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.
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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{173} A reference was made to a report by Rosen et al in 1983, of a family with three previous SIDS victims.\textsuperscript{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.\textsuperscript{175} (It was later suggested by Bacon and colleagues that this may have been inflated.)\textsuperscript{176} Beal and Blundell noted that:

\begin{quote}
for 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.\textsuperscript{177}
\end{quote}

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.\textsuperscript{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

\begin{footnotes}
\textsuperscript{175} S M Beal and H K Blundell, ‘Recurrence Incidence of Sudden Infant Death Syndrome’ (1988) 63 Archives of Disease in Childhood 924, 929.
\textsuperscript{176} C J Bacon et al, ‘How Common is Repeat Sudden Infant Death Syndrome?’ (2008) 93 Archives of Disease in Childhood 323, 324.
\textsuperscript{177} S M Beal and H K Blundell, ‘Recurrence Incidence of Sudden Infant Death Syndrome’ (1988) 63 Archives of Disease in Childhood 924, 924.
\end{footnotes}
and suggested that the chance of recurrence was very small, probably no greater than the general occurrence of such deaths.\(^\text{179}\)

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.\(^\text{180}\) However, the autopsy rate was poor.\(^\text{181}\)

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.\(^\text{182}\) 104 of the parents in the CONI program had experienced two previous baby deaths, and four had experienced three previous deaths.\(^\text{183}\) 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.\(^\text{184}\) 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.\(^\text{185}\) It was “intuitively clear” that a subsequent infant will be at increased risk, because many genetic and environmental factors will be the same.\(^\text{186}\) Hill estimated the risk of SIDS was between five and 10 times greater for a second sibling.\(^\text{187}\) He also referred to data by Carpenter in a draft report on the CONI program (presumably


\(^\text{180}\) 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.

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

\(^\text{182}\) 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).

\(^\text{183}\) 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).


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

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

\(^\text{187}\) Ray Hill, ‘Multiple Sudden Infant Deaths – Coincidence or Beyond Coincidence?’ (2004) 18 Paediatric and Perinatal Epidemiology 320, 322.
preceeding 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.
undetermined. 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. 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. 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. 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.

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. 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. Modelling suggested that that the risk of a second SIDS in families with no risk factors was very low.

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.\textsuperscript{202} On a population level there are too many variants to resolve the issue.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.


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.\(^\text{218}\)

**Inquiry**

134. Dr Cala gave evidence in the Inquiry that he has not received a case of three deaths in these circumstances since 2004.\(^\text{219}\) Professor Hilton said that he has not been directly involved in any cases with a subsequent death since 2004 (he is now retired).\(^\text{220}\) Professor Duflou recalled two cases before 2004 in which he had found two sudden infant deaths in a family.\(^\text{221}\)

**Evidence of recurrence of SIDS/unexplained deaths**

**Trial**

135. Dr Cooper gave evidence that a familial or inherited link in SIDS was extremely improbable.\(^\text{222}\) 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.\(^\text{223}\)

136. He thought that having one SIDS death did not predispose the family to another, and there was no or very slightly increased risk.\(^\text{224}\) Whereas 10 years prior, literature would have said the risk was very much increased.\(^\text{225}\) While an increased risk could not absolutely be excluded, previously the likelihood of a second SIDS death had been argued to be several-fold.\(^\text{226}\) There was still debate about whether it was a little higher or no higher at all.\(^\text{227}\)

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 arhythmias [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”.\textsuperscript{237} She agreed that this formula was “absolutely” a very significant part of her reasoning in the Folbigg case.\textsuperscript{238} Professor Herdson made a similar statement in his report.\textsuperscript{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.\textsuperscript{240} In her report she stated, \textit{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.\textsuperscript{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.\textsuperscript{242} Professor Elder said that the risk of recurrence is affected by genetic and environmental factors, mainly a prone sleeping position or bed sharing.\textsuperscript{243}

144. Professor Duflou and Dr Cala took no issue with the description of “small risk” in Duncan and Byard (2018).\textsuperscript{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.\textsuperscript{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).\textsuperscript{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

\textsuperscript{237} 28 April 2003 T986.54-T987.30.  
\textsuperscript{238} 28 April 2003 T986.54-987.24.  
\textsuperscript{239} Exhibit H, Forensic pathology tender bundle, p 275.  
\textsuperscript{240} Exhibit H, Forensic pathology tender bundle, pp 220-221.  
\textsuperscript{241} Exhibit H, Forensic pathology tender bundle, pp 264, 267, 270.  
\textsuperscript{242} Transcript of the Inquiry, 18 March 2019 T33.40.  
\textsuperscript{243} Transcript of the Inquiry, 18 March 2019 T34.9-20.  
\textsuperscript{244} Transcript of the Inquiry, 19 March 2019 T99.16-25.  
\textsuperscript{245} Transcript of the Inquiry, 19 March 2019 T75.24-43, T98.23-25.  
\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{254}

\textsuperscript{247} Transcript of the Inquiry, 19 March 2019 T98.26-35.
\textsuperscript{248} Transcript of the Inquiry, 19 March 2019 T99.41-44.
\textsuperscript{249} Transcript of the Inquiry, 19 March 2019 T99.29-35.
\textsuperscript{250} Transcript of the Inquiry, 19 March 2019 T76.5-24.
\textsuperscript{251} Transcript of the Inquiry, 19 March 2019 T94.3-11, T296.35-39.
\textsuperscript{252} Exhibit Q, Report of Professor Stephen Cordner (undated) p 90; Transcript of the Inquiry, 19 March 2019 T97.8-25.
\textsuperscript{253} Transcript of the Inquiry, 19 March 2019 T93.13-20.
\textsuperscript{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”.\(^\text{255}\)

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.\(^\text{256}\)

152. In \textit{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.\(^\text{257}\)

\(^{255}\) \textit{R v Clark} [2000] EWCA Crim 54, [122].
\(^{256}\) \textit{Meadow v General Medical Council} [2006] EWHC 146 (Admin).
\(^{257}\) \textit{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{266} The Criminal Court of Appeal found that the trial judge gave clear and correct directions on this principle, both orally and in writing.\textsuperscript{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%),\textsuperscript{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.\textsuperscript{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.\textsuperscript{270}

\textsuperscript{264} R v Folbigg [2005] NSWCCA 23, [76].
\textsuperscript{265} R v Folbigg [2005] NSWCCA 23, [50].
\textsuperscript{266} R v Folbigg [2005] NSWCCA 23, [83].
\textsuperscript{267} R v Folbigg [2005] NSWCCA 23, [83].
\textsuperscript{268} R v Folbigg [2005] NSWCCA 23, [87].
\textsuperscript{269} R v Folbigg [2005] NSWCCA 23, [88].
\textsuperscript{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.\textsuperscript{273} The trial judge added:

\textit{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.}\textsuperscript{274}

\textbf{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).

\textbf{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.

\textbf{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 \textit{Cannings}. That research, which appears to include a study by Emery and Wolkind
which has been reviewed by this Inquiry,\textsuperscript{275} \textit{did identify} such instances of three or
more natural deaths. The research itself was not received into evidence by the

\textsuperscript{273} 19 May 2003 T24.
\textsuperscript{274} 19 May 2003 T56-57.
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.