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Combining risk assessment and epidemiological risk factors to elucidate the sources of human *E. coli* O157 infection

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SUMMARY

E. coli O157 can be transmitted to humans by three primary (foodborne, environmental, waterborne) and one secondary (person-to-person transmission) pathways. A regression model and quantitative microbiological risk assessments (QMRAs) were applied to determine the relative importance of the primary transmission pathways in NE Scotland. Both approaches indicated that waterborne infection was the least important but it was unclear whether food or the environment was the main source of infection. The QMRAs over-predicted the number of cases by a factor of 30 and this could be because all *E. coli* O157 strains may not be equally infective and/or the level of infectivity in the dose–response model was too high. The efficacy of potential risk mitigation strategies to reduce human exposure to *E. coli* O157 using QMRAs was simulated. Risk mitigation strategies focusing on food and environment are likely to have the biggest impact on infection figures.

Key words: Beefburgers, *E. coli* O157, epidemiology, food poisoning, risk assessment, waterborne infection.

INTRODUCTION

Escherichia coli O157 is a gastrointestinal zoonotic pathogen of public health importance in a number of countries including North America and the UK [1]. The disease is relatively rare with the highest annual incidence worldwide being reported in Scotland (e.g. 4·7 cases/100 000 in 2008 [2]). The symptoms of the disease include bloody diarrhoea in ~90% of cases, of which 10–15% progress to haemolytic uraemic syndrome (HUS) [3], long-term risk of hypertension [4, 5] and occasionally death. The disease is most common in young children aged <5 years and incidence is highest in rural rather than urban areas [6].

The main reservoir for this pathogen is cattle and sheep [7, 8] but it can also be found in a number of other animal species (e.g. deer). Humans can become

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infected by three primary and one secondary transmission pathway: foodborne infection via contaminated food including beefburgers, dairy products and produce (e.g. vegetables) [9]; waterborne primarily through untreated, contaminated water supplies [10–12]; ingestion of faecal material by direct contact with animals, their faeces and contaminated soil [13] and the secondary transmission pathway is person-to-person contact which is particularly prevalent in nursery and care-home settings [14].

A number of major outbreaks have occurred with this pathogen, e.g. the Central Scotland outbreak in 1996 where 512 people were infected and 17 died which involved contaminated meat products [15]; the Walkerton outbreak in Canada in 2000 where 2300 people were infected from wells contaminated with ruminant faeces [11] and in the USA in 2006 where there was an outbreak associated with spinach consumption that involved 205 cases [16]. The trend in Scotland is increasingly towards outbreaks associated with sources other than foods such as contact with animals and drinking contaminated water [6]. However, the majority of human *E. coli* O157 cases are sporadic [17] and case-control studies in Scotland [13], USA [18, 19] and Wales [20] confirm the importance of the infection pathways outlined above.

The spatial epidemiology of this pathogen has been investigated in Scotland [6, 21], Sweden [22], Canada [23–25] and The Netherlands [26]. These studies demonstrate the higher incidence in rural areas compared to urban populations as well as the importance of risk factors such as contact with livestock and local socioeconomic status. One approach to determining the relative importance of each infection pathway would involve development of a regression model that combines the risk factors for each pathway and links this to the reported number of human cases. To the best of our knowledge this has not been previously attempted.

Stochastic quantitative microbiological risk assessments (QMRAs) have been developed to estimate the risk of illness associated with consuming beefburgers [27–30] and from visiting or camping on pasture that has recently been grazed by cattle or sheep [31]. A deterministic model has also been developed which estimates the number of cases associated with drinking from private water supplies (PWS) and visiting bathing waters [32]. One of the major advantages of using QMRAs is that they can evaluate the relative efficacy of mitigation strategies *in silico* prior to application in the real world. Combining epidemiological data with QMRAs (e.g. to develop dose–response relationships from outbreak data) has been reported previously [33]. However, combining risk assessments for each transmission pathway to predict the total number of human cases and the subsequent comparison with the number of reported cases in order to obtain an estimate of the importance of each infection pathway has not been attempted. This is because a human exposure survey of the risk factors (e.g. number of glasses of water drunk per day by the population on a PWS) is not available. Further, foodborne, waterborne and environmental QMRAs have not been simultaneously parameterized with data from a particular geographical area.

The aims of this paper are to assess the importance of the different transmission pathways of human *E. coli* O157 infection in the high-incidence Grampian region of Scotland by (i) developing a simple regression model parameterized with spatial data and (ii) developing and combining QMRAs to enable comparison with reported human case data. We then compare the results of these modelling techniques to obtain a greater understanding of *E. coli* O157 infection in Grampian. Finally, we evaluate a number of mitigation strategies for each transmission pathway to determine the efficacy of reducing *E. coli* O157 infections.

MATERIALS AND METHODS

Data

Anonymized human case data of *E. coli* O157 infection ($n=770$) were collected from National Health Service (NHS) Grampian for the years 1997–2008 inclusive. Of these, 694 had information containing the postcode district of residence, the date of reporting, and the age and gender of the case. The 27 cases identified from one primary school outbreak, is an outlier and these data were removed from the analysis, leaving 667.

Data on the human population at postcode district level were collected from the 2001 Scottish census [from SCROL (http://www.scrol.gov.uk/)]. Data on cattle and sheep numbers at the resolution of 2 km × 2 km tetrads were obtained from the 2000 Scottish agricultural census (from EDiNA® (http://edina.ac.uk/agcensus) revised for 2004). Data on properties on PWS ($n=13578$ in 2006) were obtained from Aberdeenshire and Moray local authorities.

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Empirical epidemiology

The incidence of *E. coli* O157 was calculated, as was the incidence ratio of rural to urban cases. Urban cases were defined as postal districts with a population density >200 persons/km² [34]. Tests of statistical significance and 95% confidence intervals were determined using a bootstrap method that involved resampling the original data with replacements using statistical software Poptools (www.cse.csiro.au/poptools/). The bootstrap sample size was 10000. This was also used to determine whether rural/urban ratios were statistically significantly different from 1. The percentage of cases that were general outbreaks was determined by consultation of reports from Health Protection Scotland (www.hps.scot.nhs.uk/ewr/index.aspx).

Data for risk assessment

A telephone-based exposure assessment questionnaire of a representative (by age, population density, socioeconomic status) subsample of the Grampian population (1068 questionnaires from a population 519979) was conducted by Aberdeen University and from this the following information was obtained: the number of 'well done' and 'rare' burgers consumed per person per year; the number of visits to camp-sites on pasture grazed by cattle and sheep per person per year; the number of glasses of water drunk per person per year and the proportion of treated PWS.

Spatial risk-factor regression model

A multivariate linear regression model was developed to link together proxy risk factors for each of the three primary transmission pathways [foodborne, environmental (e.g. direct contact with ruminants and their faeces) and waterborne (i.e. PWS)]. Person-to-person transmission was not included as it was assumed in the first instance to be similar for each pathway.

The proxy risk factors (defined below) were calculated for 2 km × 2 km tetrads. The proxy risk factor for food (F) is given by:

$$F \propto P, \tag{1}$$

where P is the number of people in the human population under consideration. This assumes that the same food is consumed and the same variation in hygiene/cooking processes are performed across Grampian. Hence the risk of contracting *E. coli* O157 infection from food is the same for each person in the population.

The environmental (E) proxy risk factor has the following form:

$$E \propto P \times N_{E.coli\mathrm{O157}}, \tag{2}$$

where $N_{E.coli\mathrm{O157}}$ is the total number of *E. coli* O157 excreted by cattle and sheep (calculated by estimating the average number of *E. coli* O157 excreted per gram of faeces multiplied by the average weight of faeces excreted per animal per day, multiplied by the number of animals in the postal sector – this was done separately for cattle and sheep and the results were summed). Hence, the greater the number of people and the greater the number of *E. coli* O157 excreted, the greater is the risk of human infection.

The waterborne (W) proxy risk is given by:

$$W \propto N_{\mathrm{PWS}} \times N_{E.coli\mathrm{O157}}, \tag{3}$$

where N_{PWS} is the number of properties on a PWS.

The regression model correlates the proxy risk factors with the actual number of reported cases (N_{cases}) and has the following form:

$$N_{\mathrm{cases}} = aF + bE + eW, \tag{4}$$

where a, b, and c are the regression coefficients.

The linear regression was performed in SPSS 17 (www.spss.com) at the postal district level ($n=38$) and to achieve this the 2 km × 2 km proxy risk-factor tetrads were integrated up to this spatial resolution using ArcView 3.3 (ESRI, USA). Normality of data was tested using XLSTAT (www.xlstat.com).

The number of predicted cases attributed to each transmission pathway for each postal district was determined by calculating the appropriate regression coefficient multiplied by the risk factor proxy (e.g. aF for food). This was then summed for all the postal districts in Grampian. The percentage attribution for each transmission pathway was then determined.

QMRAs

The QMRAs were written using @Risk (version 4.0, Palisade Corporation, USA). A manual giving full details of each of the models is available upon request to the corresponding author.

Food

The Cassin model for beefburgers was used [27]. Briefly, this process-based model has initial inputs which include the prevalence and concentration of

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Table 1. *Parameterisation of quantitative microbiological risk assessments (QMRAs) using Scottish datasets*

Parameter	Distribution	unit	QMRA model			Reference
			Food	Environment	Water	
Prevalence of *E. coli* O157 in cattle	Individual animal prevalence (138 samples positive out of 1402) RiskBeta (139, 1265)	—	Y	Y	Y	[60, 61]
	Group prevalence (23 herds positive out of 57) RiskBeta (24, 35)			Y	Y	[62]
Concentration of *E. coli* O157 in cattle faeces	RiskCumul (2, 6, {2,3,4,5,6}, {0·75, 0·84, 0·91, 0·985,1})	c.f.u./g	Y	Y	Y	[60, 61]
	Average of the discrete dataset ($1{\cdot}0 \times 10^4$, 95% CI 0–$7{\cdot}3 \times 10^4$)	c.f.u./g				
Number of burgers consumed in Grampian/year	~3·45 million	million	Y			[44]
Percentage of burgers consumed rare	1·8%	%	Y			[44]
Prevalence of *E. coli* O157 in sheep	Individual animal prevalence (85 positive out of 1066) RiskBeta (86, 982)	—		Y	Y	[63]
	Flock prevalence (6 positive out of 15) RiskBeta (7, 10)			Y	Y	[61]
Concentration of *E. coli* O157 in sheep faeces	Cumulative distribution of the discrete dataset RiskCumul (2, 5, {2, 5}, {0·553,1})	c.f.u./g		Y	Y	[63]
	Average of the discrete dataset ($5{\cdot}2 \times 10^4$, 95% CI 0–$2{\cdot}2 \times 10^5$)	c.f.u./g				[61]
Concentration of *E. coli* in cattle faeces	Average of discrete dataset ($1{\cdot}3 \times 10^6$) (see Supplementary Table A, online)	c.f.u./g			Y	Surveillance data from University of Aberdeen, 2009
Concentration of *E. coli* in sheep faeces	Average of discrete dataset ($2{\cdot}8 \times 10^6$) (see Supplementary Table B, online)	c.f.u./g			Y	Surveillance data from University of Aberdeen, 2009
Concentration of *E. coli* in PWS	Randomly selected from a discrete distribution (see Supplementary Table C, online)	c.f.u./100 ml			Y	Surveillance data from Food Standards Agency project (ref. number S14 024)
Prevalence of *E. coli* O157 in PWS	Riskbeta (2, 386) (1 positive out of 385)	—			Y	Surveillance data from Food Standards Agency project (ref. number S14 024)
Amount of water drunk/year from PWS in Grampian	~34·7 million glasses	million glasses			Y	[44]
Proportion of PWS treated	0·475	—			Y	[44]

Table 1 (*cont.*)

Parameter unit	Distribution	QMRA model			Reference
		Food	Environment	Water	
Number of cattle in the corresponding 2×2 km^2 containing the PWS	Selected according to the location of the PWS and cattle data available from agri-census	—		Y	Edina agricultural census 2004 (//edina.ac.uk/digimap/)
Number of sheep in the corresponding 2×2 km^2 containing the PWS	Selected according to the location of the PWS and sheep data available from agri-census	—		Y	Edina agricultural census 2004 (//edina.ac.uk/digimap/)
Cattle herd size	RiskTriang (20, 25, 30) Median -30	—	Y	Y	[31]
Sheep flock size	RiskTriang (2, 20, 300) Median – 40	—	Y	Y	[64]
Cattle per hectare	RiskNormal (3·57, 0·36)	—	Y		[31]
Sheep per hectare	RiskTriang (2·3, 2·3, 20)	—	Y		[65]

CI, Confidence interval; PWS, private water supply.

E. coli O157 excreted by cattle as they enter the abattoir (see Table 1). It determines the level of contamination on the meat and how this is affected by the grinding process. It simulates the survival of the pathogen in the burger, and models the die-off of the organisms during the cooking process. It then uses a beta-binomial dose–response model to determine the probability of illness. This QMRA was developed for the situation in Canada and here it is re-parameterized using data from Scotland (Table 1).

Environment

This process-based QMRA model was used to estimate the environmental risk of infection for day visitors and campers using pasture grazed by animals. Full details of the QMRA have already been reported [31] and here the data used in its parameterization were updated (Table 1). Briefly, this model uses data for human ingestion of soil that is contaminated with *E. coli* O157 shed by cattle or sheep. The model is run for the scenarios of either a day visit or an overnight camp to pasture. The same dose–response model used by Cassin *et al.* [27] was employed to calculate the resulting probability of infection. The model was run four times (overnight camping and day visits on fields each recently grazed by cattle or sheep) to cover each human exposure scenario.

Water

A process-based QMRA was developed to determine the risk of infection from drinking a glass of water from a PWS. The flow diagram in Figure 1 indicates the main steps in the process to determine the probability of illness. Briefly, a property on a PWS is randomly selected. The supply is identified as being contaminated or not using a beta distribution based on a prevalence survey of *E. coli* O157 in PWS in the Grampian region (Table 1). However, this does not give the concentration of *E. coli* O157 present and the following process is used for estimation purposes. If the PWS is negative the probability of illness from this source is zero. If positive, the number of cattle/sheep in the proximity ($2 \text{ km} \times 2 \text{ km}$ square) is determined from the agricultural census and from this the ratio of the average concentrations of *E. coli* O157 to *E. coli* excreted (Table 1) into the environment is calculated (see Supplementary material, available online). This ratio (R) is then multiplied by the concentration of *E. coli* (randomly sampled from the survey of PWS which are positive for *E. coli*) to determine the

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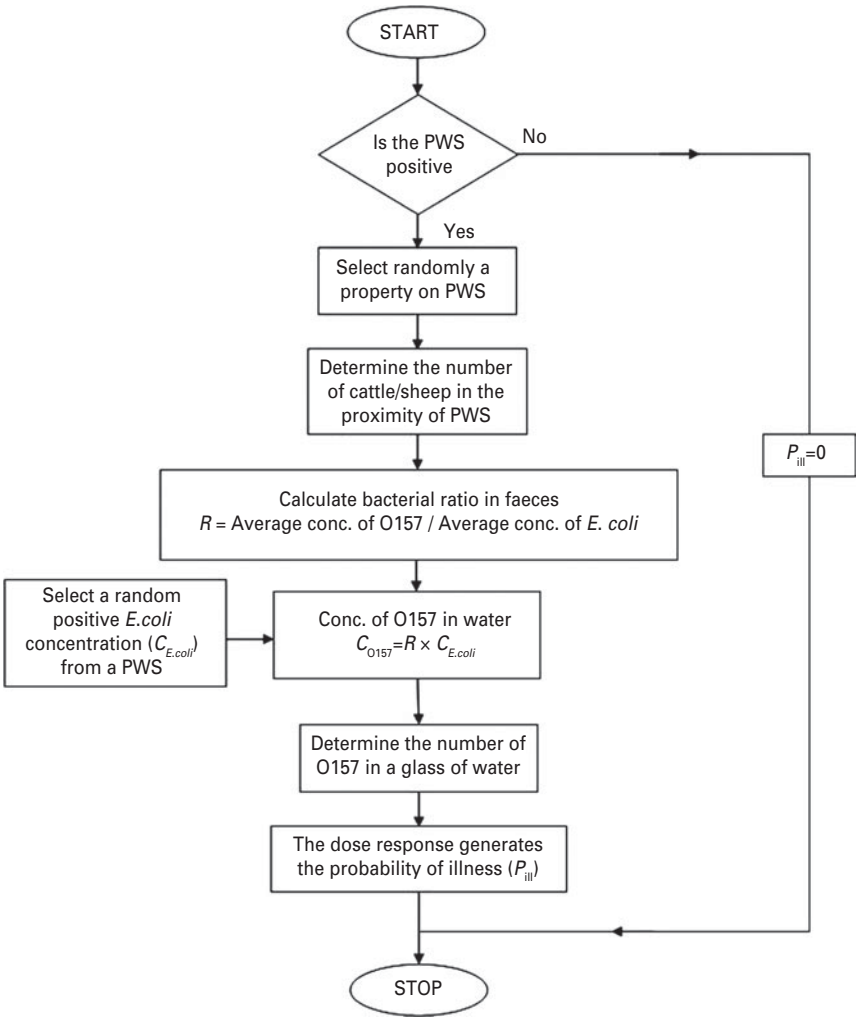

Fig. 1. Flow chart showing the private water supply (PWS) quantitative microbiological risk assessment (QMRA) which determines the probability of illness (P_{ill}) from drinking a single glass of water.

concentration of *E. coli* O157 in the PWS (see Supplementary material). The dose ingested from consuming a glass of water is calculated. The dose response is then applied to determine the probability of illness.

Incorporating exposure data into the QMRA models

The annual number of exposures, estimated from the survey, for the whole of the Grampian population ($n=519\,979$) for the different pathways were: 3·45 million beefburgers consumed, 2·48 million day visits [this excludes individuals who have contact daily (e.g. farmers who were considered to be immune to *E. coli* O157 in this study)], 0·1 million camps on pasture and 34·7 million glasses of water drunk from a PWS. Computational time and computer memory limitations meant that only a subsample of these exposures could be simulated (~2 million). Resulting values were then used to determine the number of illnesses in Grampian with associated confidence intervals. These data were then compared with the average number of cases reported in Grampian per year and also with the attribution of cases to one of the four pathways determined by the spatial risk-factor regression model.

Application of mitigation strategies in QMRAs

Risk mitigation strategies that were identified in the literature and readily operational in the QMRAs were simulated to determine their efficacy in reducing the number of cases of *E. coli* O157 infection.

The following mitigation strategies were evaluated (Table 2):

Food. (1) Removing high shedding animals prior to slaughter, (2) reducing concentration (numbers of

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Table 2. *Efficacy of risk mitigation strategies*

Pathway/ QMRA	Risk mitigation strategy	Amendment of QMRA parameter for each scenario	Predicted reduction in cases (%)
Food	(1) Remove high-shedding animals prior to slaughter	$>5{\cdot}0\ \log_{10}$	31
		$>4{\cdot}0\ \log_{10}$	34
		$>3{\cdot}0\ \log_{10}$	83
	(2) Reduce concentration shed	$0{\cdot}5\ \log_{10}$	33
		$1{\cdot}0\ \log_{10}$	64
		$2{\cdot}0\ \log_{10}$	90
		$3{\cdot}0\ \log_{10}$	98
		$4{\cdot}0\ \log_{10}$	99
	(3) Reduce rare burger consumption	0 % cooked rare	29
	(4) Decontamination of carcass at abattoir resulting in reduction in concentration*	$1{\cdot}0\ \log_{10}$	66
		$2{\cdot}0\ \log_{10}$	88
		$3{\cdot}0\ \log_{10}$	96
		$4{\cdot}0\ \log_{10}$	99
	(5) Percentage reduction in individual animal prevalence in cattle	25 %	8
		50 %	27
		75 %	51
		90 %	73
Environment	(1) Log reduction in dose by hand washing	$0{\cdot}5\ \log_{10}$	64
		$1{\cdot}0\ \log_{10}$	90
		$2{\cdot}0\ \log_{10}$	99
		$3{\cdot}0\ \log_{10}$	99·9
	(2) Banning camping in fields recently grazed by ruminants	100 %	23
	(3a) Reducing individual animal prevalence in cattle and sheep	25 %	26
		50 %	55
		75 %	80
		90 %	99
	(3b) Reducing group prevalence in cattle and sheep	25 %	20
		50 %	48
		75 %	74
		90 %	92
	(4) Reduce concentration shed	$0{\cdot}5\ \log_{10}$	65
		$1{\cdot}0\ \log_{10}$	90
		$2{\cdot}0\ \log_{10}$	99
		$3{\cdot}0\ \log_{10}$	99·9
	(5) Keeping farm animals off the pasture prior to human visit	7 days	62
		14 days	85
		21 days	94
		28 days	97
	(6) Physical removal of faeces from pasture	Triang (30 %, 50 %, 70 %)	44
Water	(1) Increasing the proportion of PWS treated to 100 %	60 %	7
		75 %	31
		80 %	63
		85 %	77
		100 %	94
	(2) Treatment of highly contaminated PWS (commensal *E. coli* counts)	>100 c.f.u./100 ml	45
		>10 c.f.u./100 ml	90
	(3a) Reducing individual animal prevalence in cattle and sheep	25 %	6
		50 %	28
		75 %	44
		90 %	79
	(4) Banning the use of PWS in areas with high density of animal numbers (no. per 2 km × 2km tetrad).	>1000 (15 % of PWS)	22 %

PWS, Private water supply; QMRA, quantitative microbiological risk assessment.

* The beefburger QMRA assumes a uniform distribution of between 0 and 2 log reduction using existing processes within the abattoir for the base scenario. This was re-run using 0 log as a baseline.

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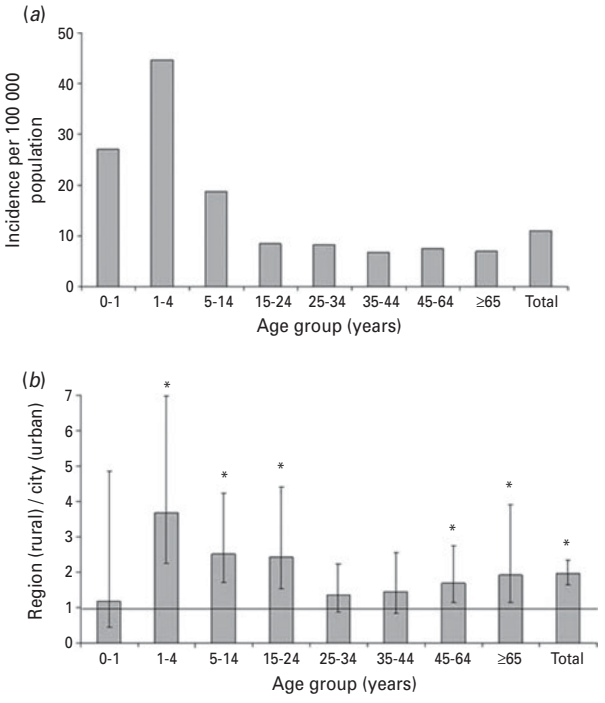

Fig. 2. (*a*) The average incidence of *E. coli* O157 infections in Grampian stratified by age for the 12-year period 1997–2008 and (*b*) the rural/urban incidence ratio with 95% confidence intervals [asterisks indicate statistically significant ($P<0{\cdot}05$) ratios $>1{\cdot}0$ determined by randomization test].

E. coli O157) shed, (3) a consumer education policy that ensures that burgers are cooked properly, (4) decontamination of carcasses in the abattoir and (5) reducing the individual animal prevalence in cattle.

Environment. (1) Washing hands after contact with animals and/or their faeces, (2) banning camping in the countryside in fields recently grazed by farm animals, (3) reducing (*a*) individual and (*b*) group prevalence in cattle and sheep, (4) reducing concentration shed, (5) keeping farm animals off pasture for a fixed period directly prior to day visit or camping visit and (6) physical removal of faeces from pasture prior to visit.

Water. (1) Increasing the proportion of PWS treated, (2) treatment of only those PWS which have a high load of commensal *E. coli*, (3) banning the use of PWS in areas of high livestock density and (4) reducing the individual animal prevalence in cattle.

RESULTS

Empirical epidemiology

The average incidence during this time period was 11·1 cases/100 000 and was highest in the youngest age groups (Fig. 2). The ratio of rural to urban incidence was 1·96 for the population as a whole and was largest for the age groups between 1 and 4 years.

Spatial risk factor regression model

Figure 3*a* presents a map of the number of reported *E. coli* O157 cases in each postcode district. Figure 3(*c–e*) show the proxy risks for food, environment and water. All three factors were included in the multivariate regression because they were all found to have correlation coefficients $>0{\cdot}4$ ($P<0{\cdot}05$) compared with the number of reported cases in univariate analysis. The multivariate regression model gave a significant fit ($R^2=0{\cdot}87$, $P<0{\cdot}0001$). The predicted number of cases by the regression model allocated to each postal district is illustrated in Figure 3*b* and the regression coefficients as well as the percentage of cases attributed to each pathway are given in Table 3. Environment was found to be the most important source while the model reported that PWS contributed the fewest cases. Cross-correlation was found between two combinations of the proxy risk variables used in the regression: food-environment ($R^2=0{\cdot}56$), food-PWS ($R^2=0{\cdot}04$), environment-PWS ($R^2=0{\cdot}67$).

QMRAs

The probability of illness (P_{ill}) for each iteration of the three QMRAs was determined and is displayed in Figure 4. The highest average probability of illness was from food ($P_{\text{ill}}=2{\cdot}65\times10^{-4}$, i.e. about 1 in 4000), which was greater than from environment ($P_{\text{ill}}=2{\cdot}15\times10^{-4}$, i.e. 1 in 5000) and PWS ($P_{\text{ill}}=1{\cdot}31\times10^{-5}$, i.e. 1 in 100 000). When this was combined with exposure information the number of cases could be determined (Fig. 4). Incorporating the exposure information permitted the number of cases associated with each transmission pathway to be determined. The food model predicted most cases (915) compared to 554 from the environment and 162 from PWS. The total number of predicted cases (1631) from the three QMRAs was ~30-fold higher than the average number of cases reported in Grampian per year. Both QMRA and spatial risk-factor regression approaches indicate that PWS are the least important pathway in terms of human cases but they order differently the importance of food and the environment (with overlapping confidence intervals).

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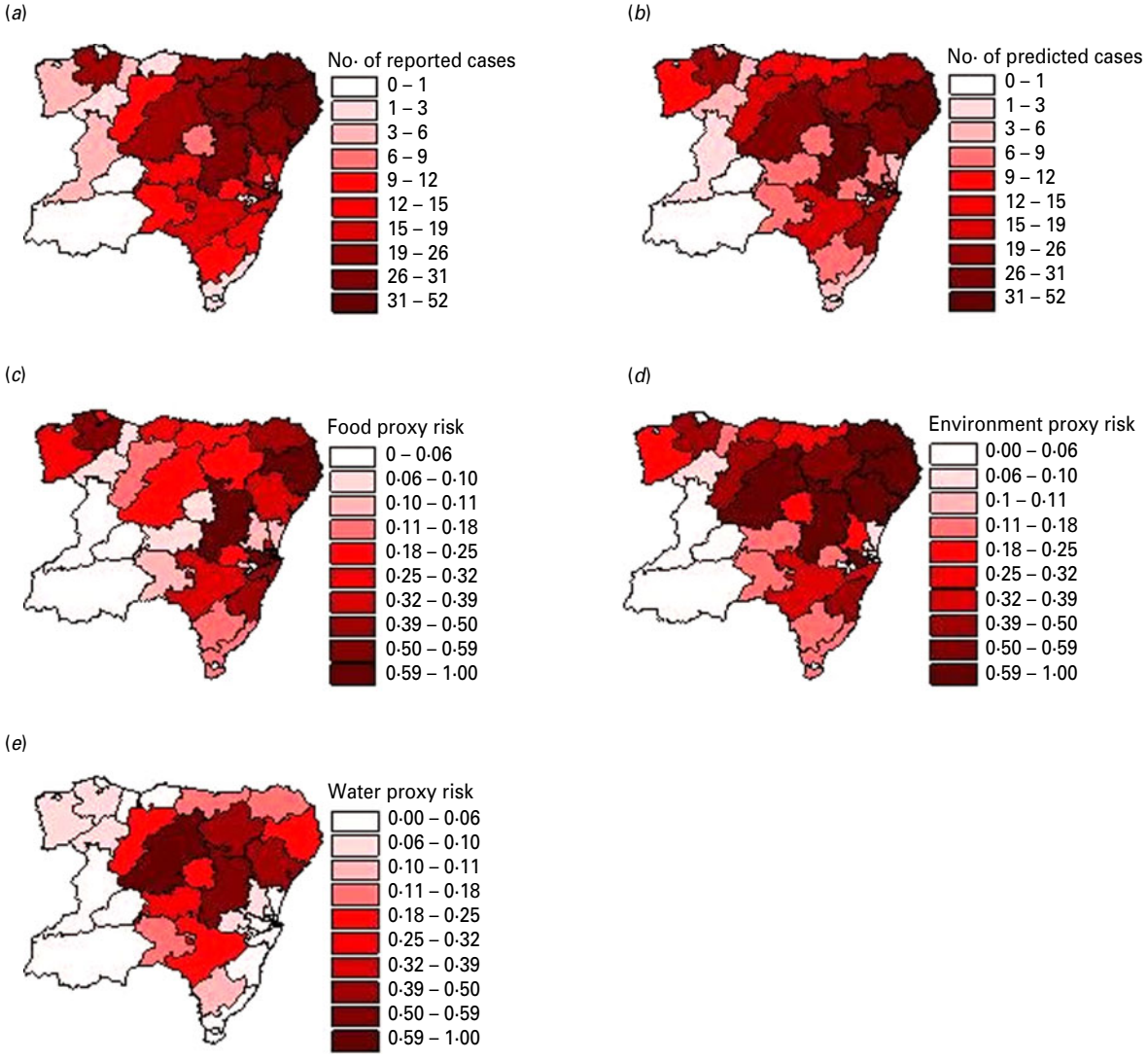

Fig. 3. Choropleth maps of the postal districts in Grampian showing (*a*) reported cases, (*b*) predicted number of cases using the regression model and proxy risks for (*c*) food, (*d*) environment and (*e*) water. The proxy risks are normalized to values between 0 and 1.

Risk mitigation

Table 2 presents the efficacy of the risk mitigation strategies for the three QMRAs. With regard to food, to achieve a reduction of ~50% in cases for that transmission pathway would require the removal of either those animals shedding high numbers of target bacteria (between 3 and 4 $\log_{10}$, which is equivalent to 1·35% of cattle) prior to slaughter, or reducing the concentration shed by 1 log, or reducing carcass contamination by 1 $\log_{10}$, or achieving a 75% reduction in individual animal prevalence. Ensuring all burgers are cooked properly would result in a 29% reduction in cases.

With regard to the environment, to reduce cases by 50% would require: reducing pathogen numbers ingested by 0·5 $\log_{10}$ through improved hand washing, or achieving a 50% reduction in individual animal prevalence, or reducing the group prevalence by 50%, or reducing concentration shed by 0·5 $\log_{10}$, or keeping farm animals off pasture prior to human visit for at least 7 days, or physical removal of faeces from pasture. Banning camping on pasture would only achieve a reduction of 23%.

Mitigations for water would achieve ~50% reduction in cases by increasing the proportion of PWS treated from 48% (current level) to ~80%, or treating all highly contaminated PWS (>100 c.f.u. commensal

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Table 3. *The mean number of cases attributed annually to each transmission pathway by the spatial risk-factor regression model and quantitative microbiological risk assessment (QMRA)**

Risk factors	Regression coefficients	*P* value	No. of cases attributed (95% CI)	Percent cases attributed (95% CI)
Regression model				
Food	$a=13{\cdot}8$	0·100	12·4 (5·1–19·8)	26·9 (11·0–42·8)
Environment	$b=36{\cdot}8$	<0·001	30·4 (22·9–38·0)	65·8 (49·6–82·0)
Water	$c=8{\cdot}3$	0·406	3·4 (0–7·5)	7·3 (0–16·0)
		Total	46·2	100
QMRAs	Average P_{ill}	Exposure		
Food	$2{\cdot}65\times10^{-4}$	3·45 million burgers/year	915 (879–954)	56·1 (52·2–60·4)
Environment	$2{\cdot}15\times10^{-4}$	2·48 million day visits, 0·1 million overnight camps	554 (525–584)	34·0 (28·7–39·4)
Water	$1{\cdot}31\times10^{-5}$	12·4 million people (34·7 million glasses/year)	162 (144–180)	9·9 (0·0–11·1)
		Total	1631	100

CI, Confidence interval; P_{ill}, probability of illness.
* The average of the annually reported number of cases is 55·6.

E. coli/100 ml). Banning the use of PWS in areas of high density of animal numbers (>250 animals/km²) would only reduce the number of cases by ~22%.

DISCUSSION

Within Grampian, *E. coli* O157 infection is dominated by cases from rural areas, particularly in children. This high incidence is probably due to two main factors: the immature immune system of the child and/or the child's increased exposure from one or more of the infection pathways. It is interesting to note that children aged <1 year old, although having a relatively high incidence do not over-present with the disease in rural areas compared with their urban counterparts. This could be because they are less likely to come in to contact with the pathogen through direct contact with farm animals. However, it would be expected that some of these children would be drinking from untreated PWS. Foodborne transmission is possible in this group (post-weaning) but this is most likely to be from meat products or contaminated foods other than milk which cannot be sold unpasteurized in Scotland. Children aged >1 year present on average much more frequently in rural areas. There is evidence from outbreaks (e.g. camping in Scotland [35]) which demonstrates that direct contact with animals and their faeces are significant risk factors in this age group. This is further exacerbated with person-to-person transmission being particularly important in young children [36] (with poor hygiene awareness) within the household or to the wider community (e.g. nurseries [17]).

The QMRA approach estimates an individual's probability of risk associated with particular exposures while the spatial risk factor regression method operates at a group level (e.g. the postal district). The QMRA is based on probability distributions whereas the regression model uses risk factors and is essentially deterministic. Further, the regression model requires the number of cases to be known to generate a model fit while the QMRA actually predicts this. Hence, the two approaches are quite different in trying to answer the same question of determining the relative importance of each of the primary infection pathways.

There is good agreement with the spatial regression and QMRA approaches in attributing the fewest cases to waterborne infection; however, it is unclear whether food or environment is most important. The risk associated with eating a burger is similar to visiting a field recently grazed by animals. This is at odds with our previous study [6] which found the pasture visit was a much greater risk (100-fold). However, here the burger model predicts that 29% of cases are associated with undercooking (eating rare) whereas previously we had assumed that they were well cooked. Empirical analysis of the epidemiological data (Fig. 2) shows that the incidence is approximately twice as high in rural areas compared to urban ones, indicating that the environment plays an important role. There is a degree of cross-correlation

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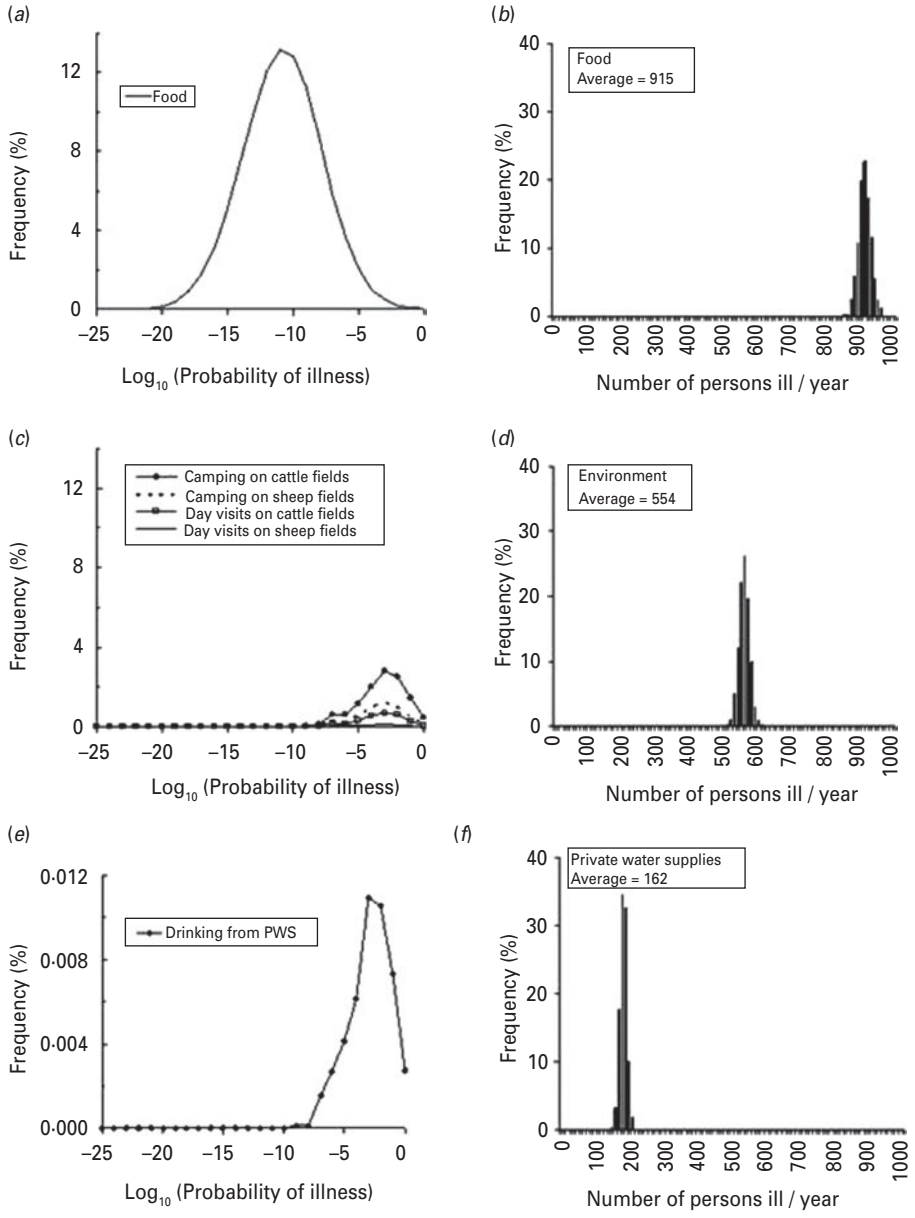

Fig. 4. The distribution of the probabilities of illness for (*a*) food (consuming a burger), (*c*) environment (four scenarios: camping/visiting pasture with sheep/cattle) and (*e*) drinking from a private water supply (PWS) in a single day (note: 99·95% of individuals have zero probability of infection – not illustrated). Inclusion of exposure information enables the number of cases to be determined: (*b*) food, (*d*) environment and (*f*) water.

between the risk proxies and this could be due to the geography of Grampian (e.g. PWS tend to be in rural farming areas where cattle and sheep are reared). The regression model could be applied at higher spatial resolution and seasonality could also be included. However, breaking the dataset down into smaller groups leads to deviation from the normal distribution. This would result in nonlinear regression techniques being required from which the number of cases associated with each pathway cannot be readily estimated. The regression model generates a good correlation between the predicted and actual number of cases but a disadvantage is that because of its simplicity it is not readily operational to evaluate risk mitigation strategies. This can of course be achieved by QMRA [27]. An error would occur in cases where the illness is contracted outside their home postal district [e.g. the person may have visited the countryside, acquired the disease from coming into contact with animals there, then returned to an urban home and subsequently fallen ill (there is a period of 3–4 days before symptom onset [37]). This would

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result in an underestimate of the importance of the environment while food would be overestimated. It is not possible to resolve this false attribution without further epidemiological data.

All of the QMRAs over-predicted the number of human cases. This suggests one or more common factors associated with the models may be error prone, e.g. the assumption that all *E. coli* O157 are equally infective (recently demonstrated not to be the case [38]) and that all organisms are released effectively from faecal material (this is reported not to be the case [39]) [17]. In addition, a proportion of the human population is likely to be immune [40] (e.g. from repeated exposure to a contaminated PWS or working on a farm that has cattle or sheep shedding this pathogen) and asymptomatic cases may be common (e.g. up to 40% of secondary [17] and between 4–15% of sporadic [36] cases). Further, the dose–response model chosen has an influence; the Cassin dose–response model was used here and another dose–response model (determined from *E. coli* O157 outbreaks) was also implemented [41]. This latter model predicted even more cases (60-fold higher than reported; data not presented). The Cassin model is a surrogate model parameterized using data from *Shigella* feeding studies. The Teunis model is based on *E. coli* O157 outbreak data which are relatively few and could involve strains that have higher than average infectivity and by definition expose a susceptible population.

The QMRAs described do not cover all the potential pathways of infection in humans. For example, foods other than burgers were not considered in this study, but are known to be associated with *E. coli* O157 infection in the Grampian region of Scotland (e.g. lettuce (www.eurosurveillance.org/ViewArticle.aspx?ArticleId=3273) and cheese [42]). Further, application of manure to pasture would pose a risk to visitors. However, if the manure had been sufficiently stored/treated [43] prior to application then it is likely that this risk would be much smaller than that from freshly voided faeces. Hence it is likely that in this light the QMRAs overestimate the number of cases. For the waterborne pathway, risks associated with swimming and water sports are not included. The average annual exposure to this pathway is more than one order of magnitude lower than from drinking water from a PWS [44]. In summary, combining the three QMRAs in this paper is a simplification of the real world. To obtain a more realistic representation, QMRAs should be developed for other vehicles and included in the analysis. This should be a focus of further research. Thus, the approach taken here will actually underestimate the number of human cases as all possible vehicles are not included. Regarding person-to-person transmission it is assumed in this paper to be a constant factor by pathway and age. It is known that secondary transmission is more important in children [17, 36] and that this is also likely to be the case in the elderly. It would be useful to incorporate data into the risk models on not just age but living conditions (e.g. attending a nursery or living in a care home) and also behaviour (hand washing, etc.). This would be possible to implement in a QMRA but would need a dynamic component [45]. Consumption of burgers has been found not to vary between urban and rural areas [44] and hence this does not explain the excess number of cases in rural areas (Fig. 2). Further, burger consumption is greatest in children aged >5 years old and so cannot explain the high incidence in children aged <5 years. Finally the 'riskier' practice of eating rare burgers is more associated with the adult section of the population.

The QMRAs facilitated the evaluation of a range of putative mitigation strategies *in silico*. However, these strategies need to be considered not only in terms of their efficacy to reduce *E. coli* O157 but also in terms of their practical implementation, acceptability and cost.

In terms of foodborne infection by burgers, a mitigation step is the removal of high-shedding animals prior to slaughter (identified using a cowside test) or alternately reducing shedding levels in ruminants. A cowside test, based on immunoassays, currently has a sensitivity limit of about 10^4 c.f.u./ml [46] which is insufficient to identify animals to achieve the potential 50% reduction in cases. A 1 $\log_{10}$ increase in sensitivity is preferable. Therapeutic agents [46] can reduce shedding by 2 $\log_{10}$ units when administered orally and by 4 $\log_{10}$ units with rectal application. However, timing and practicality of application would have to be considered. A review of the effects of diet and dietary supplements has been carried out [47] investigating the roles of fasting, feed additives (probiotics and antimicrobials) as well as dietary changes but concluded that much of the research was unclear with conflicting results. However, abruptly changing diet from a high proportion of grain to a high proportion of hay did reduce *E. coli* O157 shedding, but the practicality of carrying this out prior to slaughter might be difficult. Further, the dynamics of shedding *E. coli* O157 is unclear; animals tested (at the farm or

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lairage) as being above a safe threshold might be below that value at slaughter, and vice versa.

In terms of treating the carcass at the abattoir, a number of treatments have been evaluated, e.g. hand trimming followed by spray washing achieved a reduction of between 1·4–2·5 $\log_{10}$ of *E. coli* counts [48]. In small slaughter plants [49] a range of treatments were investigated [e.g. dry ageing, sprays of hot water (low and high pressure) or acetic acid] and found to have similar effects resulting in the reduction of Enterobacteriaceae by 0·6–2·0 $\log_{10}$. Steam treatment [50] applied to beef carcasses achieved 0·8 $\log_{10}$ reduction. Implementation in the abattoir would inevitably be costly in terms of equipment, labour and possible reduced processing speed. Finally, an educational campaign explaining the importance of correct cooking procedure for minced beef might achieve a reduction in cases and would also mitigate the risk from other gastrointestinal pathogens (e.g. *Salmonella*) that can be found in these products.

The environmental risk mitigations demonstrate that washing hands can potentially lead to a significant reduction in infection rates. Indeed, if there is a >3 $\log_{10}$ reduction in *E. coli* concentration by hand washing using antimicrobial soap [51] it is predicted that this could lead to a reduction of 99·9% of environmental infections. However, it is unknown how many existing cases had previously washed their hands before falling ill. It has been reported [35] that washing hands prior to eating food in a camp-site contaminated with *E. coli* O157 by sheep faeces reduced the infection rate significantly compared with those who had not. Implementing hand washing in rural settings with limited clean water may be problematical. Alternatives, e.g. ethanol-based antibacterial gels may be more appropriate and have been demonstrated to be equally effective as soap in an agricultural setting where >1 $\log_{10}$ reduction was achieved in more than 50% of those participating [52]. Further, it is worth noting that those aged <5 years are at greatest risk and stopping hand–oral contact would be very difficult to achieve in this age group.

Reducing *E. coli* O157 concentration shed by animals in the environment is a greater challenge than prior to slaughter as a long-term solution is preferable as opposed to a short-term measure, e.g. abrupt feed change. Banning of camping in areas recently grazed by farm animals is a potential strategy but is difficult to enforce and compulsory measures might be considered counter-productive. However, advising people that camping in this situation is a health risk does offer the opportunity of allowing individuals to make an informed decision.

Reducing infection rates by 50% from the PWS pathway would involve increasing the percentage of PWS treated from 48% to $\sim$80%. Grants for water treatment are currently available but many owners/users do not perceive PWS as a significant health concern [53]. Specifically, treating those PWS with high contamination levels could also potentially lead to a 50% reduction of cases but requires testing prior to targeting installations; further, one-off tests are inadequate to quantify microbiological quality. In the QMRA a 2 $\log_{10}$ reduction is simulated for treated PWS; however, some treatment methods are more effective than others. For example, UV treatment can reduce *E. coli* or *E. coli* O157 by 1·5–6 $\log_{10}$ [54, 55], chlorination by 3–5 $\log_{10}$ [56] and sand filtration by 0·3–4 $\log_{10}$. Banning the use of PWS in areas with high animal densities appears to have a relatively small effect on the number of cases and might be totally impractical.

A reduction in prevalence of between 50% and 75% in the ruminant population would potentially reduce infection by $\sim$50% in all of the QMRAs studied. There are a number of potential strategies to consider. A two-dose vaccine regimen reduced positive faecal samples from feedlot cattle and hides by $\sim$50% [57]. Farming practices also have the potential to reduce prevalence, e.g. not mixing young and old stock and not mixing cattle and sheep both achieved a 50% reduction in prevalence [58] while reducing cattle stocking density by 50% resulted in a 25% reduction in prevalence [59].

CONCLUSIONS

The results presented here demonstrate that the sources of *E. coli* O157 infection in humans are multifactorial but that environmental and foodborne routes are the most important. It should be borne in mind that the models do not account for all the possible transmission pathways and further research is required to fill this gap. The epidemiology reinforces the point that young children are at greatest risk. To reduce infection rates it is important that people wash their hands properly after contact with farm animals or their faeces and that educational campaigns highlight the importance of correct cooking practices and the risks associated with visiting the countryside (e.g. contact with farm animals). Although waterborne infection appears to be the least important

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pathway, encouragement to improve take-up of grants to treat PWS (particularly those with high indicator counts) will still have an effect on reducing human cases of disease. Despite their limitations, the combination of QMRAs, spatial regression models and epidemiology all contribute to an improved understanding of how humans become infected by this pathogen.

NOTE

Supplementary material accompanies this paper on the Journal's website (http://journals.cambridge.org/hyg).

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DECLARATION OF INTEREST

None.

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