct or codirect workshops for pathologists, police officers and lawyers on issues in paediatric forensic pathology and have been invited to present such material in Australia, the United States, the United Kingdom, parts of Europe, South Africa, Israel, Canada, Indonesia and Japan. I have coauthored a text on sudden childhood death (the second edition of which is pending), have edited another text on sudden infant death syndrome, and am at present coediting an Encyclopedia of Forensic and Legal Medicine. I have two higher degrees: a Doctor of Medicine (MD) and a Master of Medical Science (MMedSci), both from the University of Adelaide. The theses for these degrees both deal with aspects of sudden death in infants and children. I have performed over 600 autopsies on children, infants and fetuses and appear regularly in court. I also regularly receive paediatric medicolegal cases for opinion from colleagues in Australia and New Zealand, and occasionally the United States. I have enclosed a copy of my full CV for your information.



## OPINION AND COMMENTS:

The current cases are exceedingly complex raising many issues in paediatric forensics that are either not clear cut, or in some cases are not completely understood. Important information from the death scene or from tissue examination was sometimes not available as these examinations were not always performed. For this reason it is often difficult to make definitive statements about possible diagnoses. I will not summarise the medical and social histories of the children as this has already been done in some detail in several of the reports that I have referred to.

It should be stated at the outset that sequential deaths of four young children in the same family are exceedingly rare, are of great concern and must always raise the possibility of homicide or an inherited abnormality. For this reason it is vital at the time of autopsy to check for any evidence of underlying disease. Unfortunately the pathological findings following suffocation in infants and young children are often completely nonspecific<sup>1</sup> and so the family history and social circumstances must also be considered in formulating an autopsy diagnosis. While I found diary entries by Kathleen Folbigg concerning, I would not feel qualified to comment on their psychiatric significance. They require expert assessment.

The most likely causes of multiple infant deaths in a family with no abnormalities clinically or at autopsy are inflicted suffocation or rare inherited disorders of metabolism. However, this refers to cases where no abnormalities are detected, whereas the current cases are quite different in that unequivocal abnormal findings were present. i.e.:

- 1) Patrick had chronic brain damage and epilepsy that was difficult to control;
- 2) Laura had established myocarditis.

These are well recognised and accepted causes of death in children<sup>2,3</sup>. Thus, while I would agree that suffocation cannot be excluded in any of these children, I would also not be able to exclude underlying organic illness as a cause of death in two of the four children (Patrick and Laura). There was also clinical evidence of an organic disorder that may be related to airway compromise and respiratory arrest in a third child<sup>4</sup> (Caleb), and autopsy evidence of airway narrowing in the remaining child (Sarah).

If these children presented as individual isolated deaths in separate families I would have listed the major issues and causes of death as:

- 1) Caleb, aged around 19 days, DOB 1/2/89.

Caleb was allegedly found deceased in his bassinette by his mother on 20/2/89.

Sudden infant death syndrome or SIDS is defined as 'the sudden death of an infant under one year of age which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene and review of the clinical history'<sup>5,6</sup>. A death scene examination by a pathologist or a trained person is therefore required before a diagnosis of SIDS can be made. This is in part to exclude the possibility of accidental asphyxia. As I was unable to find a formal death scene examination for Caleb I would not be able to exclude the possibility of a sleeping accident and so would not be able to make a diagnosis of SIDS. I will not use the term SIDS if there has not been formal assessment and recording of the death scene findings.

Another point of concern is the issue of Caleb having episodic respiratory difficulties with a diagnosis of a floppy larynx (voice box). I would not diagnose SIDS in any infant who has had a history of airway narrowing with breathing difficulties as I could not say that this was not involved in the fatal episode. As no histologic examination was conducted of the larynx at the autopsy (not a routine examination), it is uncertain whether there were any structural abnormalities of cartilage present. Laryngomalacia has been associated with airway obstruction and recurrent apnoea of infancy with some infants requiring resuscitation. Three infants with laryngomalacia in one study had episodes of collapse during hospitalisation observed by medical personnel<sup>4</sup>. Two infants in another family who died suddenly have also been reported with a similar condition (softening of the airway below the larynx, the bronchi) raising the possibility of this being involved in their deaths<sup>7</sup>.

Another significant omission in this case was that the brain did not appear to have been examined histologically.

Subsequent examination of lung sections by Prof. Berry revealed scattered iron containing macrophages (scavenger cells). While this has been claimed to be highly suggestive of an asphyxial episode<sup>8</sup> I have found in a separate study that nearly one in five infants who die of SIDS have this finding, in addition to infants dying of nonasphyxial disorders; i.e. it is not specific for asphyxia<sup>9</sup>.

Given the above points and omissions I would have to label the cause of death as 'undetermined', noting a history of breathing problems involving a floppy larynx (laryngomalacia).

## **2) Patrick, aged around 8 months, DOB – 3/6/90**

Patrick was allegedly found deceased by his mother in his cot on 13/2/91. His medical history included an episode of previous severe brain damage resulting in a seizure disorder.

In isolation, the cause of death would appear to be reasonably clear cut given the history of frequent seizures. Dr. A. Kan in his neuropathology report found changes of scarring, atrophy and inflammation that were in keeping with seizures, previous cardiorespiratory arrest and possibly

treated encephalitis. The changes were however, chronic and relatively nonspecific and could have arisen from a variety of quite different diseases and conditions.

The frequency of sudden death in epilepsy (known as SUDEP) in children is unknown, however in general epileptic populations estimates have ranged from one in 200 to one in 680 patients. The typical case of unexpected death encountered in paediatric autopsy practice is of an epileptic child, often with mental retardation, who is found dead in bed with minimal external or internal findings.

The association of sudden death with sleep is noteworthy and most likely relates to reduction in seizure threshold, with an increase in epileptic discharges. A variety of theories have been proposed to explain the occurrence of sudden death in epilepsy including suffocation from bedding, asphyxia, pulmonary fluid overload (edema) and cardiac arrhythmia. Suffocation and aspiration of food or foreign material are considered unlikely in most cases.

The most popular theory to explain why apparently stable epileptic children are at increased risk of sudden death involves nervous system instability with abnormal cardiac rhythms during seizure activity.

The absence of death scene and autopsy findings of disturbed bedding, urinary or faecal incontinence, bite marks on the tongue and foam in the mouth or trachea, does not mean that an epileptic episode did not occur, as these features have been absent in fatal episodes that have been witnessed. As any type of fit may precede sudden death, not just generalized tonic/clonic convulsions this could explain minimal external findings. Autopsy investigations may show pre-existing chronic brain damage or developmental malformations with loss of nerve cells (neuronal depopulation) and scarring (gliosis) of the hippocampus secondary to past hypoxic episodes, usually with no evidence of an acute lesion<sup>2</sup>.

In Patrick's case the event that provoked the episode of oxygen deprivation to the brain is less clear. However a CT scan from the Newcastle Mater Hospital dated 23/10/90 stated that the image was 'compatible with encephalitis' and a follow-up scan dated 5/11/90 noted 'generalised loss of brain substance' which 'could be related to post inflammatory change'. There was no mention of intracerebral or retinal haemorrhage or diffuse cerebral oedema to suggest possible inflicted injury. Although Dr. M. DeSilva considers that the findings were compatible with shaking, they were relatively nonspecific, without any of the characteristic features of shaking-impact syndrome such as bleeding and tears within the brain or its coverings being identified on admission.

With such an abnormal brain and history, I would have attributed death to epilepsy against a background of possible encephalitis. There was no clinical documentation of features to support a diagnosis of shaking-impact syndrome<sup>10</sup>.

**3) Sarah, aged around 10.5 months, DOB – 14/10/92.**

Sarah was allegedly found dead in her bed by her mother on 30/8/93.

Again I could find no evidence of a death scene examination performed by, or involving, a pathologist.

Prof. Hilton has commented on an unusually congested uvula which produced an 'obstructive element in the airway'. I am not sure of the significance of this finding as it is not something that I have personally seen, however, I do not think that the observation of upper airway narrowing by such an experienced pathologist should be discounted. Sudden and unexpected death is well-recognised in infants with narrowing of the upper airways due to a variety of cysts, tumours and malformations<sup>11</sup>.

Given the above points, with no other abnormal findings present at autopsy, I would have to label the cause of death as 'undetermined', with an autopsy finding of narrowing of the upper airway.

**4) Laura, aged around 19 months, DOB – 7/8/97.**

Laura was allegedly found not breathing by her mother on 1/3/99. She had a recent history of an apparent upper respiratory tract infection.

I would agree with Dr. Cala and Prof. Berry that the slides from the heart demonstrated myocarditis. Myocarditis is a well-known cause of sudden and unexpected death in children of all ages and may be found in infants who present in a similar manner to SIDS. Although some children may have symptoms and signs of heart failure a significant number of cases will have nonspecific clinical features giving no indication of a primary cardiac problem prior to autopsy.

Myocarditis is most commonly caused by microbiological agents, in particular to coxsackie B viruses. Other viruses such as coxsackie A, polio, Echo, influenza A, adeno, cytomegalovirus HIV and parvovirus may also cause myocarditis and death due to cardiac involvement. I could not find any evidence that confirmatory viral studies were performed at the time of autopsy, presumably because the inflammation was not detected until microscopic examination was performed<sup>3</sup>.

Given the finding of extensive myocardial inflammation with no other abnormalities present I would have attributed the death to myocarditis. An identical conclusion would be drawn by 'most pathologists' according to Prof. Berry. This is with the recognition that myocarditis may be found coincidentally at autopsy in children dying of a wide range of other conditions.

The autopsy findings, however, cannot be taken in isolation and with the occurrence of 4 deaths within the same family and police concerns I would list the causes of death as follows:

- 1) Caleb: Undetermined, with laryngomalacia;
- 2) Patrick: Undetermined, cannot exclude epilepsy;
- 3) Sarah: Undetermined, with narrowing of the upper airway;
- 4) Laura: Undetermined, cannot exclude myocarditis.

#### CONCLUSIONS:

In my view the critical issue in the pathology of these cases is the presence of underlying conditions which are known to cause sudden death in young children and babies. I am certainly concerned that there may have been inflicted suffocation but could not state unequivocally that this had occurred, and could not agree that their autopsies have failed to 'identify any known natural disease or disease processes that could explain the sudden deaths', as has been stated. by Dr Ophoven.

Although these cases are discussed in several of the expert reports as SIDS deaths they cannot, by definition, be regarded as such, either on their own or together. Thus, comments on the significance of the presence or absence of SIDS risk factors and use of statistics derived from SIDS deaths are not applicable.

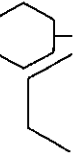
The unusual background of this family with many issues of concern does not negate the fact that potentially significant organic illness was present in these children. Upper airway narrowing, epilepsy and myocarditis may have been coincidental to their deaths, but alternatively may have been causative or contributory; unfortunately this issue cannot be clarified from the autopsy records. Given the information that I have been provided with I simply cannot see how the significance of these conditions can be down-played as potential causes of death, no matter how worrying the circumstances are.



Clinical Professor Roger W. Byard  
BMedSci, MB, BS, MMedSci(Paed), MD,  
CCFP, MACLM, FCAP, FRCPC, FRCPath

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7 March 2019

Professor Caroline Blackwell



**By Email: [c\\_c\\_blackwell@hotmail.com](mailto:c_c_blackwell@hotmail.com)**

Dear Professor Blackwell,

**Re: Kathleen Megan Folbigg**

We advise that we act on behalf of Ms Kathleen Megan Folbigg in relation to the Inquiry ordered into her convictions by the NSW Governor on 22 August 2018.

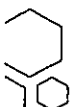
**Report**

We have been instructed to seek your expert opinion report in relation to the cause of death of Caleb Gibson Folbigg, Patrick Allen Folbigg, Sarah Kathleen Folbigg and Laura Elizabeth Folbigg. I draw your attention to the following Court Rules, which have been provided to you:

1. UCPR 31.23 Code of Conduct;
2. UCPR 31.27 Experts' reports; and
3. UCPR Schedule 7 – Expert witness code of conduct.

In order for the report to be of value in the current proceedings, your report must state:-

1. Your name and address.
2. That you comply with Regulation 31.27,
3. That as an expert, you have specialised knowledge based upon your training, study or experience set out in the report,



4. Sets out the opinion that you hold as an expert, and which is wholly or substantially based upon that specialised knowledge, and
5. Set out your reasons for your opinion, and to the extent that you have relied on any scientific study or other literature, refer to that literature by footnote or bibliography.
6. Attach a copy of this letter, the letter of instruction and its attachments to your report;
7. Complete and attach the Certificate - Expert Report;
8. Complete and attach the Expert Certificate, s 177 Evidence Act

### **Attached Documents**

We provided you the following documents:

1. Autopsy reports for Caleb, Patrick, Sarah and Laura Folbigg;
2. Report of Professor Duflou dated 13 February 2019;
3. Report of Professor Horne undated;
4. Report of Professor Hutchinson dated 17 April 2003;
5. Handwritten notes from Allison Colley;
6. Relevant scientific literature.

### **Questions**

We note that we have had three conferences with you where you provided us with information about the report you produced in 2004 and updated in 2006. We understand your report dated 5 March 2019 reproduces the existing report in large part, but extends on it where appropriate.

We undertake to be responsible for your professional fee.

Would you please address your tax invoice as follows:

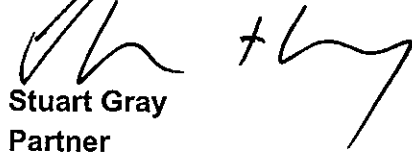
Kathleen Megan Folbigg  
CO/ Cardillo Gray Partners  
PO Box 409  
Newcastle NSW 2300

Should you have any questions or wish to discuss this matter please do not hesitate to contact us on (02) 4910 0677.



Yours Faithfully

**CARDILLO GRAY PARTNERS**

  
Stuart Gray  
Partner

***Encl.:***