EXHIBIT BM

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18th February 2003

Your ref: 02C022602

Peter Krisenthal, Solicitor,
Legal Aid NSW,
Criminal Indictable section,
PO Box 847K,
Haymarket NSW 1238,
5 SYDNEY,
AUSTRALIA

Dear Mr Krisenthal,

Re: Crown –v- Kathleen Megan Folbigg

Further to your letter of 5th February 2003, and recent emails and telephone conversation, I enclose my report. I would like to stress that there are three areas for further consideration:

a) more detailed microbiology interpretation in the case of Sarah and Laura. Briefly, species associated with SIDS and other conditions were present; the problem is are they there as contaminants (as I predict the prosecution will claim) or as pathogens? In the successful Sally Clark appeal in the UK, *Staphylococcus aureus* was found and is present here also.
b) A blood or serum sample for immunology (testing antibody levels). I cannot see any evidence that levels of antibodies were measured.
c) A blood or frozen sample or even a wax block or slide for genotyping (so-called cot-death gene). This is quite separate from the testing carried out previously and I need to stress again that the so-called 'cot-death gene' is actually a variant of a commonly occurring gene. If present it is not proof of SIDS but simply an indicator of increased risk similar to having a mother who smokes.

Yours sincerely,

Dr D.B. Drucker

Instructions

This report has been prepared upon instructions of Peter Krisenthal Solicitor of Legal Aid NSW Criminal Indictable Section, Haymarket NSW, Sydney respect to his client, Kathleen Megan Folbigg. I have inspected documents weighing 18 kilograms which were sent to me:

a) Brief of evidence with a summary
b) Medical records of the four children and their mother
c) Report of Professor Peter Herdson (17 January 2002)
d) Report of Dr Janice Ophoven (6th October 2000)
e) Report of Dr P J Berry (November 2001)
f) Psychological report of Rozalinda Garbutt (11 October 1999)
g) Report of Dr Susan Mitchell Beal (8 December 1999)
h) Report of Dr Bridget Wilcken (14 January 2000).

Professional experience and qualifications

I am Dr David Bernard Drucker. I have been a senior member of staff of the University of Manchester since 1977 and currently am a Reader in Microbiology in the School of Biological Sciences and also Head of the Oral Microbiology section of the University Dental Hospital of Manchester. By way of explanation, in UK universities a Reader is between Senior Lecturer and Professor in our hierarchy. My qualifications are separate BSc degrees in Bacteriology and in Biochemistry and two doctorates, *viz.*, PhD and DSc. I have published nearly 400 research abstracts articles and books and this includes 16 full-length papers on Sudden Infant Death Syndrome (SIDS). This sequence of published research studies on SIDS microbiology makes me one of the most prolific authors in this field in the UK or elsewhere. I have specialised in researching the **microbial causes of SIDS** and more recently some **human genetic factors associated with SIDS**. This led to our discovery of the first published association between a genotype (so-called 'cot-death gene') and SIDS generally. This study was published in a prestigious American journal, Human Immunology. Previously other genes have been described of major importance in a minority of infant deaths resulting from errors of metabolism and cardiac malfunction. I am currently providing scientific expertise in other legal cases involving infant deaths.

Comments on the reports specified in the letter of 5th February.

a) Report of Dr Bridget Wilcken (14th January 2000). I think her comment is fair that 'while the tests..do not definitely exclude the disorders mentioned there is no positive evidence indicating an inherited metabolic disorder affecting amino acid, organic acid or fatty acid oxidation pathways'. However, she has concentrated on MCAD deficiency and I am not sure what testing was done in relation to genes affecting cardiac function. She has not determined which variant of IL-10 gene is present. Other genes linked to SIDS are SCN5A, XL allele variant of 5-HTT promoter gene, CYP2C, and PPL gene which are linked to death in relatively rare cases.

b) Report of Dr Susan Beal (8th December 1999). I am not qualified to comment on the 'clues to suspecting filicide' she lists except that I was surprised to read that either 'reluctance to be visited...OR..obsessive involvement' counts as a clue. Is this backed up by a peer-reviewed research paper I wonder? Her comment that 'three (deaths) is murder until proved otherwise' has been held by a very famous paediatrician in the UK. Professional statisticians here have attacked that argument and so have the media, very extensively. I am not a professional (*ie* PhD in mathematics) statistician myself but can point you in the direction of scientists who are highly qualified to comment. She says that there have never been three or more SIDS in one family anywhere in the world. Only yesterday, someone who had seen me on TV wrote to say she and her husband had lost **four babies as SIDS cases**.

c) Report of Rozalinda Garbutt (11 October 1999). I am not qualified to comment on this report but am surprised she has not a single peer-reviewed publication listed by the MEDLINE database.

d) Report of Dr PJ Berry (November 2000). This expert admits he would have called each case SIDS initially although he is very concerned about four deaths which are..'unprecedented..I know of no substantiated claims in the literature. Nevertheless it is important to explore this possibility'. He is absolutely correct and please note my comment under 'b'.

e) Report of Dr Janice Ophoven (6th October 2000). The report expresses a strong opinion that the SIDS process is not a hereditary problem and the statistical likelihood that four children could die from SIDS is in excess of 1 in a trillion'. If we look at some facts instead of opinion, firstly SIDS is not a process, it is not an anything because the term surely refers to deaths of all kinds which fit the definition of SIDS? Secondly if SIDS babies differ genetically (IL-10 gene) from controls to a statistically significant extent, how can SIDS not be to some extent an hereditary problem? If four deaths are impossible, how come I received the letter already alluded to?

f) Report of Professor P. Herdson (17th January 2002). I note that he says 'the pattern of the mother's actions and reactions over the ten year period is not typical of so-called Munchausen Syndrome by proxy'.

Scientific Basis for SIDS (overview)

Below I explain a scientific explanation for SIDS based upon years of research by myself, and others, around the World. Obviously, SIDS is not a single disease and also some infants die for unnatural reasons. Those who die from an infective cause are believed to die because they are unable to defend themselves against toxins (poisonous products) of commonly occurring bacteria. This is because they lack immunity (antibody) and may also be genetically pre-disposed to respond less effectively to challenge by bacteria.

Scientific Basis for SIDS (microbiology)

Although it is frequently claimed that SIDS remains a mystery, it should not be. SIDS is obviously a collection of different causes of death that remain when well established known causes have been excluded. Some SIDS deaths, possibly a majority may involve commonly occurring bacteria which are simply in the wrong place at the wrong time. The babies' antibodies are also important (see below). A number of other studies we have carried out suggest to us that bacteria are important in many SIDS cases. We have noted that:

a) SIDS babies have more bacterial species in their nasopharynx than equivalent healthy babies.
b) In germ free rats which lack immunity, specific bacteria in combination produce sudden unexpected death, like SIDS. In the absence of specific bacteria, deaths do not occur. The bacteria are *e.g. E. coli* and *Staphylococcus aureus*. Both species are found in us naturally but virulent strains in the wrong place at the wrong time can cause deaths even in adults.
c) In chick eggs, toxins (poisonous substances) from bacteria, without live bacteria present produce similar results to the rat experiments. Combinations of toxins are much more lethal than a single toxin on its own.
d) Because smoking is a risk factor in SIDS (makes SIDS more likely), we would predict that toxic products of smoking (nicotine) should enhance the lethality of bacterial toxins. In experiments we find this. In fact bacterial toxins can be over a thousand times more toxic in presence of low levels of nicotine.
e) Serum from SIDS babies is lethally toxic in our test system.Its toxicity can be neutralised by preformed antibodies. This suggests that around the time of death there must be dangerously high levels of toxins in a SIDS baby's circulation.

Some would argue that the toxin moved into the circulation after death, so we have shown this is not so using animals injected with low levels of endotoxin from *E. coli* then humanely killed, stored up to a week and analysed at various times after death. Repeat analysis of a SIDS baby shows that the bacteria of interest actually decrease with time; they do not increase.

We know that sleeping position of a baby can alter the risk of SIDS. Our explanation is that sleeping position alters levels and quantities of bacteria present in the nasal passages. This is because nasal fluid cannot drain equally well in all sleeping positions.

Recent research has found that adults who have an Upper Respiratory Tract Infection and sleep on their fronts have a similar naso-pharyngeal flora to that seen in SIDS. Clearly, the flora has not required a postmortem change to occur!

Our research over the years has particularly implicated two specific groups of bacteria in SIDS, *viz.*coliforms such as *Escherichia coli* (*E. coli*) and *Staphylococcus aureus* (also called *Staph. aureus* or *S. aureus*). I should point out that *E. coli* is grouped with closely related species sometimes loosely termed coliforms. The term coliform is used when full identification has not been carried out or when it has but several related species are referred to. In adults, *S. aureus* can prove fatal and includes strains which

produce toxin TSST-1 which is responsible for Toxic Shock Syndrome. *E. coli* and related species produce highly poisonous endotoxin which again can kill adults, especially if debilitated following gut surgery or in intensive care. Both groups of organisms are common in healthy individuals and it is when they are in the wrong place and gain the upper hand over our natural defences that we have a problem.

Scientific basis for SIDS (immunology)

When a baby is born, it has antibodies from its mother; it cannot yet make all its own antibodies (it is not *immunologically competent*). After a period of time, a baby does become able to make its own antibodies in response to bacteria it meets. We would predict that a baby is especially at risk of SIDS around 2-3 months when its maternal antibody will be depleted but its own antibody production is not fully developed. We do in fact see a peak in deaths at this time.

Scientific Basis for SIDS (genetic)

When bacteria make us ill, microbiologists tend to contemplate the bacteria. However, another factor is the host defence and although this is better in healthy, immunised individuals, the hidden factor here is genetic. We know that the way the body responds to infection involves chemicals called cytokines which the body makes. Some of our genes code for the production of cytokines. Research by Professor Hutchinson of the Immunology laboratories in Manchester University School of Biological Sciences has revolutionised our ability to analyse the structure of these genes.

Important points to note are:

a) We all have the so-called 'cot-death gene' However it exists in chemically different forms or genotypes.

b) SIDS babies are much more likely than the general population to have a particular form of the cytokine IL-10 gene and this difference is statistically highly significant.

c) Such differences are important when **groups** of babies (SIDS versus non SIDS) are compared but they cannot yet predict a SIDS death for any given **individual** baby.

This is akin to seat belts in cars. We cannot say that people who do not wear them will automatically die yet we wear seat belts because not doing so increases our risk of death. Our work on genetics is quite different from work on genes (*vide supra*) which can affect metabolism or heart function in a tiny minority of cases of sudden infant death.

Relevance of microbiological knowledge to the Kathleen Megan Folbigg case

I shall consider the data per child:

a) Caleb Gibson Folbigg. I cannot find evidence of microbiological investigation.

b) Patrick Folbigg. Postmortem examination reports included analysis of blood cultures. These contained mixed cocci and bacilli identified as *Escherichia coli, Enterococcus faecalis* and *Enterococcus avium* with the comment that they 'probably reflect contamination'. Lung tissue was negative for bacteria, viruses and mycoplasma. Earlier when meningitis had been suspected samples of blood and CSF had been analysed (8 November 1990). CSF was sterile but blood contained coagulase –ve staphylococci of '? Sig' (questionable significance). In respect of the latter finding, coagulase –ve staphylococci 13 years ago were largely regarded as having no clinical significance. I think that view is outmoded but the organism was found three months **before** death. In relation to the PM findings, very few samples were taken. However the bacteria found in blood are interesting. All of them could have come from the gut flora. The question is did this arise after death (by contamination) or before death. Certainly one of the major species associated with SIDS was present, *ie E.coli* although the other species found are not characteristic of SIDS but of the gut flora. Other bacteriology done much earlier on eye swabs and mid-stream urine does not appear relevant to the death. However other experts seem to have found evidence of an epileptic fit and prior encephalitis. I note that the latter possibility impresses both professor Busuttil and professor Byard as the likely explanation for death, rather than SIDS.

c) Laura. This child was the subject of more intense microbiological investigation. The CSF yielded no growth. The rectal swab yielded 'normal flora' and no *Salmonella, Shigella* or *Campylobacter*. The latter bacteria are associated with very serious gut infections but were not detected here of course. Lung revealed 'profuse coliforms' which were dismissed as postmortem contaminants which is interesting when one considers that cultures from Patrick's lungs were negative! The spleen yielded an interesting collection of bacteria including moderate coliforms, profuse alpha haemolytic streptococci (2 colonial types) and moderate *Staph. aureus* . Dr Cala's report states that ' the diagnosis of SIDS should be made sparingly after the age of 12 months'. By some definitions, a death after 12 months would never be called SIDS. However there is no reason why in practice it might not be, though rarely observed. Coliforms and *Staph. aureus* are both associated with SIDS. In a recent successful appeal case in the UK, the bacterium to which death was ascribed and which was isolated widely from the body, was *Staph. aureus*. I do not consider that it caused a major infection in this case. The term 'alpha haemolytic streptococci' is very vague and covers many species. Some of these are pretty harmless and others not. The poor identifications make it difficult to comment further.

d) Sarah. Samples of contents of large intestine and small intestine were sent for microbiological analysis and contained various bacterial species which is not surprising really. Other samples were of spleen and lung. The spleen sample yielded coliforms of three colonial types. The lung contained profuse

coliforms, alpha haemolytic streptococci (but we do not know which species) and 'scanty *Staph. aureus'*. Again we do not know which species of *Streptococcus* were found; they may have been pathogenic or not. The presence of coliforms together with *Staph. aureus* together is interesting because both have been associated with SIDS and together their toxins act synergistically having a far greater effect than separate toxins would. This infant is the closest to SIDS of all four. I also note that antibiotics had been given for a 'bad cold'. There is a strongly held opinion that, in SIDS, bacterial colonisation of upper airways may be assisted by prior tissue damage cause by viral Upper Respiratory Tract Infection .

Relevance of genetic findings to deaths

In the case of these children, genetic analysis has been aimed at known major defects which could clearly explain death, *eg* MCAD deficiency. Known defects were not found, although the expert comments that other possibilities still exist of a genetic problem. At the time the first three babies died our research on the IL-10 gene had not been published. Unlike MCAD, the 'cot-death gene' is not a sentence of death. It is merely a naturally occurring variant of a gene we all have. However the 'wrong' variant is associated with increased risk of SIDS. (See Appendix 1) on genetics and SIDS

Relevance of immunological analyses to deaths

The samples sent for antibody analysis were specifically aimed to detect particular viral infections. There has been no attempt to measure levels of classes of immunoglobulins (antibodies) present. If we say that some cases of SIDS result from a child lacking appropriate immunity meeting commonly found toxigenic (poison-making) bacteria, then knowledge of the antibody levels is important.

Further Testing.

a) Samples for genetic testing. Blood samples are preferred. However other frozen samples are also satisfactory. We can also usually do the analyses on dried material from wax blocks or even slides. Dried material could be shipped to us for testing without being kept frozen using dry ice.

b) Samples of serum or blood for measuring antibody levels. Although the variant of the IL-10 gene is important, so are an infant's antibody levels. These have been mentioned above in relation to their role in protection against harmful effects of bacterial pathogens (disease-causing microorganisms). It is possible to measure whole classes of antibody in serum or blood from infants.

Summary

As a researcher in the field of SIDS microbiology, I would say that on microbiological evidence alone:

i) Caleb: no suitable data are available for a microbiological opinion.

ii) Patrick: Little evidence of SIDS associated bacteria.

iii) Laura: Some evidence of SIDS associated bacteria.

iv) Sarah: Species associated with SIDS present and after an URTI. It is entirely possible that Sarah died as a SIDS case.

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Appendix to Drucker-Report.

Appendix 1. Genetics and SIDS

a) 'A small percentage of apparent SIDS victims may have a metabolic disorder, Medium-chain acyl dehydrogenase (MCAD) deficiency' in which fatty acids found eg in fats cannot be correctly metabolised, resulting in a sudden unexpected death. 'A single mutation in MCAD, termed G-985, accounts for approximately 90% of MCAD deficiency mutations'. However it is not strongly associated with SIDS[1] This finding in New York has been confirmed by studies in Scotland[2] and France[3]. It is always possible that a death is due to MCAD deficiency but not to this specific mutation.
b) A study in New York found a SIDS victim was homozygous for 'the nonsense mutation at codon 49 of the myophosphorylase (PPL) gene most commonly associated with typical McArdle's disease and suggested that 'amomg children presenting as SIDS there may be some cases associated with myophosphorylase activity[4].
c) Upregulation of CYP2C gene transcription has been linked to SIDS[5].
d) The XL allele variant of the 5-HTT gene has been linked to SIDS. The gene's product is serotonin (5-HT) which operates in the brain centre for respiration[6].
e) Fatal arrhythmias from occult long QT syndrome may cause some cases of SIDS. Patients with long QT syndrome with sodium channel gene (SCN5A) defects have an increased frequency of cardiac events during sleep. 'SCN5A..(is) the leading candidate ion channel gene for SIDS'[7].

[1]Miller ME, Brooks, JG, Forbes N, Insel R (1992) Frequency of medium- chain acyl-dehydrogenase deficiency G-985 mutation in sudden infant death syndrome. Pediatr Res 31: 305-307.

[2]Dundar M, Lanyon WG, Connor JM (1993) Scottish frequency of the common G985 mutation in the medium-chain acyl-dehydrogenase (MCAD) gene and the role of MCAD deficiency in sudden infant death syndrome (SIDS). J Inherit Metab Dis 16: 991-993.

[3]Lecoq I, Mallet E, Bonte JB, Laroche D, Travert G (1995) "Screening of A985 to G mutation of MCAD gene in Normandy. Evaluation of the role of MCAD deficiency in sudden infant death. C R Sceances Soc Biol Fil 189: 295-301.

[4]el-Schahawi M, Bruno C, Tsujino S, Sarrazin AM, Shansske S, LeRoux MG, DiMauro S (1997) Sudden infant death syndrome (SIDS) in a family with myophosphorylase deficiency. Neuromuscul Disord 7: 81-83.

[5]Treluyer JM, Benech H, Colin I, Pruvost A, Cheron G, Cresteil T (2000) Ontogenesis of CYP2C-dependent arachidonic acid metabolism in the human liver: relationship with sudden infant death syndrome. Pediatr Res 47: 677-683.

[6]Narita N, Narita M, Takashima S, Nakayama M, Nagai T, Okada N (2001) Serotonin transporter gene variation is a risk factor for sudden infant death syndrome in the Japanese population Pediatrics 107: 690-692.

[7]Ackerman MJ, Siu BL, Sturner WQ, Tester DJ, Valdivia CR, Makielski JC, Towbin JA (2001) Postmortem molecular analysis of SCN5A defects in sudden infant death syndrome. JAMA 14: 2264-2269.

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5 February 2003

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MANCHESTER M15 6FH
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Dear Dr Druker,

Re: CROWN –V – KATHLEEN MEGAN FOLBIGG
NSW SUPREME COURT
CHARGE: MURDER X 4
LISTED FOR TRIAL: 10/02/03

I refer to the above matter and to our recent telephone conversations in which you agreed to review the documentation in this matter and to prepare a report outlining your opinion.

Ms Kathleen Folbigg is charged at:

- On 29 February 1989 at Mayfield in the State of New South Wales did murder Caleb Gibson Folbigg (then aged 19 days).
- On 13 February 1991 at Mayfield in the State of New South Wales did murder Patrick Folbigg (then aged 8 months).
- On 30 October 1993 at Thornton in the State of New South Wales did murder Sarah Kathleen Folbigg (then aged 10 months).
- On 1 March 1999 at Singleton in the State of New South Wales did murder Laura Elizabeth Folbigg (then aged 18 months).

Attached with this letter is the entire Brief of Evidence in the matter as well as the medical records of each of the children and also of the mother. For your benefit I also attach to this letter a summary of the brief which has been prepared by myself. Although this has been carefully prepared, I do not assert that it is a perfect summary of the brief.

As discussed with you by phone, Mrs. Folbigg has never asserted that her children have died from SIDS. She remains unclear as to why her children have died and has always sought answers in this regard. You will note from the brief that she actively sought out medical opinion as to genetic difficulties and the like before continuing to have further children. It appears from my enquiries that there may be natural causes which could be responsible for the death of a number of these children. For Example:

- Caleb ? larynx problem
- Patrick ? epilepsy
- Laura ? heart disease

The Crown have over a period of time obtained a number of medical reports which indicate that these deaths were as a result of homicide. I would particularly request that you consider and comment (where appropriate) upon the following reports:

- Report of Professor Peter Herdson (17 January 2002)
- Report of Dr. Janice Ophoven
- Report of 6 October 2000 and also 1 December 2001
- Report of Dr. P.J. Berry (November 2001)
- Psychological report of Rozalinda Garbutt dated 11 October 1999
- Report of Dr. Susan Mitchell Beal dated 8 December 1999
- Report of Dr. Bridget Wilcken dated 14 January 2000

After having considered all of the attached information, I would request that you prepare a report encompassing the following areas:

- **Your opinion as to any genetic link with "cot deaths" and an explanation for this.**
- **Your opinion as to whether that research had any application in the present matter.**
- **Your opinion as to the cause of death of Caleb Folbigg who died on the 20 February 1989.**
- **In relation to the death of Caleb Folbigg is it possible that he died from causes other than intentional murder at his mother's hand.**

- Your opinion as to the cause of death of Patrick Folbigg who died on 13 February 1991. In your opinion is it possible that he died from a cause other than intentional murder at his mother's hand.

- Your opinion as to the cause of death of Sarah Folbigg who died on 30 August 1993. In relation to the death of Sarah Folbigg, is it possible that she died from causes other than intentional murder at her mother's hand.

- Your opinion as to the cause of death of Laura Folbigg who died on the 1 March 1993. In relation to the death of Laura Folbigg, is it possible that she died from causes other than intentional murder at her mother's hand.

- Any other matter that you consider relevant.

For your information I also attached copies of report prepared by Professor Rodger Byard and also Professor Anthony Buruttil. Both these doctors are forensic pathologist and have given their opinions along those lines. They were both giving evidence for the defence in this matter.

Could you please attach to your report a copy of your curriculum vitae. As noted the trial begins in Sydney Australia on 10 February 2003. Should you be required to give evidence in the matter the Legal Aid Commission may need to fly you to Sydney in order to give your evidence.

I understand from our telephone conversation that you calculate your bill on an hourly basis. Now that you have seen the size of the brief, I would be grateful if you could advise me of an estimate of your account. For ease of communication, could I suggest that we initially communicate via email and my email address is peter.krisenthal@legalaid.nsw.gov.au.

Many thanks for your assistance in this complex and difficult matter.

Yours faithfully,

PETER KRISENTHAL
SOLICITOR

5/2/03. This letter & brief was forwarded to Dr Drucker by emergency courier / door to door. I authorised 24 hrs. Cost was $650. This gave the Dr sufficient to do [illegible] & the proximity of the trial & the need for Zahra to know whether he should opine on it.

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