

# Investigation of two clusters of shiga toxin-producing *Escherichia coli* cases in South Australia

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Shiga toxin-producing *Escherichia coli* (STEC) is an important cause of gastrointestinal illness in developed countries, and outbreaks have been reported in many countries including Australia.<sup>1,2</sup> STEC infection can cause bloody diarrhoea, with 3–7 per cent of sporadic cases developing haemolytic uraemic syndrome (HUS), a serious condition, defined by thrombocytopenia, anaemia and renal failure, which can result in death.<sup>1,2,3</sup> In Australia, the number of reported STEC cases, was 38, 48 and 52 in the years 2000, 2001 and 2002 respectively.<sup>4</sup> South Australia reported 38 (73%) of the 52 cases in 2002. Current surveillance STEC practices in South Australia involves screening of all bloody stools with a polymerase chain reaction (PCR) test for the toxin genes, which contributes to the number of cases reported from this state. Samples positive for toxin are tested for virulence and serotype genes.<sup>5</sup> This procedure complements standard epidemiological practices.

Relatively few STEC outbreaks have been reported in Australia.<sup>2</sup> This report describes the investigation of two clusters of STEC cases in South Australia, observed in February and March 2003.

## The first cluster

Between 3 and 6 March 2003, four STEC cases from Adelaide suburbs were notified to the Communicable Disease Control Branch (CDCB). Of the four cases, three were PCR positive for serotype O157 and toxin gene STX2 and negative for toxin gene STX1. One O157 case was also positive for virulence genes *eae* (codes for intimin) and *hly<sub>a</sub>* (codes for enterohemolysin). The other two cases were negative for these genes possibly due to a low number of STEC in the stool sample. These typing results suggested that the cases may be epidemiological linked.<sup>5</sup>

These three cases were interviewed with a hypothesis generating questionnaire, which included a 10 day food history prior to illness, food purchasing habits and social activities undertaken during this period.

Of the three cases, one was male and two were females and all were aged 61 years or more. All three cases had bloody diarrhoea with dates of onset of 25, 27 and 28 February 2003. Two were hospitalised for a week and there were no reports of cases developing HUS.

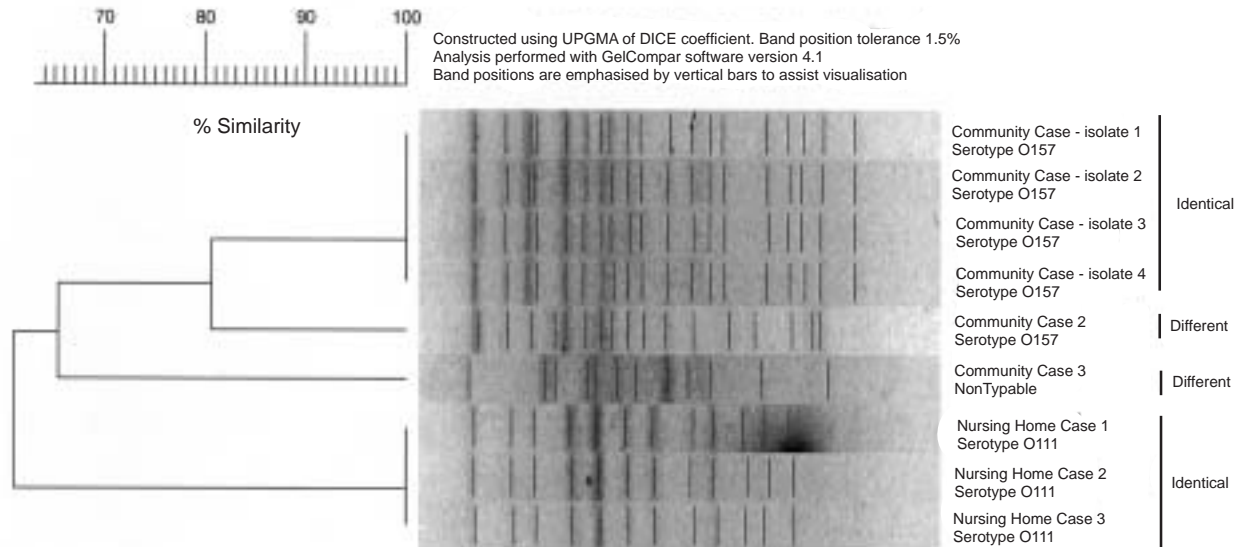
These cases had no contact with each other and the food history revealed no common food vehicle or other exposure. However, the male case and a female case reported eating Hawaiian pizza. In addition, both female cases purchased meat and small goods including fruit and vegetables from the same supermarket.

The stool samples were cultured and *E. coli* isolates screened for STEC toxin genes. Unfortunately, isolates were only recovered from the male case and one of the female cases, who did not eat pizza. These isolates were subjected to PFGE using restriction enzyme *Xba*I (New England Biolabs), and analysed using the software GelCompar 4.1 (Applied Maths, Sint-Martens-Latem, Belgium). The PFGE gel was interpreted according to Tenover criteria for strain identification.<sup>6</sup> Results showed that the isolates from community cases 1 and 2 had different patterns from each other (Figure) and from other STEC control isolates (community case 3). Overall, the molecular and descriptive epidemiology suggests that the two cases were unrelated. As an isolate was not cultured from one of the female cases it is unclear if the two female cases were associated with a common food source or other exposure. There were no further reports of STEC O157 with similar dates of onset.

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**Figure. Dendrogram of Pulsed Field Patterns of STEC isolates from a community cluster and a nursing home cluster**



### The second cluster/outbreak

On 20 March 2003, two STEC cases from a nursing home were notified to the CDCB and epidemiological, environmental and microbiological investigations were initiated. At the nursing home there were 115 residents and approximately 112 staff members. Investigations revealed 12 residents and one staff member had gastrointestinal illness. Stools from all people who were ill were screened for enteric pathogens. All were negative except for one more resident having a stool positive for STEC toxin genes.

The three laboratory confirmed cases, were aged 79 to 87 years, and all experienced diarrhoea. Two of these cases experienced bloody diarrhoea and were hospitalised. The dates of onset were 13, 14 and 16 March 2003.

The cases lived in different sections of the nursing home but staff reported occasionally working across all sections. There was a set menu for residents and two cases may have had contact with each other at lunch times in the dining room. The third case required feeding assistance and did not attend the dining room. Each section of the nursing home had a kitchen where kitchen staff prepared main meals, including snacks. The food safety methods and food hygiene practices in these kitchens were found to be satisfactory. Ingredients for meals were supplied by an external food manufacturing facility, which was reported to be well maintained and had good systems

in place for food preparation. At the manufacturing facility there was no evidence of gastrointestinal illness among staff or among people at other locations that the facility supplied. In the 10 day period prior to illness there were no social activities when all three cases could have interacted.

STEC isolates were recovered from the three cases and were serotyped O111, which is a common STEC serotype within South Australia.<sup>2</sup> The isolates were PFGE typed and were found to have a common banding pattern (Figure), further confirming that the cases were epidemiologically linked. From the epidemiological and environmental investigations no common foodborne vehicle of infection was found. The only hypothesis that the descriptive epidemiology suggested was person-to-person transmission, perhaps via staff members.

### Discussion

STEC continues to be an important cause of gastrointestinal illness in South Australia. Most infections are sporadic cases with occasional outbreaks identified. In 2002, there was an outbreak of STEC associated with children visiting a petting zoo.<sup>7</sup>

In the investigation of the O157 cluster described above, the PFGE typing later helped confirm that at least two of the cases were not linked despite cases having a similar date of disease onset, having the same serotype and toxin gene profile, and being located in the same region of metropolitan Adelaide. Evaluation of the USA Pulse Net system found that PFGE was useful as an adjunct and not a replacement of epidemiological investigation. In the United States of America (USA), PFGE of STEC isolates has been used to confirm that clusters of cases are not related thus avoiding further costly epidemiological investigation, especially when typing is carried out in a timely manner.<sup>8</sup>

In the O157 cluster and nursing home outbreak, only people over 60 years of age were involved. STEC outbreaks in nursing homes have also been reported in Canada and the USA.<sup>9,10</sup> This emphasizes the need for high levels of food hygiene and infection control procedures in institutions caring for the elderly, who are at a higher risk for STEC infection. At the South Australian nursing home, the CDCB and local government officers reinforced infection control procedures, which included the need for regular hand washing, food handling procedures, environmental cleaning and correct linen handling procedures. Intensive surveillance of the nursing home continued for four weeks and residents continued to have stools screened for STEC toxin genes.

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