

CHAPTER 6: RECURRENCE

107. The doubt or question that gave rise to this Inquiry was in relation to evidence adduced at Ms Folbigg's trial as to the incidence of reported deaths of three or more infants in the same family attributed to unidentified natural causes. The direction for the Inquiry requires a particular focus on the incidence of reported deaths of three or more infants in the same family attributed to unidentified natural causes (generally referred to as SIDS).¹⁶⁸

Recurrence of SIDS/unexplained deaths in literature

108. The Inquiry conducted research, and invited interested parties and relevant experts, to identify literature on instances of recurrence of SIDS or other sudden unexplained infant deaths. The Inquiry received considerable assistance from those representing Ms Folbigg, who provided an extensive collection of literature relevant to this issue and other issues.
109. The Inquiry has reviewed the literature which was made available on these topics. What follows is a brief overview.

Prior to 2003

110. In 1984, drawing on Norwegian data, the SIDS risk was thought to be 1.3 per 1,000 live births, however, the recurrence risk for a second sibling after a SIDS death was 5.6/1,000 and for subsequent siblings, 4.8/1,000.¹⁶⁹
111. In 1986 Emery reported on 12 families with two or more "cot deaths".¹⁷⁰ In two families, the deaths were completely unexplained. One family in which three children died (two aged one month, the third aged 12 days) seemed to have some form of pulmonary dysplasia. In another, four babies died, with two different fathers. The first was ascribed to gastroenteritis, the second two to cot death, and the fourth to drowning. Emery considered that filicide was a likely differential diagnosis. He considered the risk of cot death in an ordinary baby to be about one in 500, and of a second cot death about three times as much.

¹⁶⁸ Exhibit A, the Direction.

¹⁶⁹ Lorentz M Irgens, Rolv Skjaerven and Donald R Peterson, 'Prospective Assessment of Recurrence Risk in Sudden Infant Death Syndrome Siblings' (1984) 104(3) *Journal of Pediatrics* 349, abstract only; See also Donald R Peterson, Eugene E Sabotta and Janet R Daling, 'Infant Mortality among Subsequent Siblings of Infants who Died of Sudden Infant Death Syndrome' (1986) 108(6) *Journal of Pediatrics* 911, abstract only.

¹⁷⁰ John L Emery, 'Families in which Two or More Cot Deaths have Occurred' (1986) 327 *Lancet* 313.

112. In 1986 Diamond reported on a single case of five consecutive siblings whose deaths were ascribed to SIDS.¹⁷¹ The author suggested the subgroup may be aetiologically distinct from the general population. Research by Oren, Kelly and Shannon in 1987 included two families with four SIDS victims, and two with three SIDS victims.¹⁷² The risk of SIDS was thought to increase 3.6 to tenfold in subsequent siblings of SIDS victims, with the role of genetic or environmental factors being debated.¹⁷³ A reference was made to a report by Rosen et al in 1983, of a family with three previous SIDS victims.¹⁷⁴
113. Dr Beal co-authored a 1988 paper reporting a study which found an incidence of 21.2 per 1,000 in siblings (or 10.1 times the expected rate). The minimum incidence was 11 per 1,000, or 5.2 times the general population risk.¹⁷⁵ (It was later suggested by Bacon and colleagues that this may have been inflated.)¹⁷⁶ Beal and Blundell noted that:
- [f]or most families (92%) in which an infant died from SIDS the risk of recurrence is small (less than twice the expected risk). We have identified a small subgroup (8%) with a significantly increased risk of recurrence.*¹⁷⁷
114. In a 1990 publication, the authors found five recurrences of SIDS among 385 siblings (13 per 1,000 live births) and the risk of SIDS for next and subsequent siblings to be five to six times that for the population.¹⁷⁸ In 1993, a study of families which had experienced two or more unexpected infant deaths in England and Wales found, from 57 deaths, 24 families with two deaths and three with three deaths. However, only five of the 57 were considered to be true idiopathic SIDS. The authors observed that deaths in infants are often of multifactorial cause

¹⁷¹ Eugene F Diamond, 'Sudden Infant Death in Five Consecutive Siblings' (1986) 170(1) *Illinois Medical Journal* 33.

¹⁷² Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355.

¹⁷³ Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355, 355.

¹⁷⁴ Carol Lynn Rosen et al, 'Two Siblings with Recurrent Cardiorespiratory Arrest: Munchausen Syndrome by Proxy or Child Abuse?' (1983) 71(5) *Pediatrics* 715.

¹⁷⁵ S M Beal and H K Blundell, 'Recurrence Incidence of Sudden Infant Death Syndrome' (1988) 63 *Archives of Disease in Childhood* 924, 929.

¹⁷⁶ C J Bacon et al, 'How Common is Repeat Sudden Infant Death Syndrome?' (2008) 93 *Archives of Disease in Childhood* 323, 324.

¹⁷⁷ S M Beal and H K Blundell, 'Recurrence Incidence of Sudden Infant Death Syndrome' (1988) 63 *Archives of Disease in Childhood* 924, 924.

¹⁷⁸ Warren G Guntheroth, Rüdiger Lohmann and Phillip S Spiers, 'Risk of Sudden Infant Death Syndrome in Subsequent Siblings' (1990) 116(4) *Journal of Pediatrics* 520, abstract only.

and suggested that the chance of recurrence was very small, probably no greater than the general occurrence of such deaths.¹⁷⁹

115. Another Norwegian study in 1996 found that the SIDS rate for second babies was nearly six times higher if the first baby had died of SIDS.¹⁸⁰ However, the autopsy rate was poor.¹⁸¹
116. A report on 5,000 babies in the Care of Next Infant program (“the CONI program”) in England and Wales stated that 44 died, 35 unexpectedly. Eight (1.6/1,000) were finally categorised as true cot deaths.¹⁸² 104 of the parents in the CONI program had experienced two previous baby deaths, and four had experienced three previous deaths.¹⁸³ Of those four, only one experienced three SIDS deaths.

After 2003

117. In 2004 it was reported that in 2000 in the United States, SIDS caused 2,523 (0.62 per 1,000) deaths, with higher incidence of SIDS among infants born to mothers who smoked.¹⁸⁴ Also in 2004, Hill opined that there was no doubt that the occurrence of two or more SIDS in the same family will be a rare event.¹⁸⁵ It was “intuitively clear” that a subsequent infant will be at increased risk, because many genetic and environmental factors will be the same.¹⁸⁶ Hill estimated the risk of SIDS was between five and 10 times greater for a second sibling.¹⁸⁷ He also referred to data by Carpenter in a draft report on the CONI program (presumably

¹⁷⁹ S Wolkind, E M Taylor, A J Waite, M Dalton and J L Emery, ‘Recurrence of Unexpected Infant Death’ (1993) 82 *Acta Paediatrica* 873, 873, 876.

¹⁸⁰ Nina Øyen, Rolv Skjaerven and Lorentz M Irgens, ‘Population-Based Recurrence Risk of Sudden Infant Death Syndrome Compared with other Infant and Fetal Deaths’ (1996) 144(3) *American Journal of Epidemiology* 300, 300.

¹⁸¹ C J Bacon et al, ‘How Common is Repeat Sudden Infant Death Syndrome?’ (2008) 93 *Archives of Disease in Childhood* 323, 324.

¹⁸² Foundation for the Study of Infant Deaths, *Report on 5,000 Babies Using the CONI (Care of Next Infant Programme)* (Foundation for the Study of Infant Deaths, October 1998).

¹⁸³ Foundation for the Study of Infant Deaths, *Report on 5,000 Babies Using the CONI (Care of Next Infant Programme)* (Foundation for the Study of Infant Deaths, October 1998).

¹⁸⁴ Darios Getahun et al, ‘Sudden Infant Death Syndrome among Twin Births: United States, 1995-1998’, (2004) 24 *Journal of Perinatology* 544, 544.

¹⁸⁵ Ray Hill, ‘Multiple Sudden Infant Deaths – Coincidence or Beyond Coincidence?’ (2004) 18 *Paediatric and Perinatal Epidemiology* 320, 321.

¹⁸⁶ Ray Hill, ‘Multiple Sudden Infant Deaths – Coincidence or Beyond Coincidence?’ (2004) 18 *Paediatric and Perinatal Epidemiology* 320, 321.

¹⁸⁷ Ray Hill, ‘Multiple Sudden Infant Deaths – Coincidence or Beyond Coincidence?’ (2004) 18 *Paediatric and Perinatal Epidemiology* 320, 322.

- preceding Carpenter et al, 2005), referring to nine families with three infant deaths; in eight, all three deaths were natural including two cases of triple SIDS.¹⁸⁸
118. In 2005, Carpenter et al published the paper on CONI infants which attracted subsequent debate.¹⁸⁹ Of 6,373 babies, 57 (8.9 per 1,000) died under the age of one year. Forty-one were “natural sudden unexpected deaths in infancy”.¹⁹⁰ The relative risk of recurrence as compared with the general population was at least 5.71 (4.10-7.74). The report identified four families with three SIDS or unexplained deaths. The authors considered that their data suggested second deaths were not rare and the majority – 80-90% (40 in 45; or 18 in 20) – were natural.
119. In separate letters to *The Lancet*, in which the paper had been published, Bacon and Vincent Di Maio challenged the findings of Carpenter et al.¹⁹¹ Bacon was concerned that instead of dichotomising the cases into unnatural or natural, it would have been more accurate to have a grey area of uncertainty. He said the data did not support such clear-cut conclusions as promoted by the authors.
120. In 2006 Gornall claimed that the authors of the Carpenter report had recategorised deaths that Emery – who was listed as a co-author of the Carpenter paper but had died – classed as unnatural or indeterminate.¹⁹² The authors later denied this. In a communication with the *British Medical Journal*, Carpenter was clear that “unnatural” meant filicide – everything else was “natural”. Gornall said this created an illogical corollary that all the deaths that were not unnatural must be natural – correct in court, but not in scientific research. The immediate past president of the Royal College of Paediatrics and Child Health wrote to *The Lancet* expressing alarm at the Carpenter paper, describing the analysis as seriously flawed and the findings as seriously misleading.¹⁹³
121. In 2007, Bacon and Hey re-analysed the deaths described by Carpenter and colleagues as “natural”. “Natural” was deaths from disease or a wholly accidental event. They concluded 13% probably unnatural; 43% probably natural; 43%

¹⁸⁸ Ray Hill, ‘Multiple Sudden Infant Deaths – Coincidence or Beyond Coincidence?’ (2004) 18 *Paediatric and Perinatal Epidemiology* 320, 323-324.

¹⁸⁹ R G Carpenter et al, ‘Repeat Sudden Unexpected and Unexplained Infant Deaths: Natural or Unnatural?’ (2005) 365 *Lancet* 29.

¹⁹⁰ R G Carpenter et al, ‘Repeat Sudden Unexpected and Unexplained Infant Deaths: Natural or Unnatural?’ (2005) 365 *Lancet* 29, 31.

¹⁹¹ C J Bacon, ‘Repeat Sudden Unexpected Infant Deaths’ (2005) 365 *Lancet* 1137; Vincent J M Di Maio, ‘Repeat Sudden Unexpected Infant Deaths’ (2005) 365 *Lancet* 1137.

¹⁹² Jonathan Gornall, ‘Was Message of Sudden Infant Death Study Misleading?’ (2006) 333 *British Medical Journal* 1165.

¹⁹³ Jonathan Gornall, ‘Was Message of Sudden Infant Death Study Misleading?’ (2006) 333 *British Medical Journal* 1165.

undetermined.¹⁹⁴ Part of the purpose was to show how a comparatively small change of perspective could result in a large change to conclusions.

122. The next year, Bacon re-examined studies of recurrent SIDS and concluded that the figures suggested were mainly too high.¹⁹⁵ He also concluded that on theoretical grounds there may well be an increased (but unquantifiable) risk in a subsequent sibling because of the persistence of genetic and environmental influences. Also, risk varies widely between families. Excluding conditions that might recur (familial disease, covert homicide, major SIDS risk factors), he considered that the chance of recurrence was very small.
123. In 2008 Bacon et al again assessed that the risk for a second SIDS death was probably greater than the risk for a first death for their subgroup, but the increase could not be quantified and was almost certainly less than that suggested by most of the previous studies.¹⁹⁶ There remained a theoretical argument of increased risk because of genetic and environmental influences. They emphasised using controls matched for degree of risk – otherwise, repeat SIDS in high-risk families give a false impression for the population as a whole.¹⁹⁷ Families whose initial infant death was fully investigated and who have no major risk factors might have a slightly increased risk of a second death, but it remained very small.¹⁹⁸
124. These findings were echoed in another 2008 study which stratified risk factors in computing the probability of a second SIDS death in a family.¹⁹⁹ Emphasis was placed on the importance of considering environmental factors – in a community with high rates of risk factors, most second SIDS would occur in high-risk families. In reality, a cohort of families with a first SIDS is not a random cross-section of the population; it is a selected group with a higher proportion of “high-risk” families. The majority of subsequent children in that cohort will be exposed to the same risk factors as in the index cases and the predicted risk will be higher than in the total population.²⁰⁰ Modelling suggested that that the risk of a second SIDS in families with no risk factors was very low.²⁰¹

¹⁹⁴ C J Bacon and E N Hey, ‘Uncertainty in Classification of Repeat Sudden Unexpected Infant Deaths in Care of Next Infant Programme’ (2007) 335 *British Medical Journal* 129.

¹⁹⁵ Christopher Bacon, ‘Recurrence of Sudden Infant Death Syndrome’ (2008) 122 *Pediatrics* 869.

¹⁹⁶ C J Bacon et al, ‘How Common is Repeat Sudden Infant Death Syndrome’ (2008) 93 *Archives of Disease in Childhood* 936.

¹⁹⁷ C J Bacon et al, ‘How Common is Repeat Sudden Infant Death Syndrome’ (2008) 93 *Archives of Disease in Childhood* 936.

¹⁹⁸ C J Bacon et al, ‘How Common is Repeat Sudden Infant Death Syndrome’ (2008) 93 *Archives of Disease in Childhood* 936.

¹⁹⁹ M J Campbell et al, ‘Recurrence Rates for Sudden Infant Death Syndrome (SIDS): The Importance of Risk Stratification’ (2008) 93 *Archives of Disease in Childhood* 936.

²⁰⁰ M J Campbell et al, ‘Recurrence Rates for Sudden Infant Death Syndrome (SIDS): The Importance of Risk Stratification’ (2008)

125. Another 2008 paper identified the multiple complexities attending any attempt to assess risk with particularity: interactions in the triple risk model; unidentified metabolic disorders; impossibility of ascertaining frequency of covert homicide; wrong assumptions that families in population-based studies are broadly representative of the population.²⁰² On a population level there are too many variants to resolve the issue.²⁰³ Consideration of risk of a subsequent SIDS should always take into account the known risk factors. However, for most families, the chances of recurrence were considered to be low.²⁰⁴
126. The Utah study in 2017 indicated that SIDS remained a heterogeneous group of causal entities with common presentation and unknown recurrence risk, although families with a prior SIDS were said to have moderately increased risks.²⁰⁵ Given the role of genetics particularly post-1995, however, true family-specific recurrence risk requires accurate underlying diagnosis.
127. The topic of sibling deaths was discussed in Duncan and Byard (2018), concluding (citing Beal and Blundell, 1988) that while multiple SIDS deaths in the one family may represent a genetic component in the aetiology of SIDS, for 92% of families the risk of recurrence is considered small.²⁰⁶

Awareness of three or more deaths in a single family

Trial

128. Evidence was elicited by the prosecution from a number of medical experts of whether they were aware, from personal experience or more broadly, of any case in which three or more children had died from sudden unexplained natural causes.

93 *Archives of Disease in Childhood* 936.

²⁰¹ M J Campbell et al, 'Recurrence Rates for Sudden Infant Death Syndrome (SIDS): The Importance of Risk Stratification' (2008)

93 *Archives of Disease in Childhood* 936, 938.

²⁰² Peter S Blair and Peter J Fleming, 'Recurrence Risk of Sudden Infant Death Syndrome' (2008) 93(4) *Archives of Disease in Childhood* 269.

²⁰³ Peter S Blair and Peter J Fleming, 'Recurrence Risk of Sudden Infant Death Syndrome' (2008) 93(4) *Archives of Disease in Childhood* 269, 270.

²⁰⁴ Peter S Blair and Peter J Fleming, 'Recurrence Risk of Sudden Infant Death Syndrome' (2008) 93(4) *Archives of Disease in Childhood* 269, 270.

²⁰⁵ Erik D Christensen et al, 'Sudden Infant Death "Syndrome" – Insights and Future Directions from a Utah Population Database Analysis' (2017) 173 *American Journal of Medical Genetics Part A* 177.

²⁰⁶ Exhibit D, Jhodie R Duncan and Roger W Byard, 'Sudden Infant Death Syndrome: An Overview' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 15, 27.

Evidence was led from Professor Herdson, Professor Berry, Dr Beal and Professor Byard.²⁰⁷

129. Dr Beal said that as far as she was aware, from her experience or the literature, there had never been three or more deaths from SIDS in the one family.²⁰⁸ She had not ever come across a family in which there had been three or more children who had died suddenly from natural causes in the way that the Folbigg children had died.²⁰⁹ Dr Beal had come across a family with three deaths, albeit with causes of death that she did not believe were SIDS (see below [140]).
130. Professor Herdson also was not aware from his experience and or literature, of three or more, thoroughly investigated, infant deaths from SIDS in one family.²¹⁰
131. Professor Berry was not aware of any case, from his experience or the literature, where three or more children in one family had suffered sudden death from no obvious injury or disease.²¹¹ He nonetheless considered that it was important to explore this possibility.²¹² Except for some reports many years previously which did not withstand scrutiny, he was unaware in contemporary literature, or from his practice or research, of any families with three or more deaths from SIDS.²¹³ Nor was he aware of any three or more kindred children, previously fit, who had died suddenly due to another medical condition.²¹⁴ He also said “that’s not to say they don’t exist, but I’m personally unaware of any in the literature.”²¹⁵
132. Professor Byard had never heard of a case in which three or more children in one family had died or had an ALTE suddenly, unexpectedly, during a sleep period at home.²¹⁶ Under cross-examination, he agreed it would not be a reasonable conclusion that all the Folbigg children died from the same natural cause, although he could not exclude it; he had also never heard of a case in which four children in one family had died suddenly and unexpectedly from four different natural causes.²¹⁷

²⁰⁷ *R v Folbigg* [2005] NSWCCA 23, [49]-[50].

²⁰⁸ 5 May 2003 T1136.50-56, T1143.52-1144.2; Exhibit H, Forensic pathology tender bundle, p 216.

²⁰⁹ 5 May 2003 T1144.2.

²¹⁰ 1 May 2003 T1049.51-56; Exhibit H, Forensic pathology tender bundle, p 275.

²¹¹ 1 May 2003 T1080.5-33, T1081.27-T1082.21; Exhibit H, Forensic pathology tender bundle, p 256.

²¹² R Exhibit H, Forensic pathology tender bundle, p 256.

²¹³ 1 May 2003 T1066.36-44; See also Exhibit H, Forensic pathology tender bundle, p 256.

²¹⁴ 1 May 2003 T1066.46-58.

²¹⁵ 1 May 2003 T1066.57-58.

²¹⁶ 7 May 2003 T1222.42-46.

²¹⁷ 7 May 2003 T1249.23-26, T1253.3-13, T1258.40-58.

133. Professor Busuttil stated in his report (not tendered at trial) that it was extremely unusual and quite unprecedented to have four deaths of siblings in the same family over eight years – he had never seen or heard of this occurrence in over thirty years in pathology practice.²¹⁸

Inquiry

134. Dr Cala gave evidence in the Inquiry that he has not received a case of three deaths in these circumstances since 2004.²¹⁹ Professor Hilton said that he has not been directly involved in any cases with a subsequent death since 2004 (he is now retired).²²⁰ Professor Duflou recalled two cases before 2004 in which he had found two sudden infant deaths in a family.²²¹

Evidence of recurrence of SIDS/unexplained deaths

Trial

135. Dr Cooper gave evidence that a familial or inherited link in SIDS was extremely improbable.²²² Compared with the 1970s, when it was believed that SIDS was often familial, by 2003 an increased risk of recurrence of SIDS in a family could not absolutely be excluded but the likelihood of recurrence was probably no higher than the general population.²²³
136. He thought that having one SIDS death did not predispose the family to another, and there was no or very slightly increased risk.²²⁴ Whereas 10 years prior, literature would have said the risk was very much increased.²²⁵ While an increased risk could not absolutely be excluded, previously the likelihood of a second SIDS death had been argued to be several-fold.²²⁶ There was still debate about whether it was a little higher or no higher at all.²²⁷
137. In a statement, Dr Beal stated that “there are a few disorders which may present as recurrent infant death. These can be excluded by appropriate investigations e.g.

²¹⁸ Exhibit H, Forensic pathology tender bundle, p 305.

²¹⁹ Transcript of the Inquiry, 19 March 2019 T76.21-24.

²²⁰ Transcript of the Inquiry, 19 March 2019 T77.16.

²²¹ Transcript of the Inquiry, 20 March 2019 T175.2.

²²² 14 April 2003 T611.42, T612.6-51, T614.45-47, T615.4.

²²³ 14 April 2003 T590.31-40, T591.4-592.18, T608.8-49, T608.52-57, T610.47-T611.2, T614.40-47.

²²⁴ 14 April 2003 T610.47-T611.2, T614.40-47.

²²⁵ 14 April 2003 T610.57-T611.2.

²²⁶ 14 April 2003 T608.15.

²²⁷ 14 April 2003 T608.8-49.

metabolic disorders or cardiac arrhythmias [sic].”²²⁸ She opined that there were two more common causes in relation to recurrent sudden unexpected deaths: leaving an infant prone and unobserved; and filicide, which “is likely to continue into a third or even fourth or more children.”²²⁹

138. In a separate communication with the Crown, Dr Beal addressed the issue of whether SIDS runs in families.²³⁰ She stated that because families tend to care for all their infants in the same way, risk for a second baby if placed prone would be the same as the risk for a first infant placed prone.²³¹ In relation to the Folbigg deaths, Dr Beal stated that one of the reasons why the fourth death would “not only not be called SIDS but would alter the thinking about the first three deaths” was that:

*[i]n all the families I know where there have been more than 3 sudden unexpected deaths there have been several initially described as SIDS until another diagnosis has been discovered and the earlier SIDS diagnosis has been changed.*²³²

139. However, Dr Beal deferred to relevant experts on the cause of Patrick’s ALTE, his death and Laura’s death. Her evidence on three sudden unexpected deaths should be understood as qualified accordingly.²³³
140. Dr Beal referred to her personal experience of interviewing parents and carers of over 500 infants who died suddenly and unexpectedly, 13 families with two infants and one with three who had died.²³⁴ In six of the families with two deaths, Dr Beal believed all the deaths were SIDS. In seven, another problem was either diagnosed or suspected. In the family with three deaths, Dr Beal did not believe any of the three died of SIDS.²³⁵ She was otherwise aware of three families with more than three sudden unexpected deaths, however, it had been accepted that the children were all intentionally suffocated.²³⁶

²²⁸ Exhibit H, Forensic pathology tender bundle, p 217.

²²⁹ Exhibit H, Forensic pathology tender bundle, p 218.

²³⁰ Trial Exhibit C (VD), Facsimile from Dr Susan Beal to ODPP (24 April 2003).

²³¹ Trial Exhibit C (VD), Facsimile from Dr Susan Beal to ODPP (24 April 2003).

²³² Trial Exhibit C (VD), Facsimile from Dr Susan Beal to ODPP (24 April 2003) pp 1-2.

²³³ 5 May 2003 T1138.55-1139.12, T1147.35-36, T1142.39-44, T1149.1-27, T1139.52-T1140.2, T1146.45-1147.46. In relation to the agonal rhythm trace of Laura’s heart and evidence of whether breathing or heart rhythm stopped first, see 5 May 2003 T1143.1-17.

²³⁴ Trial Exhibit C (VD), Facsimile from Dr Susan Beal to ODPP (24 April 2003).

²³⁵ Trial Exhibit C (VD), Facsimile from Dr Susan Beal to ODPP (24 April 2003) p 2.

²³⁶ Trial Exhibit C (VD), Facsimile from Dr Susan Beal to ODPP (24 April 2003).

141. Dr Beal adhered on the voir dire to evidence she had given in a previous case that “the first one is called SIDS. The second one is undetermined and the third one should be considered murder, until it is proven not to be”.²³⁷ She agreed that this formula was “absolutely” a very significant part of her reasoning in the Folbigg case.²³⁸ Professor Herdson made a similar statement in his report.²³⁹ The prosecution did not lead such evidence before the jury. In our submission, that reasoning should not be accepted or adopted by the Inquiry.
142. Dr Janice Ophoven was a paediatric forensic pathologist based in Minnesota, USA.²⁴⁰ In her report she stated, *inter alia*, that the statistical probability that four children in one sibship could die from SIDS would be infinitesimally small less than one in one trillion.²⁴¹ Dr Ophoven did not give evidence at trial and her report was not tendered into evidence. Again, in our submission her opinion should be rejected.

Inquiry

143. In the Inquiry, Professor Horne gave evidence that SIDS itself is rare, and instances of recurrence are very rare.²⁴² Professor Elder said that the risk of recurrence is affected by genetic and environmental factors, mainly a prone sleeping position or bed sharing.²⁴³
144. Professor Duflou and Dr Cala took no issue with the description of “small risk” in Duncan and Byard (2018).²⁴⁴ Professor Duflou considered that the chances of a second or third SIDS death in the same family is unchanged with each sibling and a second death may be SIDS 2 if there are no circumstances of concern.²⁴⁵ He thought there is no dispute that some unexpected deaths in infancy which appear on the face of it to be SIDS have a genetic basis (which in any event would make them not SIDS).²⁴⁶ He regarded the debate that arose in literature, ignited by the article by Carpenter et al (2005) and referred to above, about proportions of natural/unnatural infant deaths as peripheral to the advance in thinking that the

²³⁷ 28 April 2003 T986.54-T987.30.

²³⁸ 28 April 2003 T986.54-987.24.

²³⁹ Exhibit H, Forensic pathology tender bundle, p 275.

²⁴⁰ Exhibit H, Forensic pathology tender bundle, pp 220-221.

²⁴¹ Exhibit H, Forensic pathology tender bundle, pp 264, 267, 270.

²⁴² Transcript of the Inquiry, 18 March 2019 T33.40.

²⁴³ Transcript of the Inquiry, 18 March 2019 T34.9-20.

²⁴⁴ Transcript of the Inquiry, 19 March 2019 T99.16-25.

²⁴⁵ Transcript of the Inquiry, 19 March 2019 T75.24-43, T98.23-25.

²⁴⁶ Transcript of the Inquiry, 19 March 2019 T99.16-19.

chances of a second or subsequent SIDS are unchanged from those of the first, and are not determined by simply multiplying the risks.²⁴⁷

145. Professor Hilton agreed with Professor Byard's statement (referred to in paragraph 127 above) that there appears to be some slight risk for a subsequent SIDS death in a family, given the lack of understanding of what SIDS really is.²⁴⁸ He observed that statistics on recurrence of SIDS "adopt the understanding or misunderstanding of SIDS literally from day one", agreeing with Professor Duflou that they should be put to one side.²⁴⁹
146. Dr Cala would not call a third death in a family SIDS. In a second death, before giving a SIDS diagnosis he would look very carefully at the circumstances of both deaths, both autopsies and any missing genetic metabolic or other abnormality, and also exclude suspicion of foul play.²⁵⁰
147. Professor Cordner accepted that the risk of recurrence could be described as rare or small, noting that literature referred to in Duncan and Byard (2018) on this point is not recent, but that Professor Byard is Australia's expert in overlap between forensic pathology and paediatric pathology and his opinion is very important.²⁵¹
148. In his report, Professor Cordner stated that while both situations (multiple SIDS and multiple homicide) are rare, two or three natural deaths in one family probably occurs more frequently than the same number of hidden homicides.²⁵² This appears to have been drawn from literature such as Carpenter et al (2005). In evidence he agreed it was possible that Carpenter's findings did not represent great science and that Carpenter, who adopted a natural/unnatural dichotomy, may have categorised cases as unnatural only if he had a very high level of certainty.²⁵³ Professor Cordner was ultimately content with Bacon's view of 43% natural, 43% uncertain and the rest probable homicides, noting the high level uncertainty in looking at literature alone.²⁵⁴

²⁴⁷ Transcript of the Inquiry, 19 March 2019 T98.26-35.

²⁴⁸ Transcript of the Inquiry, 19 March 2019 T99.41-44.

²⁴⁹ Transcript of the Inquiry, 19 March 2019 T99.29-35.

²⁵⁰ Transcript of the Inquiry, 19 March 2019 T76.5-24.

²⁵¹ Transcript of the Inquiry, 19 March 2019 T94.3-11, T296.35-39.

²⁵² Exhibit Q, Report of Professor Stephen Cordner (undated) p 90; Transcript of the Inquiry, 19 March 2019 T97.8-25.

²⁵³ Transcript of the Inquiry, 19 March 2019 T93.13-20.

²⁵⁴ Transcript of the Inquiry, 21 March 2019 T295.39-296.5.

149. It is clear that descriptions in literature and in evidence by experts emphasise the low nature of recurrence risk. Professor Horne said that SIDS itself is rare and recurrence is very rare. Variations on this found in evidence and literature include that the risk in a second infant in a family is the same as for the first; that it is small, very small, low, rare, very rare; that in a family with no risk factors it is very low or slightly increased but very small. The weight of the evidence is that any increased risk of recurrence in a sibling is affected by genetic and environmental factors.

The legal context

150. The debate relating to repeat SIDS deaths in England was generated in the first decade of this century largely from the cases of *Clark* [2000] EWCA Crim 54 and *Cannings* [2004] WLR 2607 where convictions of two mothers for murder of their children were each overturned. In the prosecution of Sally Clark for the murder of two of her children, Professor Sir Roy Meadow gave evidence, part of which was “the chance of two children dying naturally in these circumstances is very, very long indeed, one in 73 million”.²⁵⁵
151. It transpired his mathematical calculation was wrong. As a result the General Medical Council removed him from the medical register but he was restored on appeal because of his eminence as a paediatrician and the fact the mistake he made was outside his field of expertise.²⁵⁶
152. In *R v Cannings* the English Court of Appeal in 2004 dealt with a similar case and overturned the conviction of Angela Cannings. Ms Cannings had four children, three of whom died in infancy. She was charged with the murder of two children and convicted at trial. She was subsequently acquitted on appeal. In addition to fresh evidence including of a realistic possibility of a genetic problem in the family, the Court of Appeal decision was on the basis that the prosecution case critically relied on the coincidence of the deaths and stated that with one, two or even three deaths, the exclusion of currently known natural causes of infant death does not establish beyond a reasonable doubt that the death or deaths resulted from the deliberate infliction of harm.²⁵⁷

²⁵⁵ *R v Clark* [2000] EWCA Crim 54, [122].

²⁵⁶ *Meadow v General Medical Council* [2006] EWHC 146 (Admin).

²⁵⁷ *R v Cannings* [2004] WLR 2607, [13], [175].

“Default diagnosis”

153. Professor Cordner contended in his report that there was a “default diagnosis” of murder in the way in which the trial was conducted by admitting evidence as to the rarity of unexplained deaths.²⁵⁸ He adds in parenthesis that (“Clearly this was assisted by other circumstantial information which it is not for me to assess”).²⁵⁹
154. Professor Cordner made no complaint that any of the evidence given by the experts at the trial as to the rarity of the unexplained deaths, was (or is) inaccurate.
155. A similar argument was made by Ms Folbigg in her second appeal that the evidence of the rarity of multiples deaths has the effect of a “default diagnosis” and that such evidence reverses the onus of proof.²⁶⁰
156. As set out above, the forensic pathology and SIDS experts (i.e. Dr Beal and Professors Herdson, Berry and Byard) each gave evidence that he or she was not aware (from professional experience, the experiences of colleagues and review of the medical literature) of any family in which three or more children had died from SIDS and/or some other natural cause/s.²⁶¹ This evidence was admitted after legal argument.
157. In addition, Professor Berry stated that his research of the standard database called Medline used by medical practitioners around the world revealed no such case.²⁶²
158. As noted in Chapter 1, ground 3 of the second appeal challenged the admissibility of the expert knowledge of other incidences of multiple natural infant deaths in a family. The argument was that it ought to have been excluded pursuant to s 137 of the *Evidence Act 1995*.²⁶³ Ms Folbigg contended that the trial miscarried as a result of evidence being led from prosecution experts to the effect that they were unaware of any previous case in medical history where three or more infants in the one family died suddenly as a result of disease processes.

²⁵⁸ Exhibit Q, Report of Professor Stephen Cordner (undated) p 7.

²⁵⁹ Exhibit Q, Report of Professor Stephen Cordner (undated) p 59.

²⁶⁰ *R v Folbigg* [2005] NSWCCA 23, [71].

²⁶¹ 1 May 2003 T1049.51-56 (Professor Herdson); 1 May 2003 T1136.50-56, T1143.52-1144.2 (Dr Beal); 1 May 2003 T1066.53-1067.11 (Professor Berry); 7 May 2003 T1222.42-1223.19 (Professor Byard).

²⁶² 1 May 2003 T1080.35-58 (Professor Herdson).

²⁶³ Written submissions of the Applicant in the second appeal to the NSWCCA (3 July 2004) [108].

159. The Criminal Court of Appeal treated the ground as a challenge to the trial judge's decisions in relation to the evidence of Dr Cala and the other experts which allowed the evidence to be led.²⁶⁴ The relevance of the evidence does not appear to have been challenged by Ms Folbigg in the Criminal Court of Appeal. Nonetheless, the Criminal Court of Appeal also found the evidence to be relevant.²⁶⁵
160. The Criminal Court of Appeal rejected the argument that the jury would misuse expert evidence about the rarity of multiple unexpected deaths by reversing the onus of proof, provided that jury directions made clear that it was from first to last the burden of the Crown to prove its case and was not the burden of Ms Folbigg to prove anything.²⁶⁶ The Criminal Court of Appeal found that the trial judge gave clear and correct directions on this principle, both orally and in writing.²⁶⁷
161. In relation to the asserted unfair prejudice of the evidence, the Criminal Court of Appeal found that contrary to the argument presented by Ms Folbigg, the expert evidence on the point did not present a danger of the jury misusing the evidence in a way that statistics had been misused in DNA profiling cases. Those cases used expert opinions to propose quite precise probabilities (e.g. 220,000:1/999.9995%),²⁶⁸ different from the opinion evidence by Professors Herdson and Berry and Dr Beal. The evidence also did not fall into the template of the "Prosecutor's fallacy", which used incorrect derivatives of statistics to assert likelihood of guilt.²⁶⁹
162. Rather, the Criminal Court of Appeal found, the opinion evidence of the experts in this case did no more than establish (if accepted by the jury) that reputable and apparently reliable expert opinion could not identify another known case of four infant deaths in one family from unknown natural causes. In a circumstantial prosecution case, which this was, that fact (if accepted) was no more than a piece of circumstantial evidence to be added to all the other known facts and circumstances concerning the four deaths on which the Crown case relied.²⁷⁰

²⁶⁴ *R v Folbigg* [2005] NSWCCA 23, [76].

²⁶⁵ *R v Folbigg* [2005] NSWCCA 23, [50].

²⁶⁶ *R v Folbigg* [2005] NSWCCA 23, [83].

²⁶⁷ *R v Folbigg* [2005] NSWCCA 23, [83].

²⁶⁸ *R v Folbigg* [2005] NSWCCA 23, [87].

²⁶⁹ *R v Folbigg* [2005] NSWCCA 23, [88].

²⁷⁰ *R v Folbigg* [2005] NSWCCA 23, [91].

163. As such, the findings by the Criminal Court of Appeal in the second appeal address, in substance, the assertion by Professor Cordner effectively that the expert evidence generated a “default diagnosis”. Professor Cordner appears to have overlooked the rules of evidence and procedure applicable in criminal trials and considered by the Criminal Court of Appeal, and the process by which the Crown is, and was, required to discharged its onus. He also appears to misapprehend the nature of the circumstantial Crown case that was presented to the jury, of which the medical evidence was a significant part, but nonetheless a part only.
164. The evidence of the experts’ knowledge of other cases did not reverse the onus of proof as contended in the application. That argument has been specifically rejected by the Criminal Court of Appeal, for the reasons above, and there has been identified no basis on which it should be entertained again in the Inquiry.

Recurrence in submissions and summing up at trial

165. In his closing address at trial, the Crown Prosecutor said that there had:

never been recorded a family such as this one where four children have died of natural causes, either from the same natural cause or from different natural causes. There have never been three or more deaths in the one family recorded from SIDS... what that means... is this:... It does not mean that it could not happen. What it does mean is, it is an expression of how rare it must be that it has never been recorded. I mean it has never been recorded that the same person has been hit by lightning four times, I presume. That does not mean it has never happened. It does not mean it could never happen. You might have some person living in the backwards of India who has been hit by lightning four times, but it is an expression of its rarity that there has never been... It is probably more common that a person has been hit by lightning four times than what has happened to this family, you might think.²⁷¹

166. Later in his closing, the Crown Prosecutor submitted to the jury that he anticipated that the defence would say that the Crown had not proven that the children did not die of natural causes and drew the analogy as to piglets flying.²⁷²
167. In the summing up at end of the trial, the trial judge directed the jury on the evidence: that SIDS deaths are rare, there is no authenticated record of three or

²⁷¹ 13 May 2003 T1364.39-53.

²⁷² 13 May 2003 T1375.23-27.

more such deaths in a single family, and whilst not impossible it is an illustration of the rarity of deaths diagnosed as SIDS.²⁷³ The trial judge added:

*You appreciate that the experts, quite a number of them, expressed themselves as not being in the business of certainty. They looked at probabilities and they told you from time to time that they thought some particular thing was quite unlikely, but they could not exclude it.*²⁷⁴

168. Accordingly, the jury retired to deliberate under the impression that, not only were multiple SIDS deaths rare, but that there was no record of three or more deaths in a single family from unidentified natural causes, or SIDS. Also, as to the extent of the rarity of such a coincidence, the jury were left with the Crown's submission that it was as likely as pigs flying or a person being struck by lightning four times, as well as the trial judge's direction that four SIDS deaths were not impossible. The trial judge did not directly refer to the Crown's analogies in his Honour's summing up, although the jury ought to have appreciated that his Honour's direction left open the possibility of four SIDS deaths (more than did the Crown's flying piglet analogy).
169. In view of the qualifications and expertise of some of the experts who gave evidence at trial, it would have been open to the jury to conclude that no such case had ever been recorded. The inference available to be drawn from that proposition was that no such case had occurred within jurisdictions that have systems for recording such a case, discoverable by experts. Evidence given by Professor Berry, in particular, went further than simply what was within his own professional experience he said that his research of the standard data base "Medline" used by medical practitioners around the world revealed no case of sudden unexpected (presumably multiple) death with no preceding illness which would not be revealed by a post mortem.
170. The arguments in the conviction appeal, in relation to a different ground, raised the existence of research which was admitted into evidence on appeal in *Cannings*. That research, which appears to include a study by Emery and Wolkind which has been reviewed by this Inquiry,²⁷⁵ did identify such instances of three or more natural deaths. The research itself was not received into evidence by the

²⁷³ 19 May 2003 T24.

²⁷⁴ 19 May 2003 T56-57.

²⁷⁵ S Wolkind, E M Taylor, A J Waite, M Dalton and J L Emery, 'Recurrence of Unexpected Infant Death' (1993) 83 *Acta Paediatrica* 873.

Criminal Court of Appeal. This may have been for a range of forensic or other reasons which are not now known or relevant.

171. Even though the Criminal Court of Appeal did not receive research into evidence, the Court was plainly aware of its existence via *Cannings*. The Court was aware that, at the time of the second appeal, there were identified cases in other jurisdictions where, contrary to the inference arising from evidence given at the trial, there had in fact been three or more deaths of infants in the one family, and of the conclusion in *Cannings* that the mere fact of multiple deaths did not prove murder. There is no disagreement with, or qualification, of that proposition expressed in the Criminal Court of Appeal's judgment.

Conclusion

172. It is clear from the work of the Inquiry that before 2003 there had been reported cases involving the deaths of three or more infants in the same family attributed to unidentified natural causes, or at least not established as attributable to unnatural causes. This is not to suggest that the truth of the evidence of the four experts at trial of their unawareness of such incidence should be doubted.
173. However, the current descriptions in literature and in evidence by experts emphasise the low nature or rarity of recurrence risk. The weight of evidence is that any increased risk of recurrence in a sibling is affected by genetic and environmental factors. In the Folbigg family, no genetic factor has been identified. Environmental factors which applied in each death of the Folbigg children gave rise to a low risk of sudden unexplained infant death.
174. Thus, the observation by the trial judge that such events are not impossible and that they are rare reflected the knowledge held then and that remains the scientific evidence today. In short, it was correct. We submit that there is no basis to assert a miscarriage of justice arose following directions by the trial judge in relation to the expert evidence. Nor is there a basis to assert that they constituted an irregularity that would give rise to reasonable doubt as to guilt.