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Foodborne general outbreaks of Shiga toxin-producing *Escherichia coli* O157 in England and Wales 1992–2002: where are the risks?

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SUMMARY

Between 1 January 1992 and 31 December 2002, Shiga toxin-producing *Escherichia coli* O157 (STEC O157) accounted for 44 of the 1645 foodborne general outbreaks of infectious intestinal disease reported to the Health Protection Agency Communicable Disease Surveillance Centre. These outbreaks, although rare, were characterized by severe infection, with 169 hospital admissions and five deaths reported. STEC O157 outbreaks were compared with other pathogens to identify factors associated with this pathogen. Single risk variable analysis and logistic regression were employed. Two distinct aetiologies were identified. Foodborne outbreaks of STEC O157 infection in England and Wales were independently associated with farms, which related to milk and milk products, and with red meats/meat products, which highlighted butchers' shops as a cause for concern. The introduction and adherence to effective control measures, based on the principles of hazard analysis, provide the best means of minimizing the risk of foodborne infection with this pathogen.

INTRODUCTION

Outbreaks of shiga toxin-producing *Escherichia coli* O157 (STEC O157) infection have gained public, scientific and political prominence ever since the central Scotland outbreak in 1996 [1], and their occurrence rarely escapes media attention. The organism causes a spectrum of illness, ranging from asymptomatic carriage or mild diarrhoea through to haemorrhagic colitis, haemolytic uraemic syndrome and thrombotic thrombocytopaenic purpurea [2]. Approximately 1000 cases are confirmed annually with most infections occurring in the third quarter of each year [3]. It has recently been estimated that over a third (38%) of cases of STEC O157 infection in England and Wales in 2000 were admitted to hospital as a result of their illness, and the case-fatality rate was 2·2% [4].

The major transmission routes for STEC O157 infection are food/water, animal contact and person-to-person transmission, although the ultimate source of STEC O157 is the faecal waste of farm animals, and cattle in particular. This view was reinforced through recent epidemiological studies of sporadic STEC O157 infection in Wales [5], England [6] and Scotland [7], where contact with the environment, and with animal excreta in particular, were identified as

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major risk factors for infection. However, foodborne transmission remains important.

The aim of this study was to identify factors (season, setting, vehicles of infection and contributory faults) that distinguish foodborne general outbreaks of STEC O157 infection reported to the Health Protection Agency (HPA) Communicable Disease Surveillance Centre (CDSC) from those attributed to other pathogens.

METHODS

The sources of routinely collected data on food poisoning in England and Wales have been described in detail previously [8, 9]. The current system of surveillance for general outbreaks of infectious intestinal disease (IID) began in 1992. Upon notification of an outbreak, CDSC administers a standard questionnaire to the lead investigator, with a request that it is completed when the outbreak investigations are complete. The questionnaire seeks a minimum dataset, including details of the setting, mode of transmission, causative organism, and epidemiological and laboratory investigations [10]. Routine reminders (up to three) are sent and returned questionnaires (response rate >80% [11]) are stored in a dynamic database derived from Epi-Info version 5 [12].

Outbreaks were selected where the mode of transmission was described on the form as mainly foodborne. Binary variables were created to represent the outcome of interest (outbreaks of STEC O157 infection *vs.* other pathogens) and explanatory variables (outbreak setting, season, food vehicles and contributory hygiene faults). The first date of onset in each outbreak was used to define the month of the outbreak, and approximate seasons (spring = March to May, summer = June to August, autumn = September to November, winter = December to February) were assigned. Outbreaks with missing data on the above were omitted from the analyses using those data.

A descriptive analysis of the data was undertaken using Microsoft Excel 2000 (Microsoft Corporation, Redmond, USA), Epi-Info version 6.04b and STATA version 7 (Stata Corporation, College Station, TX, USA). Means were compared using unpaired two-sided *t* tests. Point estimates, confidence intervals (CI) and a significance test for risk ratios (RR) were calculated using STATA.

Statistical analyses were undertaken using STATA version 7 [13]. Outbreaks of STEC O157 infection (considered 'case outbreaks') were compared with those attributed to other pathogens (considered 'control outbreaks') using single risk variable analyses. Maximum-likelihood estimates of the Mantel–Haenszel odds ratios (OR) were calculated for each explanatory variable. Logistic regression was then applied to obtain maximum-likelihood estimates of the effect of exposures on the outcome, whilst controlling for confounding effects. Variables with a *P* value of <0·1 from the single risk variable analysis were included initially in the model. The model was simplified using the Likelihood Ratio (LR) test. Potential interactions (amongst the main effects included in the initial logistic regression model and between these variables and season) were also examined using this technique. Where independent factors were identified, a descriptive analysis was undertaken to further quantify the risk.

Table 1. *General outbreaks of infectious intestinal disease (IID), foodborne general outbreaks of IID, and foodborne general outbreaks of STEC O157 infection, England and Wales, 1992–2002*

	General outbreaks		
Year	All	Foodborne (%)*	All STEC O157 (%)†
1992	373	224 (60)	3 (1·3)
1993	454	225 (50)	6 (2·7)
1994	490	192 (39)	0
1995	837	183 (22)	5 (2·7)
1996	733	165 (23)	7 (4·2)
1997	591	222 (38)	4 (1·8)
1998	610	121 (20)	4 (3·3)
1999	515	92 (18)	7 (7·6)
2000	656	96 (15)	6 (6·3)
2001	526	70 (13)	1 (1·4)
2002	1225	55 (4)	1 (1·8)
Total	7010	1645	44 (2·7)

* Of all general outbreaks.
† Of all foodborne general outbreaks.

RESULTS

Between 1 January 1992 and 31 December 2002, 7010 general outbreaks of IID were reported to CDSC, of which 1645 (23%) were described as foodborne outbreaks (Table 1). Foodborne outbreaks of STEC O157 infection ('STEC O157 outbreaks') accounted for 44 (3%) of these foodborne outbreaks. Whilst the incidence of all foodborne outbreaks declined during the surveillance period, the proportion of STEC O157 outbreaks showed no discernible trend.

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Table 2. *Factors associated with foodborne general outbreaks of STEC O157 infection, England and Wales, 1992–2002 – single risk variable analysis* (*exposures with a P value of <0·1 shown*)

Exposure	Per cent reported		OR	*P* value	95% CI
	Cases*	Controls†			
Spring	31	17	2·13	0·02	1·1–4·2
Farms	23	1	27·40	<0·001	11·3–66·5
Community	9	1	7·52	<0·001	2·5–23·1
Shops/retailers	23	7	4·19	<0·001	2·0–8·8
Restaurants	14	27	0·43	0·05	0·2–1·0
Function	18	49	0·23	<0·001	0·1–0·5
Milk/milk products‡	38	3	19·81	<0·001	8·1–48·2
Red meat/meat products‡	42	21	2·80	0·008	1·3–6·2
Cross-contamination§	45	28	2·15	0·01	1·2–4·0
Inappropriate storage§	9	30	0·23	0·003	0·1–0·7

OR, Odds ratio; CI, confidence interval.
* Foodborne general outbreaks of STEC O157 infection ($n=44$).
† Foodborne general outbreaks of IID attributed to other pathogens ($n=1601$).
‡ than one food vehicle can be reported for any given outbreak.
§ More than one food hygiene fault can be reported for any given outbreak.

A total of 625 people were affected (range 2–114) in the 44 STEC O157 outbreaks, with 169 hospital admissions (range 0–28) and five deaths (range 0–2) in those reported. Infection was confirmed microbiologically for 409 out of 625 people affected (65%) – a proportion far higher than for outbreaks attributed to other pathogens (15 262/36 213, 42%, $P<0·001$). Those people affected in STEC O157 outbreaks were nearly eight times more likely to require admission to hospital than in outbreaks attributed to other pathogens (0·27 *vs.* 0·04; RR 7·6, 95% CI 6·6–8·8, $P<0·001$). Furthermore, the risk of death in STEC O157 outbreaks was nearly five times higher than in outbreaks attributed to other pathogens (0·008 *vs.* 0·002, RR 4·6, 95% CI 1·9–11·4, $P<0·001$).

Factors associated with STEC O157 outbreaks, England and Wales, 1992–2002 – single risk variable analysis

Foodborne outbreaks of STEC O157 infection were more likely to occur in the spring compared to those attributed to other pathogens (Table 2). Farms, the community or shops/retailers were more likely to be the source or the setting for STEC O157 outbreaks. Milk/milk products or red meat/meat products were more likely to be the reported vehicles of infection and cross-contamination was more likely to be reported as a contributory food hygiene fault.

Table 3. *Factors independently associated with foodborne general outbreaks of STEC O157 infection, England and Wales, 1992–2002 – logistic regression analysis*

Exposure	OR	*P* value	95% CI
Spring	2·93	0·03	1·1–7·7
Farms	157·27	<0·001	41·8–592·5
Community	30·13	<0·001	4·8–189·4
Shops/retailers	6·98	0·001	2·2–22·3
Red meat/meat products*	6·72	0·001	2·1–21·4

OR, Odds ratio; CI, confidence interval.
* More than one food vehicle can be reported for any given outbreak.

Factors independently associated with STEC O157 outbreaks, England and Wales, 1992–2002 – logistic regression analysis

Outbreaks of STEC O157 were more likely to occur in the spring months than outbreaks attributed to other pathogens, and were more likely to be linked with farms, the community and shops/retailers (Table 3). Red meats/meat products were more likely to be reported as the likely vehicle of infection. No significant interactions were identified.

Further descriptive analysis of the factors independently associated with STEC O157 outbreaks showed that STEC O157 outbreaks associated with farms were commonly linked to milk and milk products

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[9/10; mainly unpasteurized milk (4) and milk sold as pasteurized (3)] whilst all (10/10) the outbreaks associated with shops were butchers' shops where red meat/meat products (7/10) were implicated in the infection.

DISCUSSION

Approximately one fifth of STEC O157 infections diagnosed in England and Wales occur as part of outbreaks [14], and foodborne STEC O157 outbreaks constitute approximately one third of these (CDSC, unpublished data). The projected 30-year cost of a foodborne outbreak of STEC O157 infection, with 71 laboratory-confirmed cases and 24 hospital admissions, has recently been estimated at £119 330 347 [15]. The cost of the 409 laboratory-confirmed cases and the 169 hospital admissions in the 44 outbreaks described above is, therefore, likely to be considerable.

We have applied logistic regression successfully to identify factors that distinguish foodborne general outbreaks of STEC O157 infection in England and Wales from those attributed to other pathogens. There was considerable overlap between the factors identified (especially between farms and milk and milk products, where collinearity probably explains the absence of the latter from the final model), underlining the common role of cattle as a source of STEC O157 infection. Were the dataset larger, it is probable that significant interactions between these variables would have been identified. None were found, suggesting that weak residual confounding exists, however, this is unlikely to have a great effect on our findings.

The epidemiology of foodborne outbreaks of STEC O157 infection in England and Wales mirrors that of the United States with a shift in recent years away from infection linked to the consumption of burgers [16]. Although outbreaks of STEC O157 infection associated with burgers have occurred occasionally in England and Wales [17], and raw burgers in this country can be contaminated with STEC O157 [18, 19], in a recent survey of 3440 cooked burgers the pathogen was not detected [20]. This suggests that the Government's advice [21] to cook burgers to a minimum temperature of 72 °C for 2 min or equivalent is being followed. The policy of zero-tolerance towards the presence of STEC O157 in ground beef, adopted by the United States Department of Agriculture in 1994, might explain the observed shift in epidemiology in the United States [22].

Two distinct aetiologies appear to exist in foodborne outbreaks of STEC O157 infection in England and Wales. Outbreaks were independently associated with farms, which relates to milk and milk products. The association with red meats and meat products points to butchers' shops in particular as a cause for concern.

The continued sale of unpasteurized drinking milk in England and Wales is contentious. Despite recommendations for its banning from a number of scientific bodies [2, 23], many believe that the health benefits from its consumption outweigh the potential public health risk, and that the public have the right to choose. However, choice depends on the provision of accurate information, and stricter labelling requirements for unpasteurized milk ('this milk has not been heat-treated and might therefore contain organisms harmful to health' [2]) recommended in January 1999, have yet to be implemented [24]. Furthermore, a proportion of STEC O157 infections are asymptomatic, but these individuals might represent a source of secondary infections through person-to-person transmission [2]. Therefore, whilst some individuals might make the informed choice to drink unpasteurized milk, some cases of STEC O157 infection might not be afforded this opportunity.

Outbreaks attributed to milk sold as pasteurized are perhaps more serious than unpasteurized milk outbreaks in public health terms, as the number of people at risk from infection is far greater. The first outbreak of STEC O157 infection attributed to pasteurized milk occurred in Scotland in 1994 [25], and the largest foodborne outbreak of STEC O157 infection in England to date was also associated with this product [26]. Producers must take adequate precautions to ensure that heat treatment is successful and that post-pasteurization contamination does not take place. A well-designed system, based and run on the principles of Hazard Analysis and Critical Control Points (HACCP), is the best means of ensuring this.

The central Scotland outbreak highlighted butchers' shops as a source of STEC O157 infection [1] although outbreaks associated with these premises had been described previously [27]. Workers in such establishments need to ensure effective food hygiene practices from the introduction of the carcass to the point of sale. The accelerated HACCP initiative for butchers' shops, introduced by the Department of

Health and the Meat and Livestock Commission (MLC) and completed in March 2000 [28] is, therefore, welcomed. However, the principal point of control with red-meat-associated foodborne pathogens is the slaughterhouse where the proper application of hygiene can reduce carcass contamination [29]. Equally important is the reduction in STEC O157 carried by cattle entering the abattoir.

The association between spring and foodborne outbreaks of STEC O157 is intriguing (the peak incidence in laboratory-confirmed cases (i.e. sporadic and outbreak cases; all transmission routes) occurs in late summer/early autumn [3]). In their 15-month study of STEC O157 in a dairy herd, Mechie and colleagues noted a peak in excretion in late spring/early summer, and suggested that this was due to the movement of non-lactating cows and heifers into the lactating herd at calving and the close contact between calving cows and their calves [30]. Irrigating grazing land with faecal slurry has also been identified as a risk factor for the carriage of STEC O157 in dairy cattle [31], as has a grain-based (as opposed to a hay-based) diet [32, 33]. Further work to identify factors affecting carriage and shedding of STEC O157 by cattle should be encouraged. An alternative explanation is the high proportion of non-STEC O157 outbreaks which occurred in the summer months inflating the role of springtime in STEC O157 outbreaks.

CONCLUSIONS

Foodborne outbreaks of STEC O157 infection occur infrequently, but the disease is severe. The identification of factors associated with outbreaks therefore forms an important aspect of prevention. The natural ecology of STEC O157 is such that certain premises and produce will present a potential risk of infection. The introduction and adherence to effective control measures, based on the principles of hazard analysis, provide the best means of minimizing this risk. Work to reduce carriage in cattle and cross-contamination in the abattoir will serve to reduce the risk further.

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