Serogroup-Specific Risk Factors for Shiga Toxin–Producing *Escherichia coli* Infection in Australia

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Background. Shiga toxin–producing *Escherichia coli* (STEC) is an important cause of foodborne illness. In Australia, risk factors for STEC infection have not been examined at a national level.

Methods. We conducted a case-control study in 6 Australian jurisdictions from 2003 through 2007. A case patient was defined as a person from whom STEC was isolated or toxin production genes were detected in stool. Case patients were recruited from notifiable disease registers, and 3 control subjects frequency matched by age were selected from databases of controls. Using structured questionnaires, interviewers collected data on clinical illness, foods consumed, and exposures to potential environmental sources.

Results. We recruited 43 case patients infected with STEC serogroup O157, 71 case patients infected with non-O157 serogroups, and 304 control subjects. One patient infected with serogroup O157 and 7 infected with non-O157 serogroups developed hemolytic uremic syndrome. Compared with control subjects, case patients infected with STEC O157 were more likely to eat hamburgers, visit restaurants, have previously used antibiotics, or have family occupational exposure to red meat. Case patients infected with non-O157 STEC were more likely to eat sliced chicken meat or corned beef from a delicatessen, camp in the bush, eat catered meals, or have family occupational exposure to animals. Negative associations were observed for certain foods, particularly homegrown vegetables, fruits, or herbs.

Conclusion. This study of risk factors for STEC infection by serogroup highlights risks associated with eating hamburgers and occupational handling of raw meat. To prevent infection, hamburgers must be cooked thoroughly, and people handling raw meat or who have close contact with animals must ensure adequate hygiene.

Shiga toxin–producing *Escherichia coli* (STEC) infection causes acute gastroenteritis characterized by abdominal cramps and bloody diarrhea [1]. Approximately 3%–7% of STEC cases present with hemolytic uremic syndrome, which can be fatal. Large outbreaks over wide geographic regions are not uncommon and have been associated with animal contact, meat consumption, and increasingly, fresh produce [2–5].

In industrialized countries, STEC serogroup O157 is the predominant strain causing infection, and many countries have conducted studies to identify associated risk factors, including the United States [6, 7], Scotland [8], Canada [9], and Finland [10]. Consumption of hamburgers, ground beef, and cooked or sliced meats; contact with animals, farms, or animal feces; and travel are associated with increased risk of STEC infection [6–9, 11–17]. A small Australian case-control study in South Australia identified an association with consumption of berries, including strawberries, blackberries, and blueberries [18].

The incidence of STEC infections in Australia is lower (0.4 cases per 100,000 population) than that in many other developed countries [9, 19]. Since 2001, public health legislation in Australia has required laboratories and/or medical practitioners to report STEC infections to state and territory health departments. One Australian state—South Australia—has a higher screening rate of stool specimens for STEC by public health laboratories and a higher rate of notification (2.4 cases per 100,000 population) than those of other Aus-

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tralian jurisdictions [20]. There is considerable variation in the diagnostic techniques used to detect STEC among different Australian public health laboratories. Some laboratories use polymerase chain reaction (PCR)–based tests that include panels for common serogroups, whereas others use culture and/or immunological methods to detect toxins [21]. The higher rates of STEC in South Australia are likely because this jurisdiction uses PCR for diagnosis, which is more sensitive than culture [22, 23], and because of the increased numbers of tests performed. In Australia, serogroup O157 is the predominant strain of STEC, causing 60% of all infections [20].

In 2000, the Australian Government established OzFoodNet to enhance surveillance of foodborne diseases, including STEC infection [24]. To investigate risk factors for STEC infection, OzFoodNet conducted a case-control study of patients infected with serogroup O157 and all other (non-O157) serogroups.

METHODS

Study Design

We conducted a case-control study of sporadic STEC infection from July 2003 through April 2007 in 6 of the 8 states and territories in Australia. Ethics approval was gained through the relevant state or regional ethics committees. South Australia began recruiting case patients in July 2003, whereas all other jurisdictions began recruiting in January 2005.

Study Population

Cases. Doctors and laboratories were required by public health law to notify of cases of STEC infection in all Australian jurisdictions during the study period. Eligible case patients were patients diagnosed with STEC infection whose cases were notified to state or territory health departments and for whom Shiga toxin–producing *E. coli* were isolated from feces, Shiga toxin was detected in feces, *or* genes associated with the production of STEC toxin (*stx1* or *stx2*) were detected by nucleic acid testing (i.e., PCR) of an isolate of *E. coli* or directly in bloody diarrhea. Cases were classified as O157 serogroup on the basis of a positive result of PCR test or culture. All other cases were classified as non-O157 serogroups. Australian laboratory detection methods for STEC have been described elsewhere [21]. Determination of serogroup was performed by multiplex PCR for common serogroups (O157, O111, and O113) [25] or by traditional serotyping using O and H antigens [21].

Case patients were excluded if they (1) had coinfection with another pathogen, (2) had travelled overseas in the previous month, (3) had another person in their household with STEC infection or diarrhea in the previous month, (4) had been part of an STEC outbreak, (5) had no diarrhea or were unable to recall when diarrhea began, (6) had not been interviewed within 30 days after onset of illness, (7) had a delay of $\geqslant$10 days between the start of diarrhea and the collection of a stool specimen, or (8) had no telephone or were unreachable after 6 attempts. All jurisdictions except the Northern Territory and Tasmania participated in this study. Two jurisdictions—Queensland and New South Wales—had regionalized public health units contributing data to this study that covered 84% and 20% of their total populations, respectively.

Controls. Three control subjects per case patient were recruited when possible from control databases in every jurisdiction. These were matched to case patients in 5-year age groups, except for those aged <12 months, who were matched in 6-month age groups. The control databases were derived from previous research in which participants indicated they were willing to be contacted for additional surveys. Control subjects were excluded if they or another person in their household (1) had gastrointestinal illness in the 4 weeks before the interview, (2) had travelled overseas in the previous month, (3) had no telephone or were unreachable after 6 attempts, or (4) had not been interviewed within 30 days after the case was reported to the Department of Health.

Questionnaire

A telephone-administered, structured questionnaire was used to collect information on demographic, health, and exposure information, and case patients were asked additional questions about the clinical course of their illness and treatment. For food exposures, case patients were asked about the 10 days before the onset of illness; control subjects were asked about the 10 days before the interview. For environmental exposures, case patients and control subjects were asked about the previous 4 weeks before illness onset and the interview, respectively.

Data Analysis

The data set was divided into 2 groups for analysis based on serogroup (O157 or non-O157) and for comparison with controls. Descriptive, univariate, and multivariate analyses were conducted using STATA Intercooled, version 10.0 (Stata Corp.). All variables with P values $\leqslant$.10 were included in multivariate models for further assessment. Exposures with <5 cases were excluded, and similar variables were combined where appropriate. Multivariate analysis was conducted using backwards stepwise logistic regression, and the likelihood-ratio test determined which variables were excluded in each step. Age, sex, and state of residence were considered design variables and were included in the final model.

RESULTS

Study population. There were 246 cases of STEC infection notified in Australia during the study period. Twenty-two (9%) of the case patients were in states or territories that did not participate in the study, and 98 (40%) were ineligible. Of the

Table 1. Characteristics of case patients infected with Shiga toxin–producing *Escherichia coli* serogroup O157 and non-O157 serogroups and control subjects in Australia, 2003–2007.

Characteristic	Case patients infected with serogroup O157		Case patients infected with non-O157 serogroup		No. of control subjects/ total responses (%)
	No. of patients/total responses (%)	P[a]	No. of patients/total responses (%)	P[a]	
Sex		.047		.31	
Male	13/44 (30)		33/69 (48)		138/304 (45)
Female	31/44 (70)		36/69 (52)		166/304 (55)
Age group, years		.079		.655	
0–9	6/44 (14)		11/69 (16)		55/304 (18)
10–19	8/44 (18)		13/69 (19)		47/304 (15)
20–29	8/44 (18)		5/69 (7)		23/304 (7)
30–39	1/44 (2)		5/69 (7)		14/304 (4)
40–49	7/44 (16)		4/69 (6)		41/304 (13)
50–59	5/44 (11)		13/69 (19)		40/304 (13)
60–69	3/44 (7)		7/69 (10)		37/304 (12)
70–79	3/44 (7)		8/69 (12)		38/304 (12)
⩾80	3/44 (7)		3/69 (4)		9/304 (2)
State		.64		.20	
South Australia	33/44 (75)		50/69 (72)		240/304 (78)
Victoria	6/44 (14)		6/69 (9)		28/304 (9)
New South Wales	2/44 (5)		7/69 (10)		24/304 (7)
Western Australia	2/44 (5)		1/69 (1)		6/304 (1)
Queensland	1/44 (2)		5/69 (7)		6/304 (1)
Other language[b]		.081		.082	
Yes	1/44 (2)		12/66 (18)		32/304 (10)
No	43/44 (98)		54/66 (82)		272/304 (89)
Indigenous status		1.0		.45	
Indigenous	0/44 (0)		1/66 (2)		2/304 (1)
Nonindigenous	44/44 (100)		65/66 (98)		302/304 (99)
Education		.40		.28	
Primary	8/42 (19)		11/66 (17)		59/302 (19)
Secondary	8/42 (19)		19/66 (29)		94/302 (30)
Apprenticeship, certificate, or diploma	11/42 (26)		21/66 (32)		63/302 (20)
University	15/42 (36)		15/66 (23)		86/302 (28)

[a] P value for comparison with all control subjects.
[b] Answer to the question: Is any language other than English spoken in your household?

127 remaining case patients, 113 participated in the study, 3 died, 2 did not wish to participate, and 8 were lost to follow-up. Case patients recruited for the study represented 46% of all notified cases of STEC infection in Australia during the study period, and 51% of all notifications of STEC infection in participating jurisdictions. After ineligible cases were excluded, study cases represented 89% of eligible STEC infection notifications.

The reasons for ineligibility of case patients were coinfection (22 patients), recent travel (10), coinfection and travel (1), an ill person in the household (11), a defined STEC outbreak (13), no diarrhea or an uncertain onset (22), no interview within 30 days after illness (16), and ⩾10 days between the start of diarrhea and the collection of stool (3). Ineligible case patients were coinfected with *Campylobacter* (13 patients), *Salmonella* (4), *Shigella* (1), *Yersinia* (1), *Cryptosporidium* (1), *Aeromonas* (1), and *Plesiomonas* (1) species.

Participants. We recruited 44 case patients infected with STEC serogroup O157, 69 case patients infected with non-O157 serogroups, and 304 control subjects. The majority of case patients were from South Australia (33 case patients infected with O157, 50 infected with non-O157, and 240 control subjects) because of the longer duration of study recruitment and the higher rate of screening of stool specimens in that state. There was no difference between the case patients infected with O157 and those infected with non-O157 with respect to all demographic characteristics, except that case patients infected with O157 were significantly more likely to be female ($P = .02$) and

Table 2. Symptoms reported by case patients infected with Shiga toxin–producing *Escherichia coli* serogroup O157 and non-O157 serogroups in Australia, 2003–2007.

Symptom	No. of patients/total responses (%)		
	Case patients infected with serogroup O157	Case patients infected with non-O157 serogroup	*P*
Stomach cramps	43/44 (98)	61/66 (93)	.23
Blood in stool	43/43 (100)	58/67 (87)	.01
Nausea	29/41 (71)	40/63 (63)	.45
Reduced urine output	15/35 (43)	35/58 (60)	.10
Fever	14/43 (33)	34/65 (51)	.06
Chills	11/42 (26)	34/63 (54)	.005
Vomiting	14/43 (33)	30/67 (45)	.20
Headache	14/40 (35)	29/61 (48)	.21
Muscle/body aches	14/39 (36)	22/60 (37)	.94

were less likely to speak a language other than English at home ($P = .03$). There were no differences between case patients infected with STEC O157 and control subjects with respect to demographic characteristics, but significantly higher proportions of case patients infected with non-O157 were indigenous or spoke a language other than English at home, compared with control subjects (table 1).

Non-O157 cases included 14 cases of serogroup O111 infection, 7 cases of serogroup O26 infection, and 1 case each of infection with serogroups O103, OR:H−, O113, and O172. A serogroup was unable to be determined for 36 cases by using multiplex PCR for common serogroups. In addition, there were 7 cases for which serogroup was not tested or was unknown. A positive Shiga toxin gene was detected in 97% of non-O157 cases, with 48% positive for both *stx1* and *stx2,* 30% positive for *stx1* only, and 19% positive for *stx2* only. Among O157 cases, a positive toxin gene was detected in 95%, with 73% positive for both *stx1* and *stx2,* 2% positive for *stx1* only, and 21% positive for *stx2* only.

Blood in stool was reported by all case patients infected with O157, which was significantly more than was reported by case patients infected with non-O157 ($P = .01$; table 2). Case patients infected with non-O157 were significantly more likely to report chills than were case patients infected with O157 ($P = .01$). The proportion of case patients reporting all other symptoms was similar for both O157 and non-O157 serogroups. The median duration of diarrhea for case patients infected with O157 was 5 days (range, 2–22 days), compared with a median of 6 days for case patients infected with non-O157 (range, 1–20 days; $P = .3$). Half (49%) of case patients infected with O157 were hospitalized (median duration, 3 nights in the hospital; range, 1–17 nights), compared with 59% of case patients infected with non-O157 (median duration, 5 nights in hospital; range, 1–21 nights; $P = .4$).

One case patient infected with O157 (2%) and 7 case patients infected with non-O157 (10%) received a diagnosis of hemolytic uremic syndrome ($P = .15$). The non-O157 serogroups resulting in hemolytic uremic syndrome were O111 (3 cases), O113 (1 case), O26 (1 case), OR:H− (1 case), and untypeable (1 case). The median age of case patients with hemolytic uremic syndrome was 4 years (range, 1–62 years), and the male-to-female ratio was 1:1. When case patients with hemolytic uremic syndrome were compared with all other case patients, there was no significant difference in use of medications ($P = .09$) or antibiotics ($P = .34$) or in chronic illness ($P = .46$) before diagnosis.

Univariate analysis. In univariate analysis, infection with *E. coli* serogroup O157 was associated with eating at a restaurant or at an event that was catered (table 3). Eating hamburgers was associated with illness, as was the case of another household member having occupational exposure to raw red meat. Prior use of antibiotics was associated with serogroup O157 infection. Eating homegrown vegetables, fruits, or herbs was negatively associated with O157 infection.

Case patients infected with non-O157 serogroups of *E. coli* were more likely to have eaten at a catered event or eaten chicken, meat, or corned beef bought from a delicatessen. Case patients were also more likely to have camped in the bush, worked or had a family member who worked with animals, or lived on or visited a farm. Negative associations were observed with eating pork; eggs within the 2 days before illness; raw vegetables; homegrown vegetables, fruits, and herbs; or at a buffet or smorgasbord.

Univariate analysis of 14 case patients infected with *E. coli* serogroup O111, compared with all control subjects, showed similar findings to those for all case patients infected with non-O157 serogroups. Risk factors associated with O111 infection were eating meat samples at shopping centers (odds ratio, 8.0;

Table 3. Univariate analysis of risk factors for infection with Shiga toxin–producing *Escherichia coli* serogroup O157 and non-O157 serogroups for food consumed in the 10 days and environmental exposures in the 4 weeks before illness for case patients and before interview for control subjects in Australia, 2003–2007.

Exposure	Case patients infected with serogroup O157			Case patients infected with non-O157 serogroup			
	No. of patients/total responses (%)	OR (95% CI)	*P*	No. of patients/total responses (%)	OR (95% CI)	*P*	No. of control subjects/total responses (%)
Food from hamburger outlet	15/44 (34)	1.8 (0.9–3.8)	.08	20/69 (29)	1.4 (0.89–2.7)	.22	67/304 (22)
Hamburger chain	13/40 (33)	1.5 (0.7–3.2)	.27	18/56 (32)	1.5 (0.7–2.9)	.22	63/259 (25)
Other hamburger outlet	6/40 (15)	6.4 (1.6–23.4)	.004	4/57 (7)	2.7 (0.6–11.1)	.11	7/260 (3)
Any restaurant meal	26/44 (59)	2.1 (1.1–4.2)	.02	32/69 (46)	1.3 (0.7–2.2)	.40	124/304 (41)
Catered event	7/39 (18)	3.3 (1.1–9.3)	.01	9/58 (16)	2.8 (1.0–7.2)	.02	16/260 (6)
Raw vegetables	32/41 (78)	1.0 (05–2.6)	.94	35/69 (51)	0.3 (0.2–0.5)	.000	235/303 (78)
Homegrown fruit, vegetables, or herbs	9/41 (22)	0.4 (0.2–1.0)	.03	12/66 (18)	0.3 (0.2–0.7)	.001	117/299 (395)
Beef	40/43 (93)	2.4 (0.7–12.5)	.15	56/68 (82)	0.8 (0.4–1.8)	.6	257/303 (85)
Any hamburger	21/44 (48)	2.8 (1.4–5.7)	.001	15/69 (22)	0.9 (0.4–1.7)	.65	74/304 (24)
Pork	22/44 (50)	0.6 (0.3–1.3)	.15	31/68 (46)	0.5 (0.3–0.9)	.02	186/303 (61)
Eggs	29/42 (70)	0.8 (0.4–1.7)	.44	47/68 (69)	0.8 (0.4–1.4)	.35	227/304 (75)
Eggs in previous 2 days[a]	6/32 (19)	0.4 (0.1–1.0)	.04	13/51 (25)	0.6 (0.3–1.2)	.11	104/279 (37)
Deli meats	35/44 (80)	1.7 (0.8–4.2)	.18	52/69 (74)	1.2 (0.6–2.4)	.49	212/304 (70)
Chicken	4/42 (10)	2.2 (0.5–7.4)	.18	8/67 (12)	2.8 (1.0–7.5)	.02	14/303 (5)
Corned beef	3/42 (7)	0.6 (0.1–2.1)	.45	14/66 (21)	2.1 (1.0–4.4)	.03	34/304 (11)
Bush camping	3/43 (7)	2.0 (0.3–8.0)	.3	8/68 (12)	3.6 (1.2–10.1)	.006	11/304 (4)
Work with animals[b]	6/43 (14)	2.4 (0.7–6.8)	.07	12/68 (18)	3.2 (1.3–7.3)	.002	19/303 (6)
Work with raw red meat[b]	7/44 (16)	3.4 (1.1–9.4)	.008	6/69 (9)	1.7 (0.5–4.8)	.27	16/304 (5)
Live on or visit a farm	12/44 (27)	1.9 (0.8–4.2)	.07	19/69 (28)	2.0 (1.0–3.7)	.02	49/303 (16)
Antibiotic use	7/42 (17)	2.9 (0.9–7.7)	.02	9/68 (13)	2.2 (0.8–5.4)	.06	19/291 (6)

NOTE. The results in this table are based on comparisons between case patients infected with serogroup O157 and control subjects and between case patients infected with non-O157 serogroups and control subjects. CI, confidence interval; OR, odds ratio.

[a] This category was not statistically significant for the previous 10 days but was for the previous 2 days.

[b] By study subject or another member of the household.

95% confidence interval, 1.2–37.1), eating fruits and vegetables (odds ratio, 0.29; 95% confidence interval, 0.08–1.0), and having a family member who worked with animals (odds ratio, 6.0; 95% confidence interval, 1.2–23.0).

Multivariate analysis. For case patients infected with O157, 9 variables in addition to age group, sex, and state of residence were included in the model. Eating at a hamburger outlet was not included, because it was highly correlated with eating hamburgers (correlation coefficient $\kappa = 0.58$; $P < .001$), which was in the model. After backwards stepwise regression, we excluded variables that regarded contact with pet feces, a household member with occupational animal contact, and living on or visiting a farm (table 4). With this model, case patients infected with O157 were significantly more likely to have had a person in their household with occupational exposure to raw red meat, used antibiotics in the 4 weeks before illness, eaten a hamburger, and visited a restaurant. Infection with serogroup O157 was negatively associated with consumption of homegrown fruit, vegetables, and herbs.

For analysis of case patients infected with non-O157 serogroups, 11 variables were included in the model, in addition to age group, sex, and state of residence. After backwards stepwise regression, we excluded variables that regarded eating out and living on or visiting a farm (table 5). According to the model, case patients infected with non-O157 serogroups were more likely to have worked or had a family member work with animals, eaten sliced chicken meat or corned beef from a delicatessen, camped in the bush, and eaten at a catered event. Infection with non-O157 serogroups was negatively associated

Table 4. Multivariate analysis of risk factors for infection with Shiga toxin–producing *Escherichia coli* serogroup O157 for food consumed in the 10 days and environmental exposures in the 4 weeks before illness for case patients and before interview for control subjects in Australia, 2003–2007.

Risk factor	OR (95% CI)	*P*
Any restaurant	2.3 (1.1–5.0)	.032
Any hamburger	2.7 (1.2–5.9)	.015
Sausage	1.8 (0.8–3.9)	.12
Homegrown fruit, vegetables, or herbs	0.4 (0.2–0.95)	.038
Occupational exposure to raw red meat[a]	4.6 (1.4–14.5)	.010
Antibiotic use	3.7 (1.3–10.4)	.015

NOTE. CI, confidence interval; OR, odds ratio.

[a] By study subject or another member of the household.

Table 5. Multivariate analysis of risk factors for infection with Shiga toxin–producing *Escherichia coli* non-O157 serogroups, for food consumed in the 10 days and environmental exposures in the 4 weeks before illness for case patients and before interview for control subjects in Australia, 2003–2007.

Risk factor	OR (95% CI)	*P*
Catered event	3.1 (1.0–9.6)	.05
Sliced processed chicken	4.0 (1.3–11.6)	.013
Sliced corned beef	2.6 (1.1–6.0)	.023
Pork	0.5 (0.2–0.9)	.027
Raw vegetables	0.3 (0.2–0.6)	.001
Homegrown fruit, vegetables, or herbs	0.4 (0.2–0.8)	.01
Bush camping	3.7 (1.1–13.7)	.038
Occupational contact with animals	5.0 (2.1–12.1)	<.001
Antibiotic use	1.5 (0.5–4.6)	.45

NOTE. CI, confidence interval; OR, odds ratio.

with consumption of pork, raw vegetables, and homegrown vegetables, fruit, or herbs.

DISCUSSION

This Australian study of STEC infections identified important risk factors for both O157 and non-O157 serogroups. Risk factors for O157 infection were consistent with those reported internationally—consumption of hamburgers [7, 14] and eating at restaurants [9, 12]. Although occupational contact with raw red meat has not been reported previously, one study reported an association between O157 infection and meat purchased through a private slaughter arrangement [12], and several studies have reported associations with consumption of undercooked meat [6, 11]. Other studies have reported associations between O157 infection and consumption of pink hamburgers or ground beef in different settings [6, 9, 11, 12, 16] and other animal- and/or farm-related risk factors, including contact with farm animals [15], animal feces [8], and cattle on a farm [16]. Whether eating at a restaurant is a risk factor or is an indicator of a risk factor, such as cross-contamination of other foods with red meat, remains unclear. In Australia, hamburgers and ground beef have not been implicated in outbreaks of STEC and have not previously been considered a source of infection.

To our knowledge, this is the first study examining risk factors for non-O157 infection in a study population that included both children and adults. STEC serogroups may be associated with specific animal reservoirs [26, 27], making it important to consider risk factors by serogroup. Differences between O157 and non-O157 infections have been observed in studies conducted among children [28]. Risk factors for case patients infected with non-O157 serogroups were different from those identified for case patients infected with O157 and included occupational exposure to animals, consumption of sliced processed chicken meat, bush camping in Australia, eating out of the home at a catered event, and eating sliced corned beef. The different observed associations for O157 infection and non-O157 infection may reflect the respective host reservoirs for these serogroups.

In univariate analyses, each individual risk factor accounted for <20% of cases, which highlights the fact that that multiple factors are responsible for non-O157 infections. Animal contact at work is consistent with the findings for non-O157 serogroups of *E. coli* in domestic food animals, especially ruminants [1, 26]. The association with camping in the Australian bush may be linked to a range of factors, including poor food preparation, drinking of contaminated water, swimming in contaminated water, contact with wild animals, and lack of washing facilities. Although sliced chicken meat and corned beef have not been previously reported as risk factors for non-O157 infection, other studies have reported an association with O157 and cooked and/or sliced meats from a caterer [29] and rare chicken [13].

Although cases of infected with non-O157 STEC represented almost two-thirds of the cases in this study, infections due to these serogroups are not as well studied as those due to STEC serogroup O157, despite that non-O157 serogroups are as prevalent as or more common than serogroup O157 in Europe [30] and the United States [31]. This may be because of the difficulties in testing for non-O157 serogroups, for which nonculture methods are required, or because non-O157 serogroups often occur concurrently with serogroup O157 but only O157 is detected [1]. For example, in a large outbreak of hemolytic uremic syndrome in South Australia that was caused by serogroup O111, serogroups O157 and O160 were also recovered from patients. Mettwurst was the source of this outbreak, and implicated samples also contained a range of different STEC organisms [32]. It may be that if serogroup O157 is detected first, testing for other non-O157 serogroups may not be done.

For O157 and non-O157 serogroups, consumption of raw vegetables and/or homegrown fruit, vegetables, or herbs was negatively associated with infection, which has been observed in previous studies of *E. coli* serogroup O157 infection [12]. A possible explanation may be that there is a higher likelihood that control subjects reported consumption of fruit and vegetables because they had a lower consumption of meat. Other possible explanations might be that fruits and vegetables have high levels of antioxidants and carotenoids, which could boost general immunity, or that a diet of fruit and vegetables could affect intestinal microflora, which may alter host susceptibility to infection. There is a need for further research in this area, given how often these negative associations are observed in case-control studies of enteric pathogens, such as campylobacteriosis [33, 34] and salmonellosis [35].

There are several potential limitations to our study, many of

which relate to differences in surveillance for STEC throughout Australia. The higher proportion of cases from South Australia included in our study reflects the increased screening of bloody stools and the use of PCR for diagnosis in this state. This suggests that not all cases of STEC infection are diagnosed in other jurisdictions where these diagnostic practices are not in place. Also, the longer period of recruitment in South Australia contributed to a higher proportion of cases included from this state.

Many cases were not characterized beyond the most common serogroups, which were included in the non-O157 group for analysis. Laboratories commonly used PCR panels identifying at least serogroup O157 and other common serogroups. We may have introduced some misclassification bias by including in the non-O157 group 7 cases that were not tested for common serogroups, although analysis without these cases did not affect the findings (results not shown). Risk factors identified for non-O157 infection may not reflect those for specific non-O157 serogroups. Two smaller Australian jurisdictions did not participate in this study, although they reported <2 cases per year [20]. Despite these limitations, we believe that the results of this study are generalizable to STEC infections throughout Australia.

This study highlights that risk factors for STEC infection in Australia are serogroup specific, although contact with animals and eating outdoors were common to both analyzed groups. Education aimed at people who live or work with animals or raw meat, especially on farms, as well as those who enjoy camping, may reduce the incidence of infection with STEC. Although the absolute risk of infection with *E. coli* serogroup O157 from consumption of hamburgers is considered low in Australia, it is important to educate consumers and food handlers to cook hamburgers thoroughly.

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