IN THE CORONERS COURT

OF VICTORIA

AT MELBOURNE

Court Reference: COR 2014 5251

FINDING INTO DEATH WITHOUT INQUEST

Form 38 Rule 60(2)

Section 67 of the ***Coroners Act 2008***

I, AUDREY JAMIESON, Coroner having investigated the death of CHILD A

without holding an inquest:
find that the identity of the deceased was CHILD A
born 11 January 2011
and the death occurred on 13 October 2014
at Monash Medical Centre Clayton, 246 Clayton Road, Clayton Victoria 3168
from:

1 (a) THROMBOTIC MICROANGIOPATHY

Pursuant to section 67(1) of the **Coroners Act 2008**, I make findings with respect to **the following circumstances**:

1. Child A[1] was three years of age at the time of his death. He lived in Frankston with his parents and younger brother. Child A's family are practicing Jehovah's Witnesses. Child A's medical

[1] The identity of Child A has been suppressed pursuant to the *Open Courts Act 2013*, by my own motion, dated 22 June 2016.

history included reflux as an infant and varicella infection[2] at the age of five months old. Child A was immunised according to the Australian Schedule and his growth and development were normal. Apart from a seeming intolerance to dairy, gluten and eggs, Child A was predominantly a healthy child.

2. On 30 September 2014, Child A presented to the Medicentre Clinic located at Frankston Hospital, having been unwell for 24 hours with abdominal pain, diarrhoea and fever. He was seen by Dr Peter Keillar. Child A had a fever of 39.3°C at the review, but looked clinically well and his abdomen was soft to examine. He was diagnosed with a viral gastroenteritis and discharged home with advice to encourage oral fluids and for repeat review if there was parental concern.

3. On 2 October 2014, Child A re-presented to the Medicentre GP Clinic with ongoing diarrhoea, vomiting, and abdominal pain associated with passing bowel actions. He was seen by Dr Subodhani Sirisena and was assessed as having ongoing gastroenteritis with no signs of dehydration. His abdomen was assessed as being soft, non-tender with no rebound tenderness[3] or guarding[4]. Child A was discharged home with advice about hydration and a pathology request for a stool sample to be sent for microscopy and culture in case of a bacterial form of gastroenteritis. He was for repeat review if there were further concerns. The stool sample was collected by Child A's parents and sent for microscopy and culture on 3 October 2014 to Healthscope Pathology in Frankston.

4. On the afternoon of 4 October 2014 Child A presented to Frankston Hospital Emergency Department (ED) after developing blood in his stools and had stopped the intake of oral fluids. He was reviewed by an ED Doctor who noted that he had experienced five days of diarrhoea and one day of mucous and blood in his stool, with three days of vomiting and reduced oral intake. It was noted that Child A had cramping abdominal pain associated with passing bowel actions. He was assessed and found to have a soft abdomen and perianal excoriation. He had a heart rate of 105, blood pressure of 95/60mmHg, oxygen saturations of 98 percent on room air and a temperature of 36.5°C, all within normal limits for his age. Child A was then referred to a

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[2] Chicken pox.

[3] A clinical sign of peritoneal inflammation in which pain is elicited upon removal of pressure rather than application of pressure to the abdomen.

[4] A clinical sign of peritoneal inflammation in which palpation of the abdomen elicits contraction of the abdominal muscles.

paediatric registrar for admission to hospital for ondansetron[5] and a trial of oral intake. Child A initially had a nasogastric tube[6] inserted and gastrolyte[7] commenced, however he removed the nasogastric tube one hour later and it was not reinserted. He was assessed by a paediatric registrar as having viral gastroenteritis with poor oral intake but no dehydration.

5. Sources of infection were discussed and it was documented that both of Child A's parents had possible loose stools. Child A had also attended a party eight days prior to admission and one of the other children in attendance possibly had gastroenteritis. It would later be noted by nursing staff on 5 October 2014 in the medical record that Child A's father identified other possible sources of infection including swimming at the new Frankston pool two weeks prior to this presentation, and drinking unpasteurised milk.

6. On 5 October 2014 at 9.15am Child A was reviewed by Paediatrician Dr Jeevani Ranaweera on the ward round. It was noted that his hydration status had improved with 300g of weight gain, but that he had ongoing frequent diarrhoea (12 episodes since midnight) with blood and mucous present in the stool. His abdomen was soft to examine but tender on the left side with no guarding. At this point the impression was that he had gastroenteritis but could possibly have colitis.[8] The plan was to continue to treat his symptoms with ondansetron and carry out investigations with blood tests as well as obtain the result for the stool sample taken at Healthscope. A repeat stool sample was also sent for testing on 5 October 2014.

7. The blood tests showed that Child A had signs of infection. He had a high neutrophil count[9] and an elevated C - reactive protein[10] of 16mg/L. These results were discussed with Dr David Burgner, a paediatric Infectious Diseases Consultant at Monash Medical Centre (MMC). Child A was commenced on oral azithromycin[11] with plans to have further communication with Dr Burgner as more test results became available. After this discussion, on the evening of 5 October 2014, a verbal report from Healthscope Pathology became available showing the initial

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[5] A meidcation used for its antiemetic (anti-nausea) properties.

[6] This tube is inserted through the nose and into the stomach and can be used to provide nutrition and fluids.

[7] An electrolyte containing rehydration solution.

[8] Inflammation of the colon.

[9] White blood cells that may be elevated in infection.

[10] The C-reactive protein (CRP) is a protein in the blood that participates in the immune response to pathogens and tissue inflammation. It is a sensitive but non-specific marker of infection or inflammation occurring at some site in the body. It is not specific for any diagnosis, but its elevation can be a 'flag'.

[11] An antibiotic with action against bacterial agents that can cause gastroenteritis including *E. coli*, salmonella species, Shigella species and campylobacter jejuni.

stool sample from 3 October 2014 was positive for cryptosporidia species.[12] The plan by the medical team was to discuss this with the Monash Infectious Diseases team the following day.

8. On the afternoon and evening of 5 October 2014 it was also noted on blood tests that Child A was hyponatremic[13] with a sodium level of 125 millimole per litre (mmol/L) and a plan for treatment and monitoring of this was instituted by the Paediatric House Medical Officer (HMO) and Registrar in discussion with the on call Paediatrician Dr Ranaweera. This involved intravenous fluid treatment with 0.9 percent saline and 5 percent dextrose at maintenance rate,[14] and four hourly blood tests to check sodium levels.

9. Overnight on 5 October 2014 to 6 October 2014 Child A had two coffee coloured vomits and ongoing abdominal pain despite treatment with buscopan.[15] However, his diarrhoea appeared to be improving. The nursing staff notified the overnight paediatric registrar of the vomiting and he was reviewed in the early morning on 6 October 2014 by the paediatric registrar. He was noted to have a stable blood pressure and heart rate, generalised tenderness on examining his abdomen, but no signs of guarding or rigidity[16]. The paediatric registrar noted that his hyponatremia had been stable overnight, and that his sodium had continued to be low at 124mmol/L and 123mmol/L, but that he had a normal neurological state. At this point his treatment with ondansetron, buscopan and paracetamol for symptom relief was continued.

10. Child A was reviewed on 6 October 2014 on the morning ward round by Paediatrician Dr Peter Francis and assessed to be stable. The result of cryptosporidium in the stool test was noted. There was no comment in the medical note at this review of Child A's abdominal examination or symptoms of blood stained stool or coffee ground vomiting. On review of the fluid balance chart, Child A's diarrhoea had improved, his weight had increased by 1.1kg and he continued to pass urine, although the exact amounts were not measured. The plan from Dr Francis was to continue with supportive management and take blood tests two times per day.

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[12] Cryptosporidium is a parasite that is associated with gastrointestinal diseases. The main sources of infection are contaminated water sources and food.

[13] A low blood sodium level. The normal range is between 135 and 145 mmol/L.

[14] Rate of intravenous fluid administration required to maintain hydration in a fasted child without significant fluid losses.

[15] A medication that is an antispasmodic, used to treat cramping abdominal pain.

[16] A clinical sign of peritoneal inflammation in which the abdominal muscles are contracted such that the abdomen feels hard on examination.

11. The nursing notes indicate that up until 2.30pm on 6 October 2014, Child A had ongoing abdominal pain, diarrhoea and coffee coloured vomiting despite ondansetron. The nurse discussed Child A's progress with the Nurse Unit Manager (NUM) and the paediatric registrar.

12. Over the course of the afternoon of 6 October 2014, Child A developed worsening coffee-ground vomiting and in the evening had frank blood in his vomits. He also had worsening diarrhoea with fresh blood noted in his stools from 3.30pm onwards. He was reviewed at 5.30pm by the Paediatric HMO and Registrar when he was found to have a low blood pressure and elevated heart rate on his observation chart. He was treated with intravenous fluids with mild improvement. At this time a note was made of results of the repeat electrolyte check at 12:53pm on 6 October 2014, which demonstrated a worsening sodium level of 121mmol/L and a newly abnormal urea (7.6mmol/L) and creatinine (68umol/L) indicating an acute kidney injury[17].

13. Child A continued to look unwell despite intravenous (IV) fluids and pain relief. At 6.30pm the Paediatrician Dr Francis reviewed Child A and noted that his abdomen was tender with guarding and rebound tenderness. Dr Francis assessed Child A as possibly having intussusception[18] with ischaemic bowel[19] and evolving shock[20]. He documented a risk of multiorgan failure and hyponatraemia. Child A was commenced on IV ceftriaxone[21], and his transfer to Monash Medical Centre (MMC) was arranged on discussion with the Paediatric Surgical Registrar and the Paediatric ED Consultant at the MMC. Transfer occurred at 8.40pm that night by Mobile Intensive Care Ambulance (MICA).

14. Child A arrived at MMC at 9.23pm on 6 October 2014. In the ED he was assessed as being very unwell. He was treated with further intravenous fluids and antibiotics and was admitted to the paediatric Intensive Care Unit (ICU). Child A was found to have worsening acute kidney injury (AKI) necessitating dialysis, no urine output and signs of haemolysis on his blood tests raising the possibility of Haemolytic Uraemic Syndrome (HUS). He had a low haemoglobin and platelet count. However, given Child A's family are Jehovah's Witness, transfusion of blood products was initially delayed until deemed absolutely necessary by medical staff. A diagnostic

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[17] Acute loss of kidney function resulting in the retention of urea and other nitrogenous waste products and in the dysregulation of extracellular volume and electrolytes.

[18] Intussusception is the invagination (telescoping) of a part of the intestine into itself causing a bowel obstruction.

[19] Condition where the blood supply to the bowel is compromised.

[20] Life threatening circulatory failure.

[21] An intravenous broad spectrum antibiotic.

laparotomy was performed on 7 October 2014 demonstrating the entire large bowel to be affected by haemorrhagic colitis. No perforation was noted. An ileostomy was performed and a Permacath[22] inserted for haemodialysis. He received a platelet transfusion prior to surgery.

15. Child A remained in the ICU; he was intubated, ventilated and sedated with midazolam and fentanyl. He developed bilateral pleural effusions, with a right sided intercostal catheter inserted for drainage on 8 October 2014. A red blood cell transfusion was administered at this time. Child A required a noradrenaline infusion for low blood pressure and a sodium bicarbonate infusion for significant metabolic acidosis. He had an echocardiogram on 9 October 2014 demonstrating a normal heart structure and reduced contraction of the septum but otherwise normal function and a small pericardial effusion. Child A continued to be anuric[23] with worsening kidney function and received haemodialysis. He was treated with intravenous tazocin[24] for possible sepsis. Blood culture, stool culture and peritoneal fluid[25] cultures taken at Monash Medical Centre were negative apart from cryptosporidium and enterovirus[26] being detected in the stool.

16. Child A was reviewed by the vascular team and plastic surgery team for concerns regarding possible compartment syndrome[27] in the lower limbs or abdomen but these possible diagnoses were not found. He was commenced on parenteral nutrition[28] while unable to tolerate enteral nutrition and he continued to have watery bleeding from his rectum which was not associated with a significant decrease in haemoglobin. Child A's condition appeared to be stable and on 12 October 2014 sedation was weaned to prepare for extubation.

17. Child A's condition unexpectedly deteriorated after midnight on 13 October 2014. His blood pressure dropped, despite increasing inotropic support and intravenous fluid bolus. Following cessation of haemodialysis he deteriorated further with increasing abdominal distension and a drop in his heart rate and blood pressure. Cardiopulmonary resuscitation was promptly commenced and Child A received multiple doses of adrenaline, intravenous fluid boluses, blood

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[22] A central venous catheter used in dialysis.

[23] Absence of urine output.

[24] A broad spectrum intravenous antibiotic.

[25] Fluid from the peritoneal cavity.

[26] Enterovirus is a group of viruses which can occur in the gastrointestinal tract.

[27] Occurs when increased pressure within a compartment compromises the circulation and function of the tissues within that space.

[28] Provision of calories, amino acids, electrolytes, vitamins, minerals, trace elements, and fluids via a route other than the digestive tract, for example, the intravenous route.

transfusion and further inotropic support. Child A had further cardiac arrests resistant to prolonged resuscitation attempts and died at 4.30am on 13 October 2014.

STANDARD OF PROOF

18. All coronial findings must be made based on proof of relevant facts on the balance of probabilities. In determining whether a matter is proven to that standard, I should give effect to the principles enunciated in *Briginshaw v Briginshaw* (1938) 60 CLR 336. These principles state that in deciding whether a matter is proven on the balance of probabilities, in considering the weight of the evidence, I should bear in mind:
    - the nature and consequence of the facts to be proved;
    - the seriousness of an allegation made;
    - the inherent unlikelihood of the occurrence alleged;
    - the gravity of the consequences flowing from an adverse finding; and
    - if the allegation involves conduct of a criminal nature, weight must be given to the presumption of innocence, and the court should not be satisfied by inexact proofs, indefinite testimony or indirect inferences.

19. The effect of the authorities is that coroners should not make adverse findings against or comments about individuals, unless the evidence provides a comfortable level of satisfaction that they caused or contributed to the death.

INVESTIGATIONS

Forensic pathology investigation

20. Dr Sarah Parsons, Forensic Pathologist at the Victorian Institute of Forensic Medicine performed a full post mortem examination upon the body of Child A, reviewed a computed tomography (CT) scan and e-medical deposition from Monash Medical Centre, and referred to the Victoria Police Report of Death, Form 83.

21. Dr Parsons opined that the sequence leading to Child A's death and the laboratory findings prior to his death, were consistent with haemolytic uraemic syndrome secondary to *E. coli* infection. Haemolytic uraemic syndrome is a severe complication of *E. coli* infection. Post diarrhoeal haemolytic uraemic syndrome is signified as D+HUS. Thrombotic thrombocytopenic purpure (TTP) and HUS were described as distinct clinical entities, however they are mainly described together on the spectrum of Thrombotic Microangiopathies.

22. Dr Parsons noted that a histology review by a paediatric pathologist had diagnosed gross and histological features most in keeping with thrombotic microangiopathy associated with extensive renal infarction and bowel necrosis. Dr Parsons reported that *eaeA* and *stx* genes were detected in an ante mortem stool sample provided on 5 October 2014; these two genes can be seen in haemolytic uraemic syndrome. The histological features seen at autopsy were also typical of a diagnosis of D+HUS.

23. Cryptosporida was detected in post-mortem bowel contents, while Epstein-Barr virus was identified in lung and abdominal fluid. Dr Parsons noted that both *E. coli* and cryptosporidium can be found in cases of unpasteurised milk, but can also be seen from other sources. The role of cryptosporidium in contributing to the significant pathology identified in this case was unclear.

24. Toxicological analysis of post mortem specimens identified drugs administered by medical staff; fentanyl,[29] midazolam,[30] lignocaine[31] and paracetamol.[32]

25. Dr Parsons reported that Mountain View Bath Milk with a use by date on the cap of 5 October 2014, was tested at Monash Pathology on 10 October 2014. While *E. coli* was not detected in this sample, Dr Parsons noted that Child A had been experiencing symptoms for a week prior to his admission on 4 October 2014.

26. Following the review by a second pathologist, Dr Parsons ascribed the cause of Child A's death to thrombotic microangiopathy.

Police investigation

27. Detective Senior Constable Kerryn Merrett, the nominated coroner's investigator,[33] conducted an investigation of the circumstances surrounding death, at my direction, including the preparation of the coronial brief. The coronial brief contained, *inter alia*, statements made by Child A's father, General Practitioner at Medicentre Clinic Dr Subodhani Sirisena, Consultant Paediatrician at Frankston Hospital Dr Peter Francis, Intensive Care Specialist at Monash

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[29] Fentanyl is a narcotic (opioid analgesic) used as perioperative analgesic and as an adjunct to surgical anaesthesia.

[30] Midazolam is a short acting benzodiazepine used intravenously in intensive care patients.

[31] Lignocaine is a local anaesthetic often administered to patients prior to surgery or during resuscitation attempts.

[32] Paracetamol is an analgesic drug available in many proprietary products either by itself or in combination with other drugs such as codeine and propoxyphene.

[33] A coroner's investigator is a police officer nominated by the Chief Commissioner of Police or any other person nominated by the coroner to assist the coroner with his/her investigation into a reportable death. The coroner's investigator takes instructions direction from a coroner and carries out the role subject to the direction of a corner.

Medical Centre Dr Paul Ritchie and Manager of Communicable Disease Prevention and Control at the Department of Health and Human Services (DHHS) Philip Clift.

28. Child A's father stated that a naturopath had assessed his son as intolerant to dairy, gluten and eggs in around June 2014. The family had subsequently changed his diet and he appeared to sleep better and have more energy. In the 12 months prior to Child A's death, his parents had been buying fresh, largely organic produce from several locations.

29. Child A's father reported that they went to a lot of trouble to get milk, purchasing unpasteurised Mountain View Organic Dairy milk from a store on the Mornington Peninsula over the two or three months before October 2014. He reported that they understood the milk was labelled as not to be drunk, but stated he would be surprised if anyone used it for cosmetic purposes. He described the milk as packaged in a two litre plastic container, and said it looked like every other milk container. Child A's parents would use it in their tea and drink it weekly. Child A's father said they thought it might be easier for Child A to drink, and occasionally they would give him a small amount of the milk with his formula. Child A's father stated that this occurred a maximum of twice per month, and it would have been only one eighth of a sippy cup. Their other son never drank the milk. When Child A became ill, his father stated that he could not remember if his son had drunk any of the milk in the bottle they currently had in the fridge at their home. There was a possibility that he had, but also a chance he had not because he so rarely drank milk. The bottle would have been purchased on the Friday or weekend a week before Child A fell ill.

30. According to Child A's father, other possible sources of illness included his recent visit to Frankston beach and in a storm water drain, as well as a recent child's party where he had eaten some food containing dairy, gluten and eggs. However, no other children were known to have become ill following the party.

31. Philip Clift, the DHHS Manager of Communicable Disease Prevention and Control, reported that the Department was initially notified of Child A's death on 23 October 2014, after a Microbiology Registrar at MMC had contacted the Microbiological Diagnostic Unit. Child A was suspected of having Haemolytic Uraemic Syndrome and Cryptosporidiosis, and they are both notifiable conditions under the *Public Health and Wellbeing Act 2008* (Vic) and *Public Health and Wellbeing Regulations 2009* (Vic).

32. Mr Clift stated that cases of similar illness, identified as having consumed the same brand of unpasteurised milk (Mountain View Organic Dairy Bath Milk) were considered to be linked for

the purpose of this outbreak, and no other common factor was identified. Mr Clift reported that the outbreak resulted in three cases of HUS, and two of cryptosporidium; all five cases involved the drinking of Mountain View Organic Dairy brand unpasteurised milk.

33. Mr Clift reported that the DHHS arranged for the testing of 39 samples of one and two litre bottles of Mountain View Organic Dairy Bath Milk in November 2014. A shiga toxin producing *E. coli* was cultured from one sample of the milk.

34. Mr Clift stated that Dairy Food Safety Victoria (DFSV) is the independent regulator of Victoria's dairy industry. DFSV had advised the DHHS that as of December 2014 it was aware of four dairy industry licence holders distributing unpasteurised milk labelled as 'bath milk' and 'not for human consumption' and one individual selling the product without a licence. Mr Clift stated that DFSV believed that all licence holders had ceased to distribute unpasteurised milk labelled as not for human consumption, and investigations were continuing in relation to the person alleged to be distributing it without a dairy industry licence.[34]

35. Mr Clift reported that on 2 December 2014, the former Chief Health Officer issued a health advisory to health professionals and consumers, warning against consumption of raw milk products sold for cosmetic applications. This was followed by a media release on 11 December 2014, again warning consumers of the dangers of drinking unpasteurised milk. In addition, the Chief Health Officer wrote to Consumer Affairs Victoria and the Australian Competition and Consumer Commission regarding concerns about the issues surrounding misleading labelling.

36. Following consultation with DFSV, Mr Clift noted that DFSV licence holders producing dairy product not for human consumption were not considered to be in breach of legislation, licence conditions, the Code of Practice or the Food Standards Code at the time of the investigation of illnesses in late 2014, and as such no prosecution of these producers was contemplated. Specifically, the relevant local council investigated the sale of Mountain View Organic Dairy bath milk on the Mornington Peninsula and no breaches of the Food Act or the Food Standards Code were identified.

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[34] I note that Mr Clift's statement was dated 3 June 2015.

Victorian Department of Health and Human Services Outbreak Investigation Summary Report – Haemolytic Uraemic Syndrome (HUS) 2014

37. The Victorian DHHS released an Outbreak Investigation Summary Report dated 18 June 2015. The report detailed that the overall evidence linking the consumption of Mountain View Organic Dairy Bath Milk with three HUS cases (including Child A) in 2014 was:

    - Haemolytic uraemic syndrome is an extremely rare condition. The five yearly average (2009 to 2013) of HUS cases notified in Victoria was three cases per year and in 2014, five cases were notified. Two cases notified on the same day (the DHHS was notified of two HUS cases, including Child A on 23 October 2014) was an exceptionally rare occurrence, indicating a potential point source exposure;
    - Consumption of unpasteurised milk is a rare exposure. Information obtained from a recent food frequency survey, conducted between November 2014 and January 2015 revealed that 1.9% of children aged less than five years reported consumption of unpasteurised cow's milk in the seven days prior to interview;
    - Two of the HUS cases (including Child A) had an onset of illness within three days of each other, suggesting a common source exposure;
    - There were no other common risk factors which could be identified for the three HUS cases;
    - Shiga toxin producing *E. coli* was isolated from a sample of Mountain View Organic Dairy Bath Milk obtained from the marketplace in early November;
    - An stx 2 gene was detected in a faecal specimen for one of the HUS cases; and
    - Two cases of cryptosporidiosis living in the same geographical area as the HUS cases also reported consumption of Mountain View Organic Dairy Bath Milk prior to the onset of their diarrhoea, which was within a 10 day period of onset date for two of the HUS cases, reinforcing the evidence of a common exposure.

38. The report noted that the consumption of unpasteurised milk is a well-documented risk factor for gastrointestinal diseases including Shiga toxin producing *E. coli* infection and was the suspected source of illness in this outbreak. It was also noted that two cases of cryptosporidiosis in 2014 also identified consuming Mountain View Organic Dairy Bath Milk in their incubation period, which further supported this conclusion. It was noted that the cause of Child A's death was 'thrombotic microangiopathy', which is a feature of HUS.

39. The report noted that in addition to the action taken by the DHHS, the Mountain View Organic Dairy conducted a product recall of its bath milk in one and two litre containers, on 12 December 2014. The product recall was conducted under the Australian Consumer Law which is overseen by the Australian Competition and Consumer Commission (ACCC).

40. In addition, it was noted that on 22 December 2014, a joint meeting was held between the DHHS, Department of Environment and Primary Industry (now the Department of Economic Development, Jobs, Transport and Resources) and Victoria's dairy regulator, DFSV. At the meeting, DFSV advised that it would impose licence conditions on Victorian dairy producers to ensure that cosmetic dairy products, and other dairy products not for human consumption, be treated so as to deter human consumption and such that the milk or milk product could not reasonably be mistaken as being for human consumption. It was reported that this new licence condition came into effect on 1 February 2015.

41. Forensic Pathologist Dr Parsons provided a supplementary report dated 18 February 2016. In the report, Dr Parsons noted that she had read the DHHS Outbreak Investigation Summary Report, but had nothing further to add. Dr Parsons opined that the report made a good case that the likely source of the shiga toxin is the milk; the cryptosporidium however was less convincing.

Family concerns

42. In his statement, Child A's father noted a number of concerns about his son's medical management.

43. At the presentation to Frankston Emergency Department when Child A was admitted, he reported that his son was initially going to be discharged:

> *'My wife and I refused to let them send us home. We knew he was sick and we insisted that he was admitted.'*

44. Child A's father reported that his son had worsening blood stained diarrhoea and abdominal pain but that his treatment was unchanged.

> *'I felt like they were still treating Child A like a severe case of gastro. All they wanted to do was get fluids into him and get him to start eating.'*
>
> *'The nurses would listen to me tell them about the blood. They would listen but it was like they didn't feel it was serious.'*

'Frankston Hospital didn't seem to treat Child A seriously enough when we first arrived. If he had had the surgery or been given the right antibiotics then I feel like this could've been fixed and we would still have our son.'

45. Child A's father also expressed concern that his son was not transferred to the Monash Medical Centre earlier.

'They (nursing staff) seemed shocked that Child A had not been transferred earlier. I remember that well because it was exactly what I had been thinking.'

Coroners Prevention Unit review

46. Following my reading of the coronial brief and the family's concerns, I asked the Coroners Prevention Unit (CPU)[35] to review the circumstances surrounding Child A's death, in particular relating to the medical management by his General Practitioner and Frankston Hospital. The CPU reviewed Child A's medical records at Frankston Hospital and Monash Medical Centre Clayton, as well as a statement from Dr Kathy McMahon, Clinical Director of Paediatrics at Frankston Hospital, dated 12 May 2016.

Haemolytic Uraemic Syndrome[36]

47. The review noted that Haemolytic Uraemic Syndrome (HUS) is defined by the simultaneous occurrence and sudden onset of three features: microangiopathic haemolytic anaemia[37], thrombocytopaenia[38] and acute kidney injury[39]. These three features are detectable on blood tests. Shiga toxin-producing *E. coli* HUS is the most common cause of paediatric HUS, accounting for 90 percent of cases and primarily affects children under five years of age. Children typically have a prodromal illness with abdominal pain, vomiting and diarrhoea that

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[35] The Coroners Prevention Unit (CPU) was established in 2008 to strengthen the prevention role of the coroner. The unit assists the coroner with research in matters related to public health and safety and in relation to the formulation of prevention recommendations, as well as assisting in monitoring and evaluating the effectiveness of the recommendations. The CPU comprises a team with training in medicine, nursing, law, public health and the social sciences.

[36] Calderwood, S. 'Clinical manifestations, diagnosis and treatment of enterohemorrhagic Escherichia coli (EHEC) infection'. Accessed 18 May 2016. Available on UpToDate.com; Niaudet, P. 'Overview of haemolytic uremic syndrome in children'; 'Clinical Manifestations and diagnosis of Shiga toxin-producing Escherichia coli haemolytic uremic syndrome in children'; 'Treatment and Prognosis of Shiga toxin-producing Escherichia coli haemolytic uremic syndrome in children'. Accessed 24 April 2016. Available on UpToDate.com

[37] Reduced circulating red blood cells due to destruction of red blood cells and associated abnormalities in the microvasculature (small blood vessels).

[38] Low platelet count.

[39] Acute loss of kidney function resulting in retention of urea and other nitrogenous waste products and in the dysregulation of extracellular volume and electrolytes.

generally precedes HUS by five to 10 days. In the majority of cases of Shiga toxin-producing Escherichia coli (STEC) infection, spontaneous resolution occurs. Enterohaemorrhagic *E. coli* (EHEC) are strains capable of producing Shiga toxin. HUS complicates six to 9 percent of EHEC infections overall and 15 percent of EHEC infections in children under age 10 years.

48. Other organ systems can be affected by HUS. Manifestations in the central nervous system include seizures, coma, stroke, weakness and blindness. In the gastrointestinal tract, severe haemorrhagic colitis[40], bowel necrosis[41], peritonitis[42] and intussusception[43] may occur. Cardiac dysfunction due to cardiac ischaemia[44] can arise. Transient diabetes mellitus[45], deranged liver function and liver enlargement can also occur.

49. The diagnosis of HUS is made on the basis of these characteristic clinical and laboratory findings. Detection of the STEC infection can be conducted from stool testing, including stool culture and testing for Shiga toxins. However, this can be unreliable because the bacteria are only present in the stool for a few days and, even if present, may not be detected by culture from stool samples.

50. The CPU noted that there is no curative treatment for HUS and management is usually supportive care. Blood transfusions for anaemia, platelet transfusions pre-procedure or for active bleeding, and dialysis therapy as indicated for acute kidney injury are the mainstays of management. Treatment also involves careful monitoring of fluid input and output, management of high blood pressure with antihypertensives,[46] and management of seizures with antiepileptic medications. Serial abdominal examinations should be conducted as severe colitis can lead to intestinal perforation requiring surgical intervention and parenteral nutrition[47] may also be required.

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[40] Bleeding and inflammation of the bowel.

[41] Death of cells in the bowel.

[42] Inflammation (irritation) of the peritoneum, the thin tissue that lines the inner wall of the abdomen and covers most of the abdominal organs.

[43] Invagination of a part of the intestine into itself.

[44] Reduced blood supply to the heart muscle.

[45] A condition of impaired carbohydrate metabolism resulting in high blood glucose.

[46] Medications used to lower blood pressure.

[47] Provision of calories, amino acids, electrolytes, vitamins, minerals, trace elements, and fluids via a route other than the digestive tract, for example, the intravenous route.

51. There is no known effective therapy to prevent progression from the bloody diarrheal acute infectious phase to the post diarrheal phase of HUS. Antibiotics and antimotility drugs[48] do not reduce the progression to HUS due to STEC infections.

52. The mortality rate in HUS is less than five percent, however another five percent have significant sequelae such as stroke or end stage kidney disease. In those who recover from the original illness, the risk of ongoing kidney disease exists and long term follow up and monitoring is recommended.

53. The review identified that Enterohemorrhagic E coli (EHEC) is the most common cause of STEC HUS, with main vectors being cattle as it is present in cattle intestine and faeces. Infection in humans occurs following ingestion of contaminated undercooked meat, unpasteurised milk or milk products, contaminated water, fruits or vegetables. Secondary human to human transmission is also possible. Prevention of HUS is dependent on measures that decrease the risk of infection, for example, proper cooking of foods, adequate food storage and preparation practices, and avoidance of unpasteurised dairy products.

Cryptosporidium infection[49]

54. The CPU noted that cryptosporidium is a parasite that is associated with gastrointestinal diseases. The main sources of infection are contaminated water sources and food. It can cause an asymptomatic infection, a mild diarrhoeal illness, or a severe enteritis with or without biliary tract[50] involvement. The incubation period is usually seven to 10 days but can range up to 28 days. Patients who develop diarrhoea can also have nausea, poor appetite, abdominal pain and low-grade fever. However, blood in the faeces is rare unless there is co-infection with another enteric pathogen. Chronic diarrhoea can also develop. The illness usually resolves without therapy in 10 to 14 days in immunologically healthy people, however if symptoms persist then treatment with an antimicrobial agent may be necessary.

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[48] Medications used to alleviate the symptoms of diarrhoea, for example, loperamide is a medication that inhibits intestinal peristalsis.

[49] Up To Date articles: Epidemiology, clinical manifestations and diagnosis of cryptosporidiosis; Treatment and prevention of cryptosporidiosis.

[50] The pathway by which bile is secreted by the liver then transported to the small intestine.

Review of Health Care Diagnosis, Treatment and Follow Up

55. The review noted that nausea, vomiting, diarrhoea and cramping abdominal pain is a common presentation for acute gastroenteritis[51] in the paediatric population. The most common cause of acute gastroenteritis in children in developed countries is viral pathogens, but bacterial and parasitic enteritis can also occur. Acute viral gastroenteritis in a child who is not immunocompromised[52] is managed symptomatically with maintenance of hydration.

56. The CPU reported that the assessments and management by the two general practitioners who reviewed Child A on 30 September 2014 and 2 October 2014 were appropriate. Child A was presenting with symptoms and signs of an acute gastroenteritis and he was adequately maintaining his hydration.

57. In addition, the review assessed that Child A's management at Frankston Hospital on 4 October 2014 was appropriate. Child A presented with ongoing symptoms of vomiting and diarrhoea and now had blood and mucous in his stool. Blood and mucous can be present in viral gastroenteritis but is more common in bacterial or parasitic gastroenteritis. For many forms of bacterial gastroenteritis in the immune-competent host the illness is still self-limiting and does not require treatment with antibiotics. Thus it was reasonable to admit Child A for observation without antibiotics but continue supportive care with anti-nausea medication and monitoring of fluid intake. At this point, a stool sample had already been sent by the GP for microscopy and culture to detect bacterial or parasitic pathogens.

58. The review posited that when Child A's symptoms did not improve on 5 October 2014, the paediatric team appropriately escalated care and investigated further, ordering a repeat stool sample, performing blood tests and inserting an IV cannula for fluids. Further expert opinion was sought with Child A's case being discussed with the Infectious Diseases Consultant at the MMC and antibiotic therapy appropriate for treatment of bacterial gastroenteritis being commenced. The abdominal examinations documented on this day indicated a tender abdomen but no signs of peritonitis which would prompt a surgical review and thus earlier transfer to MMC was not indicated at this stage.

59. Child A was also found to have a low blood sodium level, which the review identified can occur in dehydration and gastroenteritis. According to the CPU, appropriate assessment and

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[51] Acute gastroenteritis is a clinical syndrome defined by increased stool frequency with or without vomiting, it can be caused by a viral pathogen or bacteria or parasites.

[52] Immune system suppressed or weakened, for example, by illness or medications.

management plans were instituted that were in keeping with the treatment of hyponatraemia recommended in the Royal Children's Hospital Clinical Practice Guidelines[53]. However, the CPU noted that the frequent monitoring of sodium levels required was challenging due to difficulty obtaining blood samples from Child A.

60. The initial assessment and management of Child A's small coffee coloured vomits was reasonable given he continued to have normal vital signs, and on examination a soft abdomen, reduced diarrhoeal output and ongoing urine output. Coffee-ground vomits can be indicative of upper gastrointestinal tract bleeding the cause of which in a child with this history would most likely be a small tear in the oesophagus from forceful retching, or inflammation of the oesophagus or stomach lining from recurrent vomiting. These causes can be self-limited and thus observation is a reasonable action.

61. At the time of Consultant review on the morning ward round on 6 October 2014, Child A appeared to have improving diarrhoea and continued to have normal vital signs. The plan to continue supportive care was appropriate in this setting. The CPU was unable to comment on whether signs of peritonism[54] were present at this time, given the absence of documentation of an abdominal examination at this review. However, it was noted that the documentation of a non-peritonitic abdominal examination at 5.30pm that day by the Paediatric Registrar would suggest that signs of peritonism were not present at the earlier Consultant review.

62. The review noted that during the afternoon of 6 October 2014 the nursing staff appropriately escalated the change in Child A's symptoms to the Paediatric Registrar. The Paediatric Registrar reviewed Child A at 5.30pm at which time Child A had developed cardiovascular instability. The Paediatric Registrar appropriately escalated to the Paediatric Consultant. The Paediatric Consultant then attended the hospital at approximately 6.30pm when initial management with IV fluids and pain relief failed to improve Child A's condition. The Paediatrician then appropriately arranged transfer to MMC for surgical review.

63. The CPU assessed that in retrospect, an earlier Paediatric Registrar review in the setting of worsening vomiting and changes in the stool on the afternoon of 6 October 2014 would have been desirable. However, it was noted that this may not have significantly changed Child A's management given he continued to have vital signs within the normal range until 5.20pm on 6

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[53] RCH CPG 'Hyponatraemia' available on: http://www.rch.org.au/clinicalguide/guideline_index/Hyponatraemia/ Accessed on 17 May 2016.

[54] Signs of peritonitis.

October 2014, at which time he was promptly reviewed in response to an elevated heart rate. At the time of that review the paediatric registrar noted a non peritonitic abdominal examination and it was not until his condition continued to worsen at 6.30pm that the Paediatric Consultant found Child A to exhibit signs of peritonism on abdominal examination, prompting transfer to the MMC for surgical care.

64. In her statement, Frankston Hospital Clinical Director of Paediatrics Dr McMahon identified that Child A's care management was escalated to the consultant multiple times during his admission, and that consultation with expertise at the MMC was sought from the second day of admission. Dr McMahon also provided the Peninsula Health guideline for 'Recognising and Responding to the Deteriorating Patient' and the guideline for 'Medical Emergency Team Response'. The CPU reported that these guidelines were both followed in this case.

65. Dr McMahon identified that Child A had difficult IV access and frequent blood sampling for monitoring of hyponatraemia was difficult, with some samples being haemolysed and unsuitable for testing. She identified that the initial abnormal sodium result had been phoned by pathology staff to the ward. She also discussed that the change in urea and creatinine noted on 6 October 2014 was consistent with dehydration, for which the treatment with IV fluids was appropriate.

66. Dr McMahon discussed that blood and mucous stained stools are a common finding in bacterial gastroenteritis and that treatment for this in Child A's case had been discussed with the Infectious Diseases Consultant at MMC. She also discussed that the coffee ground vomits were initially thought to be secondary to a Mallory Weiss tear but when Child A became more unwell, the coffee ground vomiting was thought to be caused by a possible bowel obstruction, given the changing clinical picture.

67. Child A's case was presented and reviewed at the Frankston Hospital Paediatric transfer meeting and Morbidity and Mortality meeting on 25 November 2014, a meeting attended by Paediatricians, Registrars, HMOs and nursing staff. No issues were identified at this meeting. Dr McMahon comments that:

> *"On review it was thought that Child A's medical treatment at Frankston Hospital was appropriate. He received supportive therapy with intravenous fluids (normal saline) for initial presumed gastroenteritis. His treatment was discussed with an infectious disease consultant who suggested antibiotics although usually gastroenteritis is treated conservatively with oral rehydration and no antibiotics. The hyponatraemia was identified although bloods were not analysed as frequently as wanted as many samples*

were spoiled. Child A had difficult venous access. He was transferred when his condition deteriorated. Unfortunately there is no treatment for HUS except supportive treatment."

68. The CPU did not identify any issues with the care provided at MMC Clayton. Child A had multiple teams involved in his care and was appropriately managed in the ICU. The CPU assessed that his deterioration was unexpected and rapid, and that he died despite extensive resuscitation attempts.

Mention Hearing on 27 June 2016

69. A Mention Hearing was held on 27 June 2016, in order to progress my investigation, and enable parties to raise any further matters that might warrant the holding of an Inquest, or alternatively an in-chambers Finding.

70. In advance of the Mention Hearing, I sought information from Dairy Food Safety Victoria (DFSV). By way of email dated 15 June 2016, MinterEllison lawyers provided information on behalf of Jennifer McDonald, Chief Executive Officer for DFSV.[55] It was noted that DFSV is a statutory authority that reports to the Victorian Minister of Agriculture and is responsible for regulating the Victorian dairy industry. DFSV licences all dairy businesses operating in Victoria, including dairy farmers, manufacturers, carriers and distributors, and approves and monitors compliance with dairy food regulatory requirements.

71. It was noted that the *Australia New Zealand Food Standards Code* requires that milk for human consumption be pasteurised, or treated in a manner which is lethal to pathogenic organisms to an equivalent degree to pasteurisation.[56] The basis for this requirement was not that all raw milk is unfit for all human consumption, but that the potential risks posed by allowing the sale of raw milk for human consumption are unacceptable.

72. DFSV were of the view that dairy milk is to be considered as sold for human consumption, unless it has been transformed so that it is no longer a substance of a kind used for food. In DFSV's view, changes to labelling and shelf placement are not sufficient to transform dairy milk so that it is no longer a substance of a kind used for food. It was explained that DFSV was hence of the view that dairy milk should not be sold to the public unless it has been pasteurised;

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[55] I note that the information was provided in the format of a drafted, unsigned and undated statement as Ms McDonald was overseas during this period.

[56] See Clause 15 of Standard 4.2.4

treated in a manner which is lethal to pathogenic organisms to an equivalent degree to pasteurisation; or transformed so that it is no longer a substance of a kind used for food.

73. It was confirmed that in late 2014, in response to publicity about intentional consumption of raw milk and public statements issued by the then Victorian Chief Health Officer linking instances of serious illness to raw milk, DFSV clarified its approach to monitoring and ensuring compliance with food regulatory requirements. A licence condition attaching to dairy industry licences was introduced requiring dairy farmers or producers intending to sell any dairy product not intended for human consumption (and not produced in accordance with food regulatory requirements) to: advise DFSV of its intention; and seek DFSV's approval of the process by which the producer would ensure that the dairy product was not able to be consumed or mistaken for food (such as by adding a colouring or bitter agent). It was noted that DFSV had received no requests for approval to produce 'bath milk' under these licence conditions, and does not have any evidence that 'bath milk' is currently available for sale from dairy farms, markets or retail outlets.

74. At the Mention Hearing, Ms Rose Raniolo, appearing on behalf of Mountain View Farm Pty Ltd, noted that the Court had not obtained the DHHS HUS questionnaire, which was completed by Child A's family. Ms Raniolo contended that the linking of unpasteurised milk to Child A's ill health was inappropriate in light of the rare occasions on which he drank it, and without knowing what other possible exposures he had. I asked Ms Raniolo to provide the Court with any further material supporting her submissions.

Submissions on behalf of Mountain View Farm Pty Ltd

75. The Court received submissions from Ms Raniolo on behalf of Mountain View Farm Pty Ltd, dated 4 July 2016. The submissions incorporated a report by microbiologist Ronald R. Hull dated 30 June 2016, and noted that an Inquest would not be in the public interest or desirable given the distress it would be likely to cause Child A's family. However, Ms Raniolo contended that the coronial investigation was incomplete and has not produced information which could lead me to find that Child A's illness and death were caused by the consumption of unpasteurised milk.

76. Mr Hull reviewed the coronial brief on behalf of Mountain View Farm Pty Ltd and formed the opinion that Child A suffered from multiple primary infections of the gastrointestinal tract progressing unchecked to fatal secondary infections of vital organs and that the primary infections did not arise from drinking unpasteurised milk. Mr Hull concluded that Child A

suffered multiple gastrointestinal infections of Cryptosporidium, Adenovirus and Shiga toxin producing *E. coli* which likely proceeded in sequence as his immune-competence decreased. Mr Hull was of the view that the likely source of the Cryptosporidium and the Shiga toxin producing *E. coli* were the brackish water at the beach or in the case of the Shiga toxin, hospital acquired (nosocomial) and he reported that the Adenovirus is spread by person to person contact.

77. Ms Raniolo referred to the leading authoritative case of *Briginshaw v Briginshaw* (1938) 60 CLR 336,[57] and submitted that I must be actually persuaded that Child A's death was a consequence of his having drunk unpasteurised milk having regard to the serious consequences of such an adverse finding to Mountain View Farm. Ms Raniolo submitted that without an examination of the material that the DHHS report was reliant upon, including *inter alia* the pathology reports of faecal testing performed on Child A's stools and the HUS questionnaire completed by Child A's parents, I would be effectively adopting the conclusions of the DHHS and could not be reasonably satisfied.

78. In particular, Ms Raniolo submitted that the DHHS report was premised on a 'clearly inaccurate and unsupported belief that Child A had been drinking the milk for 1-2 weeks prior to becoming ill'. Ms Raniolo noted that Child A's father listed Mountain View milk on paperwork at Frankston Hospital in October 2014 and later stated 'I could not remember, and either could Child A's mother, as to when Child A had last drunk the milk or if he'd had any from the bottle that we currently had in the fridge at home. There is a possibility that he had, but also a chance he had not because he rarely still drank milk at all.'

79. Ms Raniolo submitted that other possible sources, such as exposure to a public swimming pool, a stormwater outlet, and a child's party, were not considered by the DHHS. On this issue, Mr Hull opined that brackish-water is a well-recognised source of Cryptosporidium and Shiga toxin E.coli. It was his opinion that 'the most common route of infection in HUS by *E. coli* is via the urethra' and was likely to have originated while Child A played in the brackish water.

80. Ms Raniolo also noted that pathology testing of Child A's faecal stools requested on 2 October 2014, had not been produced to the Court, and should be obtained and reviewed to see whether it tested positive for Shiga toxin E.coli, or just Cryptosporidium only. Ms Raniolo submitted that testing undertaken by the Microbiological Diagnostic Unit, which indicated direct DNA extraction from one of three faecal specimens collected from Child A was positive for stx 2

[57] See coronial application in *Thales Australia Ltd v Coroners Court (Vic)* (2011) VSC 133.

eaeA and ehxA consistent with STEC infection, was not accredited or verified for use on human samples. Ms Raniolo added that this stx 2 gene was not detected in the testing of 39 samples of Mountain View milk by the MDU on 13 November 2014. Rather, one of the 39 samples tested positive for the stx1 gene, the eaeA Intimin and ehxA enterohaemolysin. Ms Raniolo argued that there could hence be no link between the testing on the milk sample and the direct DNA extraction from Child A's stool, as different pathogens were found.

81. Ms Raniolo submitted that the DHHS had failed to take into account appropriate material, including possible other sources regarding the two other cases of HUS, and two cases of cryptosporidium referred to in the outbreak report. She also noted that Child A's family had engaged in faecal specimen testing on 29 October 2014, which had tested negative for Shiga toxin producing E.coli and Cryptosporidium. In addition, a small quantity of bath milk taken from Child A's home and tested on 23 October 2014 by the DHHS had tested negative for Shiga toxin E.coli.

Further investigations

82. Following the receipt of the submissions made on behalf of Mountain View Farm Pty Ltd, I directed that further information be obtained as part of my investigation.

83. Child A's pathology test that was requested on 2 October 2014, was obtained by the Court and indicated that Cryptosporidium, but not Salmonella, Shigella nor Campylobacter were detected in stool collected on 3 October 2015. Child A's medical records were also obtained from his General Practitioner, which indicated little contact with clinicians throughout his life.

84. By way of letter dated 14 September 2016, the DHHS provided the HUS questionnaire completed during an interview with Child A's father. The questionnaire indicated that Child A's illness started on 29 September 2014. A box was ticked 'yes' regarding whether unpasteurised milk was consumed in the 10 days prior to the illness. While a box was ticked 'no' regarding consumption of products made from unpasteurised milk, a note was made: 'make own yoghurt from milk (bath milk used)'. On the food history page of the questionnaire, a note was made that Child A 'never had much dairy in general. Weeks leading up to illness was almost off dairy as found out intolerant'. However, a note is then made: 'prior to 29/9/14 – would have each night bath milk for a few weeks'. It was also noted that Child A's father 'could not provide a thorough 3-day food history.'

COMMENTS

Pursuant to section 67(3) of the **Coroners Act 2008**, I make the following comment(s) connected with the death:

Child A's medical management

1. I acknowledge the immense grief endured by Child A's family following his untimely death. I also acknowledge the concerns raised by Child A's father that his son's symptoms were not taken seriously and the severity of his illness was not recognised; and that his son was not transferred to the Monash Medical Centre sooner. In view of these concerns, a review was conducted by the Coroners Prevention Unit. Following the provision of a copy of this review to Child A's father, no further concerns regarding his son's medical management were raised.

2. Child A presented to the GP and Frankston Hospital with symptoms consistent with a severe acute gastroenteritis. Treatment for acute gastroenteritis is supportive, ensuring adequate hydration and symptom relief, and as such, this management plan was instituted in Child A's case. It appears that he was managed appropriately with supportive measures and care escalated during his admission in Frankston Hospital when his symptoms did not improve. He had blood testing, stool testing and advice was sought from the Infectious Diseases team at MMC with antibiotics for bacterial gastroenteritis administered. Child A had hyponatraemia, which worsened during his time at Frankston despite appropriate monitoring and treatment with IV fluids, but no signs of HUS on blood tests taken on 5 October 2014. Over the course of 6 October 2014, Child A had worsening abdominal pain, bloody diarrhoea, coffee coloured vomiting, and developed haemodynamic instability, worsening sodium and abnormal kidney function on blood tests. Nursing staff appropriately escalated Child A's condition to medical staff during the afternoon. The Paediatric Registrar reviewed Child A when he showed signs of haemodynamic compromise and appropriately escalated to the Consultant who initiated transfer to MMC for ongoing care.

3. In retrospect, an earlier review of Child A on the afternoon of 6 October 2014 would have been desirable. However, I note that Child A had a heart rate and blood pressure within the normal range until 5.20pm on 6 October 2014 and ongoing urine output documented on his fluid balance chart. He also had a reduced amount of diarrhoea on the night of 5 October 2014 and this coincided with his morning medical review by the Paediatrician. These features would have indicated clinical stability. Importantly, the evidence suggests it would have been unlikely that earlier review of Child A on 6 October 2014 would have significantly changed his management

or outcome. Ultimately, I accept that the development of HUS could not have been foreseen or prevented. HUS is a rare condition and an uncommon complication of gastroenteritis with EHEC. It can have a rapid onset and there is no prevention or curative treatment. I note that earlier treatment with antibiotics would not have changed the outcome as antibiotics have not been found to be effective in preventing or treating HUS. Management is supportive, with surgery only indicated when there are signs of ischaemic bowel or perforation.

Matters related to Mountain View Farm Pty Ltd

4. I have carefully read the submissions made on behalf of Mountain View Farm Pty Ltd dated 4 July 2016. I remain unpersuaded that the *Briginshaw* standard applies, or that the seriousness of the allegation made or gravity of the consequences prevent me from making a finding on the balance of probabilities that consumption of the company's 'Bath Milk' led to Child A's tragic death. In coming to this conclusion, I emphasise that at no point has a prosecution been contemplated against Mountain View Farm, and the company was not considered to have breached any legislation at the time of Child A's death in 2014. At the relevant time, Mountain View Farm were selling unpasteurised milk as a cosmetic product or 'Bath Milk'. It is regrettable that the company has been referenced in media reports related to Child A's death. However, from the perspective of this jurisdiction, it cannot be deemed a reflection on Mountain View Farm's broader dairy products – which are pasteurised – that a consumer's choice to drink unpasteurised milk, a well-documented risk factor for gastrointestinal diseases, which was sold as a cosmetic product, reflects poorly on the company.

5. Furthermore, I note that following regulatory changes after Child A's death, unpasteurised milk can only be sold in Victoria if it is transformed so that it is no longer a substance of a kind used for food. Dairy Food Safety Victoria have received no requests for approval to produce 'bath milk' under these new licence conditions. In these circumstances, any potential finding that consumption of unpasteurised milk led to Child A's death will not impact on the sale of 'bath milk' by Mountain View Farm Pty Ltd.

6. The submissions on behalf of Mountain View Farm made a number of assertions about the potential sources and nature of Child A's illness, which arguably cannot be fully tested without going to a full public hearing, which has not been requested. I have partially acceded to a request to seek further information, and have since obtained Child A's pathology results from a 3 October 2014 sample and the completed DHHS HUS questionnaire. However, I do not seek to

replicate the DHHS' large scale Outbreak Investigation Summary Report, and I am unconvinced by suggestions that this report contains gross inaccuracies or assumptions.

7. Indeed, in the search for other sources of Child A's illness, the submissions avoid acknowledging that consumption of unpasteurised milk is a well-documented risk factor for diseases including Shiga toxin producing *E. coli* infection. The basis for requiring that milk for human consumption be pasteurised is not that all raw milk is unfit for human consumption, but because the potential risks posed by allowing the sale of raw milk for human consumption are unacceptable. The submissions note that the stx 2 gene in Child A's stool was detected through unaccredited testing, and that it was the stx1 gene which was found in a sample of Mountain View Bath Milk on 13 November 2014. I acknowledge that these pathogens are different, but I would also point out that the evidence still presents a picture of the ability of bacteria to exist in unpasteurised milk.

8. I note that the submissions for Mountain View Farm look to cast doubt on Child A having consumed its Bath Milk, and argue that it was a 'clearly inaccurate and unsupported belief that Child A had been drinking the milk for 1-2 weeks prior to becoming ill'. However, I note that there is contemporaneous documentation in the medical records on 5 October 2014, that Child A's father said he had drunk unpasteurised milk. In his statement dated 26 May 2015, Child A's father said he and his wife 'could not remember... as to when last [Child A] had drunk the milk... There is a possibility that he had, but also a chance he had not.' The HUS Questionnaire obtained from the DHHS was completed in October 2014 appeared to suggest that Child A had consumed unpasteurised milk in the 10 days prior to his illness, and had night bath milk for a few weeks prior to presenting with symptoms. I acknowledge that the milk obtained from Child A's home did not test positive for pathogens, but I do not view this as conclusive that Child A did not consume other unpasteurised milk in the relevant period. Moreover, the lack of Shiga toxin in the stool sample collected on 3 October 2014, does not definitively exclude the possibility of unpasteurised milk containing bacteria having been consumed.

9. In circumstances where I am subject to a standard of proof which rests on the balance of probabilities; where both the suffering of Haemolytic Uraemic Syndrome and consumption of unpasteurised milk are individually extremely rare; where reference to unpasteurised milk has been made in the DHHS report on more than one occasion, and with the supplementary report from Dr Parsons which opined that the DHHS report made a good case that the likely source of the shiga toxin was unpasteurised milk, I have not seen fit to doubt the validity of the methodology behind the DHHS Outbreak Investigation Summary Report. Pursuant to section 7

of the *Coroners Act 2008* (Vic) I am obliged to avoid unnecessary duplication of inquiries and investigations, and I do not seek to duplicate the investigation undertaken by the department.

10. I have not made recommendations as part of this Finding, because the evidence indicates that the regulation of the sale of unpasteurised milk in Victoria has been adequately managed by Dairy Food Safety Victoria, in the wake of Child A's death and the DHHS report. If members of our community choose to drink farm-gate unpasteurised milk, that is their choice. However, they should do so in the knowledge that it may contain harmful bacteria.

FINDINGS

The weight of the evidence available to me indicates that Child A's death was consistent with haemolytic uraemic syndrome secondary to *E. coli* infection. Noting that haemolytic uraemic syndrome is treated by supportive measures, I find that Child A's medical management was reasonable and appropriate in the circumstances, and I make no adverse finding against clinicians involved with his treatment.

In the absence of a specific request for an Inquest, and considering pursuant to section 8 of the *Coroners Act 2008* (Vic) that unnecessarily protracted coronial investigations may exacerbate the distress of Child A's family, I have concluded that I have sufficient evidence to complete this matter by way of this in-chambers finding.

Following the receipt of extensive material, including the Department of Health and Human Services' 'Outbreak Investigation Summary Report – Haemolytic Uraemic Syndrome (HUS) 2014', a supplementary report from Forensic Pathologist Dr Sarah Parsons, and submissions on behalf of Mountain View Farm Pty Ltd, I find on the balance of probabilities that Child A's death was most likely linked to the consumption of unpasteurised milk. In the circumstances, I have attributed more weight to the combined evidence of the Department of Health and Human Services' report, the Forensic Pathologist who examined Child A, and the clinicians at the Coroners Prevention Unit, than to the report from Microbiologist Ron Hull.

The evidence demonstrates that Mountain View Farm Pty Ltd appropriately labelled their unpasteurised milk product as 'bath milk', so as to not be for human consumption, and that no illegal activity occurred. I make no adverse finding against Mountain View Farm Pty Ltd.

I accept and adopt the medical cause of death as ascribed by Dr Sarah Parsons, and find that Child A tragically died from thrombotic microangiopathy.

Pursuant to section 73(1A) of the *Coroners Act 2008,* I order that this Finding be published on the internet.

I direct that a copy of this finding be provided to the following:

The father of Child A

Professor Charles Guest, Chief Health Officer of Victoria

Ms Kym Peake, Secretary of the Department of Health and Human Services

Mrs Kylie Burns, Medico-Legal Department, Peninsula Health

Ms Katherine Lorenz, Chief Legal Officer, Monash Health

Professor Jeremy Oats, Consultative Council on Obstetric and Paediatric Mortality and Morbidity

Mr Richard Murphy, MinterEllison on behalf of Dairy Food Safety Victoria

Ms Rose Raniolo, Colin, Biggers & Paisley Lawyers on behalf of Mountain View Farm Pty Ltd

Ms Vicki Jones, Mountain View Farm Pty Ltd

Detective Senior Constable Kerryn Merrett

Signature:

AUDREY JAMIESON
CORONER

Date: **24 October 2016**