Laboratory surveillance of Shiga toxin producing *Escherichia coli* in South Australia and the Hunter Health Area, New South Wales, Australia

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Abstract

To estimate the prevalence of Shiga toxin producing *Escherichia coli* in Australia, bloody stool samples from two Australian locations were screened for the presence of Shiga toxin genes, *stx1* and *stx2*. Four of 126 (3.2%) and 139 of 5,829 (2.4%) patients from the two locations had a positive polymerase chain reaction for Shiga toxin genes. *Commun Dis Intell* 2004;28:390–391.

Keywords: Shiga toxin, Escherichia coli*, Australia*

Shiga toxin producing *Escherichia coli* (STEC) is the commonest cause of post-diarrhoeal hemolytic uraemic syndrome (HUS) in industrialised countries including Australia.[1] The majority of STEC infections are acquired by humans via the food chain, particularly from contaminated meat sources.[2] In order to develop strategies to prevent acquisition of STEC, it is important to understand the prevalence of the organism in humans with diarrhoea.

This summary reports data on screening of stool samples for STEC toxin genes, *stx1* and *stx2,* from two Australian locations, South Australia and the Hunter Health Area of New South Wales, between January 1999 and December 2002. The Institute of Medical and Veterinary Science (IMVS) in South Australia screened all stools, including those referred from diagnostic laboratories, with macroscopic evidence of blood, or from patients with a clinical history of bloody diarrhoea, or when a sample was accompanied by a request from a physician. This included screening stools from HUS cases. In the Hunter, screening was undertaken on stool samples submitted to the Hunter Area Pathology Service (HAPS), with profuse red blood cells, from which *E. coli* but no other bacterial diarrhoeal pathogen was detected. Stool samples from HUS cases were also screened.

All samples were screened for STEC toxin genes using multiplex real-time TaqMan™ polymerase chain reaction (PCR) (Applied Biosystems). Controls used for each assay run included *stx1* and *stx2* positive *E. coli* O111, and O157 and an *stx* negative *E. coli* ATCC 25922 at HAPS, and *stx1* and *stx2* positive *E. coli* O157 and *stx* negative *E. coli* ATCC 25922 at IMVS.

During the study period, four of 126 patients from the Hunter Health Area were positive for STEC toxin genes (3.2%) of which two were positive for *stx2*, one for *stx1* and one for both genes. Positives were detected in 1999 and 2002 only. In South Australia, 139 of 5,829 patients were positive for STEC toxin genes (2.4%), 42 were positive for *stx1*, 37 for *stx2* and 60 were positive for both genes (Table). STEC positives were detected in each year of the study period.

The two cases of HUS positive for STEC reported in the Hunter Health Area included a 26-month-old male and a 62-year-old male, who was also diagnosed with *Campylobacter*. In South Australia, the three HUS patients positive for STEC comprised a 10-month-old male, a 2-year-old female, and a

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Table. Summary of Shiga toxin producing *Escherichia coli* screening of stool samples from South Australia and from the Hunter Health Area of New South Wales, Australia, between 1999 and 2002

Location	Patients tested*	Number positive for *stx1* and/or *stx2* genes	Percentage positive	Number with HUS†
South Australia	5,829	139‡	2.4	3§
Hunter	126	4	3.2	2

* Excludes repeat samples collected within 14 days.

† HUS: hemolytic uraemic syndrome.

‡ A further 2 Shiga toxin producing *Escherichia coli* cases were detected in 1999 by culture only.

§ There were three additional HUS cases in this period that were not positive for STEC.

21-year-old male. The HUS patients appeared to be sporadic cases as there was no observed link between them.

During the study period, South Australia and the Hunter using the same PCR method, found 2.4 to 3.2 per cent of bloody stools positive for STEC toxin genes, respectively, suggesting that other regions in Australia may have similar levels of STEC infection. These rates are in line with those detected in the United States of America, with studies reporting prevalence of 4.2 per cent (14/335)[3] and 2.1 per cent (39/1,851)[4] using ELISA toxin testing and PCR, respectively. In Thailand one per cent (2/211) of samples tested were positive for STEC by culture followed by PCR.[5]

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