Quantitative risk assessment of human infection from *Escherichia coli* O157 associated with recreational use of animal pasture

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Abstract

A quantitative microbial risk assessment incorporating Monte Carlo simulations is described which estimates the probability of *Escherichia coli* O157 infection of humans by visiting pasture previously grazed by cattle. The risk assessment is performed for a number of scenarios including a variation in the grazing period prior to the human visit, the duration of visit (8-h day or 24-h camp) and the level of *E. coli* O157 shed by the cattle. Assuming the cattle have been on the field for 28 days, followed directly by a human visit, and the proportion of animals shedding the organism are as described in previous surveys 5 ± 1% (Synge, B.A., Gunn, G.J., Ternent, H.E., Hopkins, G.F., Thomson-Carter, F., Foster, G., Chase-Topping, M., McKendrick, I., 2001). Prevalence and factors affecting the shedding of verocytotoxin producing *Escherichia coli* O157 in beef cattle in Scotland. In: Concerted Action CT98-3935 Verocytotoxigenic *E. coli* in Europe, 5. Epidemiology of Verocytotoxigenic *E. coli*, Dublin, pp. 98–103.), a probability of infection of 0.1% is attained for 8- and 24-h periods when the cattle are shedding approximately 10^3 and 10^4 CFU g^-1, respectively. Monte Carlo simulations demonstrated that risk mitigation strategies of removing cattle from the pasture 4 weeks prior to the human visit in addition to physical removal of faeces showed significant reductions in potential infection rates. © 2002 Elsevier Science B.V. All rights reserved.

Keywords: Quantitative risk assessment; Monte Carlo simulations; *Escherichia coli* O157; Environmental pathogens; Risk mitigation

1. Introduction

*Escherichia coli* O157 is a pathogen which is of concern worldwide with numbers of outbreaks continuing to rise in the UK (Jones, 1999). A number of environmental and food-borne sources have caused major *E. coli* O157 incidents, e.g. Illinois bathing water outbreak in 1995 where 12 people were infected (Anonymous, 1996) and the Central Scotland outbreak in 1996 where the consumption of contaminated meat led to the direct death of 17 elderly people and more than 500 falling ill (Ahmed and Donaghy, 1998). The major reservoirs of this pathogen are ruminants including beef and dairy cattle herds (Synge et al., 2000; Wallace, 1999; Cobbold and Desmarchelier, 2000) and sheep (Chapman et al., 2000; Kudva et al., 1996).

There is increasing evidence (Locking et al., 2000) of environmental infection by *E. coli* O157, particularly in children, associated with farm and countryside visits (Trevena et al., 1996). A recent outbreak in a
scout camp which took place on a pasture previously grazed by sheep at New Deer, North East Scotland resulted in 18 of the 226 scouts falling ill (Anonymous, 2000; Grampian Health Board, 2000). A point source outbreak arising from the sheep was suspected and \( E. \ coli \) O157 was isolated from samples of soil, standing water and sheep faeces taken from the field during the week following the outbreak (Strachan et al., 2001). The likely route of transmission was via hands contaminated with mud and faecal material. It was

\[ \text{Fig. 1. Flow chart of the Monte Carlo model for calculating the probability of infection in humans from visiting contaminated pasture. The number of pastures PN used was 10,000 and the number of people NP visiting each pasture was set at 100.} \]
estimated that the dose ingested by the scouts ranged between 4 and 24 organisms which is in agreement with the low infectious dose for this organism (≤10 viable cells, Griffin and Tauxe, 1991 and ≤few hundred, Doyle et al., 1997). As a result of outbreaks, the regulatory authorities in Scotland established the ‘E. coli Task Force’ which has published guidance on recreational use of animal pasture (Food Standards Agency/Scottish Executive Task Force on E. coli O157, 2001) and suggests risk mitigation strategies including the removal of animals from pasture three weeks prior to use by humans and the physical removal of visible faecal waste.

Quantitative risk assessment (QRA) is a technique, which is used to estimate the likelihood and severity of an adverse event (Cassin et al., 1998). When performed in conjunction with Monte Carlo simulation, QRA offers precise explanation of the uncertainty and variability associated with the risk (Vose, 2000). Quantitative microbial risk assessments have been performed to estimate infection rates in humans, e.g. E. coli O157 in beef burgers (Cassin et al., 1998), Cryptosporidium in drinking water (Gale, 1998) and Listeria monocytogenes in smoked salmon and trout (Lindqvist and Westoo, 2000). The terminology in risk assessment is not yet fixed but after an initial statement of purpose (Codex Alimentarius Commission, CAC, 1998; Vosey and Brown, 2000) the process involves four primary stages described below in relation to this current study (WHO/FAO, 1995; European Commission, 1997; Codex Alimentarius Commission, CAC, 1998).

1. Hazard identification—identifies the pathogenic microorganism of concern and whether it is actually a hazard in the context that it is being studied.
2. Exposure assessment—aims to determine the number of microorganisms ingested.
3. Hazard characterisation—gives a quantitative or qualitative assessment of the adverse effects of the pathogen to humans. More specifically a dose–response model can be implemented which mathematically models the variability in impact (response) following exposure to different doses (McNab, 1997).
4. Risk characterisation—gives a probability of occurrence of the illness and also the severity of the health effects in a given population.

These stages will be followed through the risk assessment described below, the objective of which (analogous to the statement of purpose of the risk assessment) is to determine the probability of E. coli O157 infection associated with visiting farm pasture previously grazed by ruminants. The effectiveness of proposed risk mitigation strategies, namely removal of animals from the field for a fixed period prior to the human visit and physical removal of faeces from the field will be estimated. This study is based on cattle data because they are readily available from prevalence studies carried out in Scotland. Risk assessments were performed under a series of different scenarios which include duration of the human visit to the pasture (overnight camp/day visit), length of time the animals grazed the field prior to the human visit and variation in concentration of organism shed by the animals.

2. Materials and methods

A flow chart detailing the steps in the risk assessment model is given in Fig. 1. Table 1 shows the different scenarios studied. The input data for the model were obtained from the scientific literature, ongoing studies performed by the authors and expert opinion, and are parameterised in Table 2. The model was con-

<table>
<thead>
<tr>
<th>Scenario no.</th>
<th>Scenario</th>
<th>Base scenario value</th>
<th>Importance analysis probability distributions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No. of days animals on field prior to human visit ( (d) )</td>
<td>28</td>
<td>RiskUniform(1,28)</td>
</tr>
<tr>
<td>2</td>
<td>No. of days animals removed before human visit ( (d_r) )</td>
<td>0</td>
<td>RiskUniform(0,28)</td>
</tr>
<tr>
<td>3</td>
<td>Type of visit (Day 8 h = 0, Camp 24 h = 1)</td>
<td>0 or 1</td>
<td>RiskDiscrete{0,1, {50,50}</td>
</tr>
<tr>
<td>4</td>
<td>Number of E. coli O157 excreted/g. If scenario is cattle, then 0 = use of Zhao et al.(1995) data or 1 = selection of a fixed input value</td>
<td>0 or 1</td>
<td>RiskUniform(1,7)</td>
</tr>
<tr>
<td>5</td>
<td>Removal of faeces from field ( (\text{No} = 0, \text{Yes} = 1) )</td>
<td>0</td>
<td>RiskDiscrete{0,1, {50,50}</td>
</tr>
</tbody>
</table>

* Defines the log number of E. coli O157 shed/g in faeces.
structured in Microsoft Excel™ using the @RISK™ software (Palisade, Ivybridge, UK) which described the variability and uncertainty of the input variables by probability distributions and enabled Monte Carlo simulations of the model to be performed. Ten thousand iterations were performed for each simulation, incorporating Monte Carlo sampling which enabled convergence of the simulation statistics (± 1.5%) (Morgan and Henrion, 1990).

This paper does not follow the usual format of scientific papers, as it gives some results prior to the formal results section. This is done for ease of interpretation of the model and of the data presented.

3. Risk assessment

3.1. Hazard identification

In this risk assessment, the hazard is E. coli O157, a microorganism which can be shed by farm ruminants resulting in human infection following ingestion of contaminated soil/faeces and water. Symptoms of this disease include bloody diarrhoea and stomach cramps. Those at greatest risk of infection are the immunocompromised and in particular the elderly and young children where the potent verotoxin generated by the organism can cause haemolytic uraemic syndrome (HUS) (mainly occurring in children), thrombotic thrombocytopenia purpura (in adults) and in the most severe cases, death (O’Brien, 2000). Risk factors which have been strongly associated with this disease include likelihood of contact with farm animals or their faeces (Reilly et al., 2000).

3.2. Exposure assessment

3.2.1. Prevalence

Several prevalence studies have been performed looking for pathogen presence in the faeces of cattle. Synge et al. (2000, 2001) showed that 22.6% of cattle herds in Scotland are positive for E. coli O157 with 9% of cattle carrying the pathogen. For cattle grazing on pasture, the prevalence was lower with 5% of individuals excreting the organism and 23% of herds positive. Paiba (2000) reported that 44% of randomly selected herds in England and Wales contained animals shedding E. coli O157. In the USA and Canada, a review of six prevalence studies of E. coli O157 in cattle showed that between 0% and 3% shed the organism (Cassin et al., 1998). The first entry in Table 2 summarises (in the form of a normal distribution) the prevalence data selected for the current study which comprises the cattle pasture data of Synge et al. (2000, 2001).

3.2.2. Concentration

3.2.2.1. Concentration in cattle faeces.

Data detailing concentrations of E. coli O157 shed by North American cattle are given in Table 3. These data were used in the risk analysis using the @RISK™ RiskCumul() function. Briefly, a cumulative distribution is
generated from the data. Then for each individual animal in the herd which is shedding the organism the distribution is sampled to generate a representative concentration of the pathogen being shed.

These North American data (Zhao et al., 1995; Shere et al., 1998) may be quite different to shedding rates in Scotland and throughout the UK. In the UK, it has been shown that sheep can shed up to the range of $10^6$–$10^7$ CFU/g (Strachan et al., 2001) while in Australia total shiga-toxin producing *E. coli* in cattle can shed at levels approaching $10^7$ CFU/g (Vanderlinde, 2001). Therefore, the risk assessment was run for a range of scenarios of animals shedding between $10^1$ and $10^7$ CFU/g. This was done to determine the risk of human infection when visiting pasture with animals shedding at different rates.

### 3.2.2.2. Concentration on the pasture.

It is unlikely, except in the case of very young children, that direct consumption of animal faeces will take place. However, once faeces have become mixed with soil, ingestion is more likely. The model developed in this paper, assumes that the faeces is mixed homogeneously in the top 1 cm of soil which is in broad agreement with the experimental results (Fenlon et al., 2000; Strachan et al., 2001).

Studies (Wang et al., 1996; Himathongkham et al., 1999; Fenlon et al., 2000) have described the fate of *E. coli* O157 in cattle faeces and slurries. The work by Wang et al. showed that at warmer temperatures (22 and 37 °C), there is initial growth of the organism followed by rapid decline. This rapid decay at elevated temperatures is in agreement with that reported by Himathongkham et al. (1999). At the lower temperature of 5 °C, Wang observed no growth of the organism but a decimal reduction time ($t_{drt}$) in the order of 15 days, which is in close agreement with field studies of *E. coli* performed by Ogden et al. (2001b) which yielded a $t_{drt}$ of $16 \pm 2$ days. This is the $t_{drt}$ used in this study. It should be noted that other factors, such as UV from sunlight, humidity and also length of grass may have an effect on $t_{drt}$ but currently no comprehensive data exist that could be built into the current model.

Fig. 2a gives an example of build-up of the organism on the pasture excreted by cattle. It shows that the rate of build-up is initially rapid but reduces with time and after 4 weeks the concentration of organisms on the pasture is almost constant. The build-up of *E. coli* O157 on the field ($N$ organisms per cm$^2$) after $d$ days can be described in the following equation where $N_0$ is the average number of organisms shed per day per cm$^2$

\[
N(t) = N_0 \times \frac{1}{2} \left( 1 + e^{-kt} \right)
\]

where $k$ is the decay rate and $t$ is the time in days.

#### Table 3
Concentration of *E. coli* O157 in faeces of shedding cattle (Zhao et al., 1995)

<table>
<thead>
<tr>
<th>Concentration in faeces [log$_{10}$ CFU/g]</th>
<th>Cumulative number of animals</th>
<th>Percentile (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1$^a$</td>
<td>0/31</td>
<td>0</td>
</tr>
<tr>
<td>&lt; 2$^b$</td>
<td>15/31</td>
<td>48</td>
</tr>
<tr>
<td>&lt; 3</td>
<td>17/31</td>
<td>54</td>
</tr>
<tr>
<td>&lt; 4</td>
<td>28/31</td>
<td>90</td>
</tr>
<tr>
<td>&lt; 5</td>
<td>31/31</td>
<td>100</td>
</tr>
</tbody>
</table>

$^a$ Minimum concentration of *E. coli* O157 in contaminated cattle faeces was 0.1 CFU/g based on a positive isolation from a 10-g enriched sample.

$^b$ $10^2$ CFU/g limit of detection of plating method for cattle samples.

![Fig. 2](image_url)

(a) Build-up of *E. coli* O157 on pasture during a 4-week period, assuming a herd of 25 cattle, 5 of which are shedding the organism at an average concentration of $10^4$/g. (b) Subsequent decay of the organism on the pasture after removal of cattle.
and \( \tau_{\text{drt}} \) is the decimal reduction time of the organism in days.

\[
N = \sum_{t=0}^{d-1} N_0 10^{-t/\tau_{\text{drt}}}
\]

If the animals are removed from the field for \( d_t \) days prior to the human visit then the numbers of organisms (\( N \)) per cm\(^2\) on the field will decay (Fig. 2b) to \( N_t \) according to:

\[
N_t = N 10^{-d_t/\tau_{\text{drt}}}
\]

### 3.2.3. Consumption/Ingestion

The ingestion of soil from the pasture will depend upon the duration of the visit and to an extent on the age of the individual (Environmental Protection Agency, 1996). Van Wijnen et al. (1990) report that soil intake by children on a camp site ranges from 30 to 200 mg per 24 h, while Haas (2000) estimates ingestion per working day by agricultural workers to have a median value of approximately 10 mg. When performing chemical risk assessments, the United States Environmental Protection Agency assumes a daily soil ingestion rate of 200 mg for each 24-h period (EPA, 1996). In this study, two scenarios with different amounts of soil ingested by humans are considered. The first involves a 24-h stay including an overnight camp and uses the soil ingestion data of van Wijnen et al. (1990) and the second involves a day visit (8 h) and uses the data supplied by Haas (2000). In the Monte Carlo model, the variation in soil ingestion for each of these two scenarios is described by Triang distributions (Table 2).

Knowing the mean number of organisms per cm\(^2\) on the pasture (\( N \)), the bulk density of the soil (\( \rho_b \)) and with the assumption that all of the organisms are contained within the top 1 cm of soil, then the number of organisms per gram in the soil can be calculated (= \( N \rho_b \)). Assuming that the organisms are Poisson distributed and determining the quantity of soil ingested by sampling the appropriate Triang distribution detailed in Table 2, the quantity of organisms ingested by each human visiting the pasture can be calculated.

### 3.3. Hazard characterisation/dose–response assessment

Data from two different sources have been used in the development of dose–response models for \( E. coli \) O157. The first (Crockett et al., 1996; Cassin et al., 1998) utilises different species of \( Shigella \) as a surrogate and is based on three feeding studies in humans. The second (Haas et al., 2000) is based on rabbits inoculated with 1 ml of \( E. coli \) O157 suspension through an oral catheter. The two dose–responses are considerably different with the rabbits requiring approximately 500-fold more organisms to cause 50% infection compared with the human data. In the current study, the surrogate \( Shigella \) dose–response model has been selected because it closely fits the outbreak in scouts at New Deer (Strachan et al., 2001) and appears to be closer to other recorded outbreak data (Ogden and Strachan, data not presented). The format of the model used is as described in Cassin et al. (1998) and is given in Table 4. Briefly, this Beta-binomial model assumes that only a single organism is required to cause infection and that each cell is equally infective. Two variables, \( \alpha \) and \( \beta \), are used to parameterise the model and are calculated from data obtained in three published human studies. The uncertainty in the values of \( \alpha \) and \( \beta \) is incorporated using study intervariability as a proxy. This results in variation of probability of infection at a given dose (Fig. 3).

### 3.4. Risk calculation

The Monte Carlo model was performed for the scenarios detailed in Table 1. For each iteration of the Monte Carlo simulation, it was assumed that 100 people visited the pasture and this was repeated 10,000 times. The risk of infection to each individual human visiting the pasture was calculated by entering the dose of organisms ingested into the dose–response model. It should be noted that if the dose was predicted by the model to be < 1 bacterium then this

<table>
<thead>
<tr>
<th>Variable</th>
<th>Description</th>
<th>Distributional assumption</th>
</tr>
</thead>
<tbody>
<tr>
<td>( P_I(D) )</td>
<td>Probability of infection from dose</td>
<td>( P_I(D) = 1 - (1 - P_I(1))^{D/\beta} )</td>
</tr>
<tr>
<td>( P_I(1) )</td>
<td>Probability of infection from a single organism</td>
<td>Beta(( \alpha ), ( \beta ))</td>
</tr>
<tr>
<td>( \alpha )</td>
<td>Susceptibility parameter</td>
<td>0.267</td>
</tr>
<tr>
<td>( \beta )</td>
<td>Susceptibility parameter</td>
<td>ln(( \beta )) ( \approx ) Normal(5.435, 2.47)(^a)</td>
</tr>
</tbody>
</table>

\(^a\) Normal (\( \mu_B \), \( \sigma_B \)).
was interpreted as the probability of a bacterium being present (e.g. if dose was 0.4, then probability of a single bacteria being present was 0.4. Since a fractional bacteria does not exist a random number between 1 and 0 was generated. If the random number was \( \geq 0.4 \), a bacterium was considered to be present otherwise there was no bacterium present).

### 3.5. Risk characterisation

The probability of infection for a person visiting a pasture is calculated using the Monte Carlo model. In this study, we consider a visit comprising either a single 8-h day visit or a 24-h overnight camp. The risk is the probability of infection with \( E. coli \) O157 associated with the visit. The effects of \( E. coli \) O157 infection can be relatively minor to extremely severe as stated above (O’Brien, 2000). Reilly et al. (2000) reported that in a study of 183 \( E. coli \) O157 cases in Scotland, 44% were in children under 10 years of age, 77% of cases reported bloody diarrhoea, 57% were admitted to hospital and 8% developed HUS. Since these cases have a significant positive association with visits to farms or farm animals, it is assumed that the proportion of symptoms/outcomes the illness takes will be replicated in the cases predicted in the current study.

### 4. Results and analysis

#### 4.1. Base results

The base results for cattle are detailed in Figs. 4 and 5. As is expected, the risk of infection is greater when the levels of pathogens being shed onto the field is high. The probability of a human becoming infected from camping on pasture that has held animals shedding according to the data of Zhao et al. (1995) is approximately 0.1%. Fig. 5a shows that when human infection does occur, only between 1% and 8% of people visiting the pasture will become infected. Only in exceptional cases (9 in 10,000 events, i.e. iterations using Monte Carlo simulations, of 100 people visiting the pasture) will more than 5% of people become infected. Using the same conditions, the probability of infection for the 8-h day visit is lower (approximately 0.01%) with a maximum of 3% of people becoming infected (Fig. 5b). However, it should be noted that if the cattle are found to shed \( 10^4 \) CFU/g (compare to mean shedding rate of approximately 660; Zhao et al., 1995) then the probability of infection would be more significant at approximately 0.1%. Fig. 5c and d shows the range of numbers of people becoming infected for cattle shedding at a high concentration of \( 10^6 \) CFU/g. These graphs demonstrate the importance of volume of soil ingestion (e.g.  

![Graph of Probability of infection against ingested dose](image-url)

**Fig. 3.** Probability of infection against ingested dose (Cassin et al., 1998).

![Graph of Probability of infection with E. coli O157 shed by cattle](image-url)

**Fig. 4.** Base results for: (a) 24-h human camp and (b) 8-h day visit to the pasture (Note: when cows shedding \( 10^4 \) CFU/g no infections are detected in simulations and hence probability of infection \(<0.0001\%\)). Cattle are on field for 28 days prior to human visit that occurs directly after the cattle are removed from the field.
from day visit or overnight camp) and also the importance of the level of concentration of shedding.

4.2. Importance analysis

An importance analysis was performed to determine which parameters in the model were most significantly correlated with the probability of infection. The different scenarios (Table 1) are now included as probability distributions. This included both the number of days the animals were on the pasture prior to the human visit and the number of days the field was left fallow (both up to a maximum of 4 weeks). The concentration of E. coli O157 shed was allowed to vary uniformly between $10^1$ and $10^7$ CFU/g. The type of visit (8-h or 24-h camp) and whether faeces were removed from the field was allowed to vary discretely (e.g. there was a 50:50 chance of the exposure being 8 or 24 h).

Fig. 6 shows the Spearman rank correlation coefficients (Morgan and Henrion, 1990). The larger the magnitude of the correlation coefficient between the model parameter and the probability of infection, the stronger the association and hence the greater the importance of the model parameter. The model param-
eters could either have a positive or negative correlation, i.e. a positive correlation would mean that if the model parameter increased then the probability of infection would increase also.

The concentration of *E. coli* O157 in faeces is the most important parameter and has a positive correlation. This demonstrates that as the concentration increases, then the probability of infection also increases. The type of visit also shows an important positive correlation. For longer visits (24-h camp), the probability of infection increases due to increased ingestion of soil quantity and hence increase in numbers of pathogen ingested. The host susceptibility (the probability of infection by a single organism) defined in the dose–response model is an important parameter and is also positively correlated. This result is expected because the greater the susceptibility of the individual then the greater the probability of infection. The other positively correlated variables are less important and it is worth noting that this includes duration of time the animals are on the field. It can be seen from Fig. 2a that the build-up of pathogen on the pasture does not increase at a significant rate once the cattle have been on the field for more than 8–9 days and hence the low correlation result was expected.

There were two important parameters with a negative correlation. The number of days the animals were removed from the field prior to a human visit and the removal of the faeces from the pasture. These correlations demonstrate their potential with regard to risk mitigation.

### 4.3. Risk mitigation strategies

The Monte Carlo model can be amended to take into account a single input parameter change or alternately a change in one of the assumptions upon which the model is based. This is particularly important when considering hypothetical risk mitigation strategies. These strategies can be implemented in the model and the change in the output (i.e. probability of infection) can be calculated to determine whether it is significant or not. The following strategies were considered.

**Strategy 1: Keeping farm animals off the pasture prior to the human visit.** The base results assumed that the humans visited the pasture directly after the animals had been removed. A mitigation strategy of ensuring animals were kept off the field for a fixed period of time prior to the visit was considered.

**Strategy 2: Physical removal of faeces from the pasture.** Removal of pathogen (in faeces) from the field should also help reduce risk. However, efficient removal is hard to achieve due to mixing of faeces with soil, particularly during wet conditions. We estimated between 30% and 70% of faeces could be removed from pasture land which was modelled as a Triang distribution with a most likely value of 50%. We assumed that the percentage removal of faeces corresponded directly to the percentage reduction in *E. coli* O157.

**Strategy 3: Combination of strategies 1 and 2.**

#### 4.3.1. Comparison of strategies

Table 5 shows the results of the risk mitigation strategies for cattle. Both the main strategies show significant reductions in probability of infection. Removal of faeces from the field has approximately the same effect as a 5-day fallow period. It appears that there is only a small reduction in the probability of infection by removing faeces from the field following a 4-week lay period. Considering the mitigation strategy applied to cattle shedding according to the data presented by Zhao et al. (1995) (see Table 3), suggests that a 4-week lay period, with or without the removal of

<table>
<thead>
<tr>
<th>Strategy</th>
<th>Days ($d_r$) cattle off field prior to human visit</th>
<th>Predicted reduction in $10^6$ CFU/g</th>
<th>Predicted reduction in infection (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Keeping farm animals off the pasture prior to the human visit</td>
<td>7</td>
<td>24%</td>
<td>54%</td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>48%</td>
<td>80%</td>
</tr>
<tr>
<td></td>
<td>21</td>
<td>67%</td>
<td>93%</td>
</tr>
<tr>
<td></td>
<td>28</td>
<td>81%</td>
<td>98%</td>
</tr>
<tr>
<td>(2) Physical removal of faeces from the pasture</td>
<td>0</td>
<td>19%</td>
<td>40%</td>
</tr>
<tr>
<td>(3) Combination of strategies 1 and 2</td>
<td>7</td>
<td>41%</td>
<td>75%</td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>64%</td>
<td>92%</td>
</tr>
<tr>
<td></td>
<td>21</td>
<td>78%</td>
<td>98%</td>
</tr>
<tr>
<td></td>
<td>28</td>
<td>88%</td>
<td>99%</td>
</tr>
</tbody>
</table>

The % reductions in infection are related to baseline results assuming cattle had been on field 28 days followed immediately by 24-h camp.
faeces, will reduce risk of probability of human infection to below maximum tolerable levels, i.e. \(< 0.01\%\) (Health and Safety Executive, 1992; Comer et al., 1998). However, this must be treated with caution as the data used (Zhao et al., 1995) may not be representative of UK cattle and is based on only 31 individual animals shedding the organism. There is the possibility of an individual herd shedding at significantly higher numbers and it seems from a practical point of view, simply leaving the field fallow for a period of 3 or 4 weeks is the easiest option to implement. This of course requires planning of the human visit to the pasture in advance which may not always be possible.

5. Discussion

The Monte Carlo model was able to predict the probability of infection for the different scenarios studied. The model showed an increased risk of infection depending on the duration of the human stay on the pasture. It also showed that the relationship between average organisms shed onto the field and probability of infection was non-linear, particularly at low shedding levels. It is not possible to relate these data directly to the number of cases of \textit{E. coli} \textit{O157} infection in Scotland because of a number of unknown factors including: the number of people visiting/camping on pastures during a year; whether the pastures have had animals grazing on them directly before the camp/visit or if there has been a lay period; the actual concentration of pathogens the cattle are shedding and also the number of cases of \textit{E. coli} \textit{O157} which can be attributed each year to contact with farm animals via pasture. However, for comparative purposes the model gives an indication of the relative risk for each of the scenarios described in this study.

The Monte Carlo model is based on a number of assumptions that need to be considered carefully. The first is the assumption that the faeces shed by the cattle is mixed thoroughly with the topsoil/grass in the pasture. This is most likely to be the case when the weather is wet. However, in drier conditions ‘hotspots’ in the field may occur, particularly if the animals have preferred areas for grazing and defecating. The authors have developed some preliminary models addressing this issue (data not presented) and have compared two pastures with the same total microbial load, one with hotspots, the other without. The authors found that the higher microbial loads in the hotspots were offset by the fact that the probability of ingestion of soil from the hotspot was correspondingly reduced. This argument is in agreement with Gale (1998) when discussing the heterogeneity of \textit{Cryptosporidium parvum} oocysts in drinking water.

The second assumption relates to the values of the parameters used in the model, in particular, parameterisation of the dose–response model, the decimal reduction time, the number of herds which carry the pathogen and the number of individual organisms shed by the cattle. We present data that are currently available and are aware that in some cases validation is incomplete.

The dose–response model generated an average probability of infection for the human population. However, it must be remembered that the immuno-compromised (e.g. the old and very young) are at greatest risk to infection and also the severity of infection. For example children under 10 years of age are approximately twice as likely to be admitted to hospital and more likely to develop HUS than adults (O’Brien, 2000). It must be noted that it is unknown in the UK whether farmers and their families have an unusually high morbidity rate for this pathogen since their exposure to \textit{E. coli} \textit{O157} must be higher than the general public’s. However, the farming community may have greater immunity which has been demonstrated for dairy farmers and their families in Canada (Wilson et al., 1996; Johnson et al., 1999).

The choice of decimal reduction time was dictated by the closest fit to existing data. However, it should be noted that at relatively warm temperatures, \(>20\,^\circ\text{C}\) \textit{E. coli} \textit{O157} can grow within cattle faeces by between 1 and 2 logs (Wang et al., 1996), which would result in potentially higher infection rates in humans. Following this growth, the organism then decays very rapidly. This fact emphasizes further the need for a fallow period to be introduced prior to recreational activity. In addition, recent work (Ogden et al., 2001a) has shown that a small percentage of the \textit{E. coli} \textit{O157} in soil survive for a much longer period than expected. It is possible that these organisms have found a protective niche in the soil environment. Since the proportion of organisms with this long survival factor is low \(<5\%\), Ogden et al., 2001b), they have been ignored in the current study as the probability of human infection arising from their direct ingestion is thought to be small.
Soil type may also need to be considered as Fenlon et al. (2000) showed decay to be faster in sandy soils compared with clay and loam.

The data used in this study for animal and herd prevalence assumed approximately 25% of herds were positive and within each positive herd there were approximately 20% of animals with *E. coli* O157. These data were based on surveillance data in Scotland, but data from elsewhere may be different and would require recalculation of the Monte Carlo model to enable accurate predictions of risk. The assumption of greatest concern is possibly that pathogen loadings in North American cattle (Zhao et al., 1995) were assumed to be the same for UK cattle; there is clear need for validation here.

The risk analysis performed in this paper has dealt directly with cattle, but sheep must also be considered. In the New Deer outbreak, it was estimated that approximately 50% of the sheep were positive and that animals were shedding between <10 and up to >10^6 CFU/g (Strachan et al., 2001). We estimated the number of *E. coli* O157 present at the time of the scout camp was approximately 60 CFU/g, which is equivalent in the current model to cattle shedding on pasture for 28 days at between 10^4 and 10^5 CFU/g. These shedding rates are higher than those for cattle generated from the Zhao data; however, these data may be atypical because they were not collected from a random sample (i.e. they were from sheep which caused a human outbreak). A waterborne outbreak at Applecross in Scotland was caused by faecal contamination from sheep where prevalence within the flock was considered to be much lower at 10% (Synge, 2001). The lack of *E. coli* O157 prevalence and concentration (n/g) data for sheep needs to be redressed by a comprehensive survey which would permit the risk of another outbreak such as New Deer occurring to be estimated.

Some of the risk factors identified in the importance analysis are not possible or very difficult to manage. For example controlling shedding is not yet possible. However, if mitigation strategies for this factor were established, perhaps by controlling feed or by vaccination (Jones, 1999). Then it is likely that the risk of infection could be substantially reduced.

The risk mitigation strategies studied in this paper and which were proposed by the *E. coli* Task Force, should significantly reduce the risk of *E. coli* O157 infection from recreational activities on pasture land. It must be noted that removal of faeces can be done most effectively directly after the animals have been removed from the field to minimise the amount of dispersion of the pathogen from the faeces into the soil/grass. However, the practical implementation of these procedures may not always be possible. For example a lay period of 3 weeks would almost certainly require cutting of the pasture grass and prior planning of the visit. Additional advice given by the Task Force which includes provision of hand-washing facilities, the adequate supervision of children and ensuring that drinking water from streams is treated are all prudent though again may not always be practical.

6. Conclusions

Quantitative microbial risk assessment is an important tool for assessing environmental risk both in the formulation of the risk analysis problem and in predicting the probability of infection for different scenarios. The risk mitigation strategies proposed by the *E. coli* O157 Task Force, i.e. requirement of a lay period of 3 weeks in addition to physical removal of faeces appear to be appropriate based on the currently available scientific data. There is the need to perform surveys to determine the pathogen loads being shed by both sheep and cattle in the UK to enable an accurate estimate of risk to be established. In addition, the prevalence of the organism between flocks of sheep requires investigation. These data are not only needed to ascertain the risk to the public from use of pastures for recreational purposes but are also needed to identify the potential loadings entering the food chain subsequent to slaughter.

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