# EXHIBIT AU

29 March 2019

Stuart Gray,
Partner,
CARDILLO GRAY PARTNERS
**via email**

**Re: Folbigg Inquiry**

My full name is Paul Nathan Goldwater. I reside at 7 Childers St North Adelaide, SA 5006. I graduated from the University of London in 1973. I have been a registered medical Practitioner in South Australia from 1986. I am also registered under the Australian health practitioners Regulation Agency since its inception. I have been a Fellow of the Royal Australasian College of Physicians since 1979 and a Fellow of the Royal College of Pathologists of Australasia since 1980. I have been a specialist in Infectious Diseases since 1979 and a specialist Clinical Microbiologist since 1980. I have been employed as a Senior Consultant at the Women's & Children's Hospital/The Children Youth & Women's Health Service, Adelaide since 1986. Through an act of parliament, as of the 1 July 2008 my employment transferred to SA Pathology. I have been Clinical Senior Lecturer in the University of Adelaide Department of Paediatrics since 1986. In January 2008 I was appointed Clinical Associate Professor in Infectious Diseases in the School of Paediatrics and Reproductive Health, Discipline of Paediatrics. In January 2011 I was appointed Professor. I am an affiliate of the Adelaide Medical School. My *curriculum vitae* accompanies this report. I have over 30 years of experience in my specialty of Clinical Infectious Diseases and have broad experience in the management of all manner of infections especially of the fetus and mother, children and adolescents. I set up and ran the Infectious Diseases Clinic at the WCH for many years. I was a chief investigator in a study of bacterial meningitis of children in 1987-2004. Over the last 30 years I have been researching the role of infection in Sudden Infant Death Syndrome and have over 30 papers in peer-reviewed journals resulting from this research. I have approximately 165 peer-reviewed papers published *in toto* covering a wide variety of infectious diseases and published one book on HIV/AIDS. I have had a special research interest in the pathogenesis of cerebral palsy and was a principal investigator with the South Australian Cerebral Palsy Research Group which conducted ground-breaking investigation of the genetic and infective causes of cerebral palsy. I have contributed to the writing and updating of the South Australian Perinatal Practice Guidelines. On the 30th of June 2015 I retired from clinical practice.

I have specialised knowledge based on my training, study or experience as specified above. My report is an opinion that I hold, and which is wholly or substantially based on that knowledge.

I confirm that

a) factual matters stated in this report are, as far as I know, true; and
b) I have made all enquires considered appropriate; and
c) opinions stated in the report are genuinely held by me; and
d) I have read the expert Code of Conduct pursuant to schedule 7 of the Uniform Civil Procedure Rules 2005 and I agree to be bound by its terms.
e) I understand my duty to the Court and I have complied with this duty.

In the preparation of my report I have been provided with the following documents

1. Report of Professor Blackwell dated 5 March 2019 and Annexures A-J;
2. Supplementary report of Professor Blackwell dated 13 March 2019;
3. Report of Professor Blackwell for the Inquiry undated;
4. Report of Professor Clancy dated 13 March 2019;
5. Supplementary report of Professor Clancy dated 17 *M*arch 2019;
6. Inquiry transcript Day 5 - 22 March 2019.

I have been asked to provide a peer review of the opinions offered by Professors Blackwell and Clancy.

**Report of Professor Blackwell** dated 5 March 2019 and **Annexures A-J;**
The 5 March 2019 report provides an accurate overview with appropriate references to the literature on SIDS/SUDI.

**Annexure A** is an additional report by Professor Blackwell that also meets peer review standards for accuracy and balance. [redacted]

One important omission from her documentation on the relationship between infection and SIDS is the much ignored fact that infection is the key factor underlying the epidemiological risk factor of prone sleep position. This was shown in the Tasmanian SIDS Study [1]. Little, if any attention was given to possible mechanisms that could explain the prone position risk factor other than mechanistic ones invoking some sought of asphyxiation or a purported link to brainstem homeostatic function. In addition to the mechanism proposed by Professor Blackwell, other plausible explanations (e.g. ingestion or inhalation of infective agents from the sleeping surface) have been proposed [2]. Published evidence that babies are at increased risk of SIDS if they sleep on a sofa [3] or sleep on a used (second-hand) mattress [4,5] or in a parental bed [6] seem to support this idea, given the common finding of *Staphylococcus aureus* and *Escherichia coli* being associated with SIDS [7]. Both of these bacteria carry lethal toxins and are commonly found on the aforementioned contaminated sleeping surfaces involved. The key to the answer was overlooked by mainstream researchers. These paths of inquiry have provided SIDS researchers with unfruitful outcomes and could explain why, after decades of research, there still is no answer.

The key research that would have been helpful in understanding a more plausible role of prone sleep position as a risk factor for SIDS was contained in the data from the Tasmanian SIDS study [1]. The Nordic SIDS epidemiological study [8] provided supportive evidence. These studies were regrettably overlooked or ignored by mainstream researchers. The Tasmanian study clearly indicated that risk of SIDS occurred in the prone position mainly when there was an accompanying illness defined as nasal congestion, cough, chest noises, fever, episodes of vomiting or diarrhoea on the day of death or previous day. These signs not unreasonably reflected either a respiratory or gastrointestinal infection. *"The prone position increased the risk of SIDS more than 10-fold among ill infants, but it was associated with only a slight increase in risk among apparently well infants. This difference in risk was significant (P = 0.02)."* [1] The Nordic study,[8] whilst primarily examining time of death, also showed increased risk of SIDS for prone-plus-infection (OR=29) but

also showed that a cold in the last 24 hours increased the risk of SIDS in supine/side sleepers. Smoke exposure, a known potentiator of acquisition and severity of viral infection was also shown to be a significant contributor to SIDS pathogenesis in both of the abovementioned studies and numerous other studies [9,10]

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The Court may wish to explore whether the Folbigg children (excepting Sarah) were found prone as this could have a bearing on their cause of death.

**Annexure B** is a price list of commercially available *Staphylococcus aureus* toxins and antibodies. These are pertinent to costs of testing mentioned in Professor Blackwell's report.

**Annexure C** contains testimony from Professor Herdson, Dr Beal, and Professor Berry.

[redacted]

None of the witnesses (including Professor Blackwell) have sought appropriate explanation in regard to the swollen uvula of baby Sarah that could be helpful to the Court; the child clearly had uvulitis. This is caused by *Haemophilus influenzae* type b or Group A streptococci or rarely anaerobic bacteria. In such infection, life threatening bacteraemia can occur [11].

[redacted]

**Annexure D** is a copy of the expert Code of Conduct pursuant to schedule 7 of the Uniform Civil Procedure Rules 2005.

**Annexure E** outlines Professor Blackwell's experience and qualifications. It is an accurate summary.

**Annexure F** is the report of Dr Janice Ophoven. [redacted]

**Annexure G** is a record of the Genetics Unit recording the histories of the four Folbigg babies. Please see my comment in regard to Professor Clancy's report.

**Annexure H** is a letter from the DPP to Peter Krisenthal, Legal Aid regarding testing for immunoglobulins and incorporates a comment from Dr Charles Hii, Immunologist. I have no comment.

**Annexure I** is a Report dated 29 April 2003 from Professor Berry [redacted]

**Annexure J** is a report by Professor Byard. His opinion is fair and balanced and he found that the conditions found at autopsy cannot be played done as causes of death. His report [redacted] did not mention the microbiological findings which I think have a major bearing on these cases in terms of being contributory or causal in the deaths of these babies..

Turning to the Inquiry testimony of Professor Blackwell and Professor Clancy (Transcript of Proceedings). Page 320: The issue of bacterial contamination and the proposition that the opinion on this of four forensic pathologists is more valuable than published research (and microbiologists) is an alarming distraction. In my experience, as a clinician and as a SIDS researcher, a situation has arisen over years where unsubstantiated ideas have become dogma. Professor Blackwell is quite right when she argues that when *E. coli* or *Staphylococcus aureus* are found in normally sterile sites (either as pure or mixed culture) this represents, on balance of probability, a true bacteraemia and should never be discarded or written off as "contamination." Similarly, in the clinical setting with living patients, if *S. aureus* is isolated in mixed culture this definitely represents a significant bacteraemia. I say this as a Clinical Microbiologist/Infectious Diseases Physician of over 30 years experience. If the four pathologists were to be asked for their opinion on the latter scenario, they would, on balance of probability, say the *S. aureus* was a contaminant. This is a serious mistake.

Professor Blackwell's statement on page 338 para 40 needs clarification. It should be stated that *Staphylococcus aureus* is a very rare contaminant and is rarely ever picked up from the

skin. Its isolation should ring alarm bells and be taken extremely seriously. The same seriousness should apply when it is isolated from cases of SUDI.

On page 343 para 5 The name "Kanu(?)" should be replaced with "Kinney."

There is mention of "Asian children" in the discussion on the cortisol switch, however, its significance appeared to have been glossed over or lost. The point of mentioning Asian children by Professor Blackwell relates to the much lower risk of SIDS in this ethnic group and this could relate to the later cortisol switch in Asians.

**Professor Clancy's report**

Prof. Clancy states…

15. I have looked at the post mortem report of Caleb Folbigg and note within the lungs there is a small amount of eosinophillic exudate in the alveoli. This means that there has been an inflammatory process in the wall of the small airways and that as a result there has been an exudate (i.e. there is protein present) as a response to probable bacteria stimulation and/or there is the 'leaky mucosa' as found in SIDS. Eosinophillic means that the eosin in the stain used has bound to protein.

Strictly speaking eosinophilic exudate means the presence of inflammatory cells which have taken up eosin from the Haematoxylin/Eosin stain. These cells reflect the existence of either a viral, bacterial, chlamydial or *Pneumocystis jerovecii* pneumonia. Pertussis typically induces an eosinophilic response. Such infections could induce an eosinophil response based on which cytokine/chemokine they happen to activate in a genetically predisposed host, e.g the production of the chemokine eotaxin (CCL11) [12-14].

I agree generally with Professor Clancy's conclusions, [redacted]

Professor Clancy comments on the microbiological findings of Sarah, and Laura in his Supplementary Report. I generally concur with his statements, however, I cannot be certain of the interpretation of the findings in the spleen culture from Sarah in which coliforms were isolated. Such a finding cannot be totally discounted as contamination given this is a normally sterile site. The isolation of *Staphylococcus aureus* from the lung is highly significant in my opinion and on balance of probability, would have played a role in her death. Laura probably died as a result of myocarditis but *Staphylococcus aureus* was isolated from her spleen; this could have played a role in her death as could the coliforms isolated from her lungs.

I have no further comment to make in regard to Professor Clancy's testimony other than to acknowledge that he is well qualified to provide testimony to the Court.

I confirm that Professor Blackwell is well qualified to give testimony as an expert SIDS researcher and microbiologist/immunologist.

**Summary**

I am of the opinion that there is cogent and persuasive evidence that the Folbigg children died of natural causes. This conclusion is upheld by historical, pathological and microbiological evidence. The opinions of Professor Herdson, Professor Berry, Dr

Ophoven and Dr Beal are, [redacted] in my view, flawed. [redacted]

I trust this report is helpful to the Inquiry and I am willing to make myself available to give evidence if required.

Yours sincerely,

Paul N. Goldwater

**References**


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