environmental microbiology

Environmental Microbiology (2010) **12**(10), 2633–2644 doi:10.1111/j.1462-2920.2010.02293.x

Minireview

Cattle, weather and water: mapping *Escherichia coli* O157:H7 infections in humans in England and Scotland

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Summary

Entero-haemorrhagic *Escherichia coli* O157:H7 is a zoonotic pathogen, responsible for a relatively small number of food poisoning and illness outbreaks each year, when compared with other food-borne bacteria capable of causing infections in the population. Nevertheless, *E. coli* O157:H7 is a bacterial pathogen associated with severe human illnesses including bloody diarrhoea and haemolytic uremic syndrome occurring in both outbreak and sporadic settings. In England and Wales approximately 1% of all laboratory-confirmed cases of food poisoning are the result of *E. coli* O157:H7; however, in Scotland this figure increases to 3%. When the size of the population is taken into account and the rate of *E. coli* O157:H7 confirmed cases per 100 000 population is examined, the rate of *E. coli* 0157:H7 infections in Scotland is much greater than England and Wales. The routes of transmission have changed over time, with new routes of transmission such as farm visits emerging. The prevalence of *E. coli* O157:H7 has a seasonal dependency, with greater faecal shedding of the organism in the warmer months; this is directly mirrored in the increased reporting of *E. coli* O157:H7 infection among hospitalized patients. This review attempts to suggest why this phenomenon occurs, paying particular attention to weather, animal movement and private water supplies.

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Introduction

The *Escherichia coli* O157:H7 serotype was first isolated in the USA in 1975 from a sporadic case of haemorrhagic colitis (Riley *et al*., 1983; Wachsmuth *et al*., 1991). It was then described by Konowalchuk *et al*., in 1977 (Konowalchuk *et al*., 1977). Since then it has become the most important serotype because it causes the majority of cases of haemolytic uraemic syndrome (HUS) (Karmali, 1989). It was primarily recognized as a food-borne pathogen in 1982 following an outbreak of haemorrhagic colitis by the consumption of contaminated beef burgers (Riley *et al*., 1983). This microorganism has now been associated with many disease outbreaks throughout the UK, USA, Canada, Japan, Sweden to name but a few (Michino *et al*., 1999; Willshaw *et al*., 2001;Woodward *et al*., 2002 Cagney *et al*., 2004; Carroll *et al*., 2005; Sartz *et al*., 2008; Uhlich *et al*., 2008).

Originally, not much was known about the risk factors or complications involved in the infection; however, the 1996 outbreak in Scotland involved the largest group of people infected at that time, which highlighted the importance of this pathogen. Of the 120 patients affected, 34 people developed HUS, 28 were adults and 16 died (Dundas *et al*., 2001). Since then, infections like this have made *E. coli* O157:H7 a global phenomenon. The organism has a very low infectious dose, where less than 100 cells may be enough to cause an infection (Caprioli *et al*., 2005), which may be a reason for the high incidence. Studies have shown that *E. coli* O157:H7 is common in cattle and ruminants are considered to be the natural reservoir for this organism and rarely cause disease in their host (Caprioli *et al*., 2005). The organism has also been reported in sheep, goats, water buffalo and deer (Rabatsky-Ehr *et al*., 2002). Although ruminants including cattle, sheep and goats have been shown to be a reservoir for the organism

the infection rate in humans is relatively low considering the frequency of domestic/agricultural interaction, suggesting that not all strains of the organism are infectious to humans (Caprioli *et al*., 2005; Baker *et al*., 2007).

E. coli O157:H7 in animals and the environment

Cattle are asymptomatic carriers of the organism, and it can be a transient member of the ruminant gut micro flora. As a result carcasses may become contaminated with the pathogen following slaughter (Chapman *et al*., 2001). One study showed that it was present in 4/132 slaughterhouse samples tested (Carney *et al*., 2006). Outbreaks have also been potentially associated with private water supplies contaminated with bovine faeces (Schets *et al*., 2005), increased visits to dairy farms (Crump *et al*., 2002), petting farms and open zoos (Stirling *et al*., 2008). Grounds where sheep have previously grazed have also resulted in infection in scouts (Ogden *et al*., 2002) and also to people at music festivals (Crampin *et al*., 1999). Person-to-person transmission is also possible with cases being reported at nurseries (Al-Jader *et al*., 1999). Various food sources have also been implicated in infections with unpasteurized apple juice, cider, mesclun lettuce and white radish sprouts being quoted (Zhao *et al*., 1993; Cody *et al*., 1999; Hilborn *et al*., 1999; Michino *et al*., 1999). Recreational water sources have also been implicated in infection (Verma *et al*., 2007).

The *E. coli* O157:H7 strain has also been sporadically detected in animals other than cattle, such as wild birds in Scotland (Wallace *et al*., 1997; Foster *et al*., 2006). One study has shown that although *E. coli* O157:H7 was present in seagulls, pigeons and chickens, only similar variants of Shiga toxin 2 (stx2) found in enterohaemorrhagic *E. coli*, designated Shiga toxin 2f (stx2f) were found in pigeons (Kobayashi *et al*., 2002). It has also been found in horses (Chalmers *et al*., 1997) and rabbits, including wild rabbits (Scaife *et al*., 2006; Assies *et al*., 2007). However, in many of these cases it is not clear whether they represented the actual hosts or infected individuals that may have had contact with the cattle faeces, although one case implicates rabbit faecal material more explicitly (Pritchard *et al*., 2001). An outbreak of *E. coli* O157:H7 occurred in 2001 between July and September. In this case 10 children (9 years old and younger) and two adults were infected after visiting an animal collection open to the public. The source of the outbreak was not clearly defined and was detailed in a paper by Pritchard and colleagues (2000). Faecal samples from rabbits were collected from areas where cattle had grazed and also from non-animal areas (picnic and play areas) and the samples collected from these areas revealed the presence of a strain that was indistinguishable from the outbreak strain found in the patients (Pritchard *et al*., 2001). More recently, Solecki and colleagues reported that prevalence of *E. coli* O157:H7 positive sheep remained constant in both the summer and winter in the same geographical location in North East Scotland (Solecki *et al*., 2007). The authors also reported that there were no high shedding animals indentified in the winter period, an interesting observation and they suggest that feed type may potentially have implications regarding *E. coli* O157:H7 infections and ultimately infections in humans by reducing shedding in the source animals over this period.

Recent epidemiology

Escherichia coli O157:H7 is responsible for only a limited number of food poisoning illness outbreaks each year when compared with other bacteria that cause food-borne infections in the population (Tables 1 and 2); nevertheless, it is still an extremely important pathogen, due to the complications it may cause.

Tables 1 and 2 detail the most recent epidemiological data (1998–2006) on gastrointestinal infections caused by bacteria, available to the general public from the Health Protection Agency from the UK (HPA) and Health Protection Scotland (HPS) websites. It is worth noting that this is only for a selection of the potential bacterial pathogens. Data on the following bacteria are shown: *E. coli*

Table 1. Statistics for England and Wales from 2000 to 2006, showing the prevalence of gastrointestinal infection causing bacteria (HPA-1, 2008; HPA-2, 2008; HPA-3, 2008; HPA-4, 2008; HPA-5, 2008; HPA-6, 2008; HPA-7, 2008; HPA-8, 2009).

Year	*E. coli* O157:H7	*Campylobacter* spp.	*Clostridium perfringens*	*Salmonella* spp.	*Listeria* spp.	*Shigella* spp.
2000	896	57 674	144	14 796	101	1 095
2001	768	54 918	439	16 475	146	1 187
2002	595	47 848	385	14 753	138	1 052
2003	675	46 181	23	15 371	234	1 049
2004	699	44 294	486	13 552	211	1 269
2005	950	46 296	509	11 921	189	1 395
2006	1003	46 603	147	13 057	185	1 082
Total	7560	456 920	2913	140 714	1416	10 701

Table 2. Statistics for Scotland from 2000 to 2006, showing the prevalence of bacterial gastrointestinal infections (HPS-1, 2006; HPS-2, 2007; HPS-3, 2007; HPS-4, 2007; HPS-5, 2008; HPS-6, 2009; SSRL-2b, 2008).

Year	*E. coli* O157:H7	*Campylobacter* spp.	*Clostridium perfringens*	*Salmonella* spp.	*Listeria* spp.	*Shigella* spp.
2000	197	6 482	27	1 720	11	90
2001	235	5 435	46	1 571	15	91
2002	229	5 121	40	1 149	20	78
2003	148	4 445	35	1 254	14	79
2004	209	4 365	41	1 143	15	104
2005	172	4 558	21	1 127	31	118
2006	243	4 865	4	1 029	17	136
Total	1947	47 517	265	12 981	144	902

O157:H7, *Campylobacter* spp., *Clostridium perfringens, Salmonella* spp., *Shigella* spp. and *Listeria* spp.

As both tables demonstrate, *E. coli* O157:H7 is not the most prevalent food-borne pathogen in England, Wales or Scotland; the most prevalent is *Campylobacter* spp., which accounts for approximately 70% of all the laboratory-confirmed cases in the aforementioned countries. The rate of *E. coli* O157:H7 infection may be relatively small; nevertheless serious clinical complications such as kidney failure and HUS make it an important pathogen (Kawano *et al*., 2008). Complications resulting from infections with *Campylobacter* spp. are relatively rare with an incidence of approximately 0.05 per 1000 infections; however, most uncomplicated clinical cases result in acute cases of fever and diarrhoea (Allos, 2001).

In England and Wales, *E. coli* O157:H7 comprises approximately 1% of all the laboratory cases of food poisoning, whereas in Scotland, *E. coli* O157:H7 cases account for approximately 3% of all the laboratory cases of food poisoning. This is relatively small in relation to the size of the populations. Nevertheless, when the size of the population is taken into account and the rate of *E. coli* O157:H7 confirmed cases per 100 000 are examined, the rate of *E. coli* O157:H7 in Scotland is much more than England and Wales. This incidence between 2002 and 2008 is shown in Fig. 1. In mid-2005, the estimated population of England and Wales was 53.4 million (ONS, 2006) and Scotland had an estimated population of 5 094 800 (GRO, 2006). When these population sizes are included, the question arises, why does Scotland have a greater incidence of *E. coli* O157:H7 compared with England and Wales, when their borders are adjacent? Could there be a difference in the prevalence and strains of *E. coli* O157:H7?

Strains of *E. coli* O157:H7 can be differentiated into more than 80 different phage types (Preston *et al*., 2000; Smith *et al*., 2000). A number of studies have looked into the prevalence of *E. coli* O157:H7 and phage types in England, Scotland and Wales (Table 3). Phage types (PT) 2, 4, 8 and 21/28 appear to be the most common in these countries. These studies indicate that the distribution of *E. coli* O157:H7 phage types throughout the UK shows no marked geographical variations (Frost *et al*., 1989). Some of the same phage types, for example PT 2 and 8, can also be found as far as Spain (Mora *et al*., 2004).

The question remains, why does Scotland have a greater incidence of *E. coli* O157:H7 when compared with England and Wales? The countries are geographically close and the phage types are similar, yet the rate is greater north of the border. What other factors can be involved? To date relatively few studies have examined

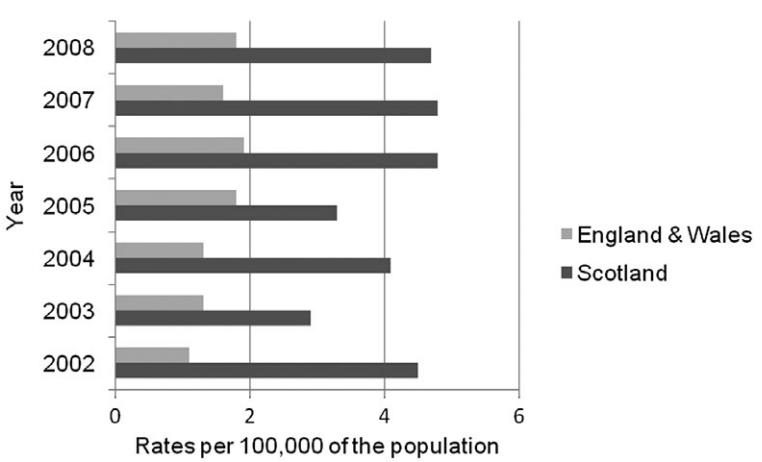

Fig. 1. Comparing the rates of *E. coli* O157:H7 cases per 100 000 of the population in England, Wales and Scotland between 2002 and 2008 (HPA-4, 2008; HPS-6, 2009).

Table 3. Various studies showing the prevalence of different *E. coli* O157:H7 phage types in England, Scotland, Wales and Spain.

Phage types	Country	Presence/outbreak	Date of study/incidence	Reference
2, 49, 8, 1, 4	England and Wales	Various outbreaks reported to the laboratory of enteric pathogens	1992–1994	Thomas *et al.* (1996)
2	Sunderland, England	14 cases, eating precooked meats	August 1995	Stevenson and Hanson (1996)
21/28	North Cumbria, England	88 confirmed cases, drinking pasteurized milk	March 1999	Goh *et al.* (2002)
2, 8, 21/28 (cattle), 4, 32 (sheep)	Great Britain	118 abattoirs which included 3939 cattle and 4171 sheep faeces samples	Jan 1999–Jan 2002	Paiba *et al.* (2002)
2, 4, 28	Scotland	7 outbreaks including largest milk-borne outbreak	April 1994–March 1995	Allison *et al.* (1998)
8	Scotland	37 people excreting organism, hospital	May 1997	O'Brien *et al.* (2001)
21/28	Scotland	Private water supplies, Northern Scottish cattle farms	Feb 2002–Feb 2004	Halliday *et al.* (2006)
2	Paisley, Scotland	Food-borne at supermarket	August 2007	Stirling *et al.* (2007)
4, 8, 14, 2, 49	UK	Various outbreaks	Since 1983	Frost *et al.* (1989)
2, 49, 1, 4	UK	Various outbreaks reported to the laboratory of enteric pathogens	1989–1991	Frost *et al.* (1993)
2, 8, 14, 21/28,54	Spain	67 humans, 82 bovines, 12 ovines and 10 beef products	1980–1999	Mora *et al.* (2004)

these phenomena with respect to private water supplies, animal movement and weather patterns.

Survival of *E. coli* O157:H7 in the environment

Escherichia coli O157:H7 can survive in a variety of different environmental settings: soil, water, raw sewage and even on a range of metal surfaces (Ogden *et al.*, 2002; Wilks *et al.*, 2005; Muniesa *et al.*, 2006; Nwachuku and Gerba, 2008). The survival of *E. coli* O157:H7 in water and soil is dependent on a variety of different factors such as soil composition (presence of manure/ organic matter), temperature, pH, competition with soil microorganisms and adverse environmental conditions (Jiang *et al.*, 2002). This bacterium can survive for long periods of time in water, especially at cold temperatures, with studies suggesting that *E. coli* O157:H7 can enter a viable but non-culturable state (Wang and Doyle, 1998). LeJeune and colleagues (2001) showed that cattle troughs may serve as a long-term reservoir for *E. coli* O157:H7 on farms, and as a potential source of infection to cattle. *Escherichia coli* O157:H7 was able to survive for approximately 245 days in the troughs and the surviving microorganisms continued to be infectious to 10-week-old calves over this time period (LeJeune *et al.*, 2001). Ogden and colleagues (2002) investigated the long-term survival of *E. coli* O157:H7 in a field, following an outbreak of illnesses connected with a scout camp; previously, the field had sheep grazing in it. The survival of the organism was observed for 15 weeks after which it still possessed virulence genes that suggested it had the possibility of causing human illness. Recently, a laboratory study has shown that *E. coli* O157:H7 survives and replicates in *Acanthamoeba polyphage* (Barker *et al.*, 1999). This protozoan inhabits a variety of soil and water environments; Barker and colleagues have suggested that this organism may therefore be an important reservoir for the transmission of *E. coli* O157:H7, potentially causing infection to humans and other animals (Barker *et al.*, 1999).

Seasonality of infection in cattle and humans

The prevalence of *E. coli* O157:H7 in cattle depends upon the season, with studies reporting greater faecal shedding of the organism in the warmer seasons (spring and summer) (Hancock *et al.*, 1997). Non-O157 *E. coli* infections are also increasingly prevalent in the summer months (Brooks *et al.*, 2005). This is directly mirrored by the seasonal trends of bacterial infections among hospitalized patients, with a significantly higher rate of Gram negative infections (including *E. coli*) occurring during the summer months (Fig. 2) (Perencevich *et al.*, 2008).

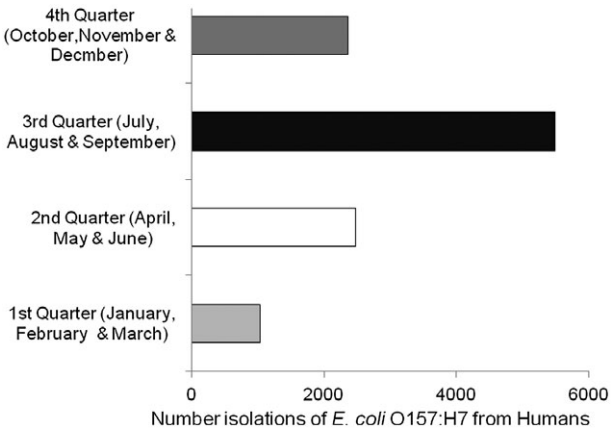

Fig. 2. The number of *E. coli* O157:H7 isolations from Humans reported to the Health Protection Agency (UK), on a quarterly basis from England and Wales in the period 1992 to 2006 (HPA-2, 2008).

The majority of the reported *E. coli* O157:H7 infections occurred in the third quarter of the year (July, August and September), which coincides with the warmest times of the year in the UK (HPA-2, 2008).

Studies on the presence of *E. coli* O157:H7 have been performed worldwide: in Argentina, data suggest that a relationship between the type of stx and the season exists, with greater prevalence of stx positive cows in spring (44%) and summer (56%) when compared with autumn (22%) and winter (11%) (Fernandez *et al*., 2009). In Canada, the faecal shedding of *E. coli* O157:H7 on a dairy farm peaked from June to September, with the collection of positive samples being 15 times more likely in this time period when compared with other months (Stanford *et al*., 2005). In another UK study, rectal samples collected immediately post slaughter from 400 cattle, each month for a one-year period, were studied (Chapman *et al*., 1997). The monthly prevalence was at its highest in spring and late summer with 100% of sheep and 99.6% of cattle samples being positive for vero cytotoxigenic *E. coli* (Chapman *et al*., 1997). Douglas and Kurien (1997) reported both HUS and *E. coli* O157:H7 infections peaked in patients less than 15 years of age in July/August. Interestingly, Alam and Zurek (2006) found no statistically significant difference in the prevalence of *E. coli* O157:H7 in beef cattle faeces between the seasons, and although Ogden and colleagues (2004) found similarly contradictory evidence, that the prevalence of *E. coli* O157:H7 in Scottish beef cattle at abattoir was greater during the cooler months than the warmer months (Ogden *et al*., 2004), their data were significantly influenced by a group of high shedders within an otherwise apparently uniform sample of cattle shedding the organism.

Reasons for seasonality of infection with *E. coli* O157:H7

Seasonal shedding of *E. coli* O157:H7 is well documented, there is, however, conflicting evidence to support different ideas, suggesting there is no single reason but rather a collection of different factors. Factors that may potentially affect the seasonality of faecal shedding and human infection include increased housefly populations in the summer (Alam and Zurek, 2004), environmental factors (such as rainfall) (Gagliardi and Karns, 2000) and livestock feed and waste handling practices including microbial interactions in soil (Rasmussen and Casey, 2001). However, increased temperatures due to increased sunshine in the summer months, which may promote the growth and aid survival in of *E. coli* O157:H7, has been suggested as a likely factor (Perencevich *et al*., 2008). However, Edrington and colleagues (2006) suggested that faecal shedding of *E. coli* O157:H7 and day length have a higher correlation than shedding and ambient temperature. Further research examined the effect of artificial lighting on *E. coli* O157:H7 prevalence in a commercial feedlot, where all conditions such as location were kept the same and the only variable was the lighting (Edrington *et al*., 2006). The research concluded that the faecal prevalence of *E. coli* O157:H7 remained constant in the artificially lit pens, while prevalence was lower in the control pens that received no artificial lighting. After the removal of artificial lights from the pens, the prevalence of the organism returned to levels similar to that of the control pens. Edrington and colleagues (2006) suggested an underlying mechanism relating to day length and physiological response (Edrington *et al*., 2006). Further studies by Edrington and colleagues (2008a,b) proposed that hormones such as melatonin respond to day length and may be the underlying mechanism for seasonality in cattle. Results from their experiments showed that it was possible that gastrointestinal melatonin was involved in the gastrointestinal population and faecal shedding of the organism. The authors therefore hypothesized that the seasonal variation is a result of physiological responses within the host animals to changing day length (Edrington *et al*., 2008a,b). Melatonin, a pineal hormone present in animals such as cattle, has previously been shown to mediate seasonal adjustments in immune function with the gastrointestinal tract being a major source, exceeding that of the pineal gland by almost 400 times the amount (Nelson and Drazen, 1999; Bubenik, 2001; 2008). Melatonin, however, shows an inverse seasonal pattern to that of *E. coli* O157:H7. Melatonin serum concentration is low in the summer and generally increases through autumn, reaching a peak in the winter (Edrington *et al*., 2008a,b).

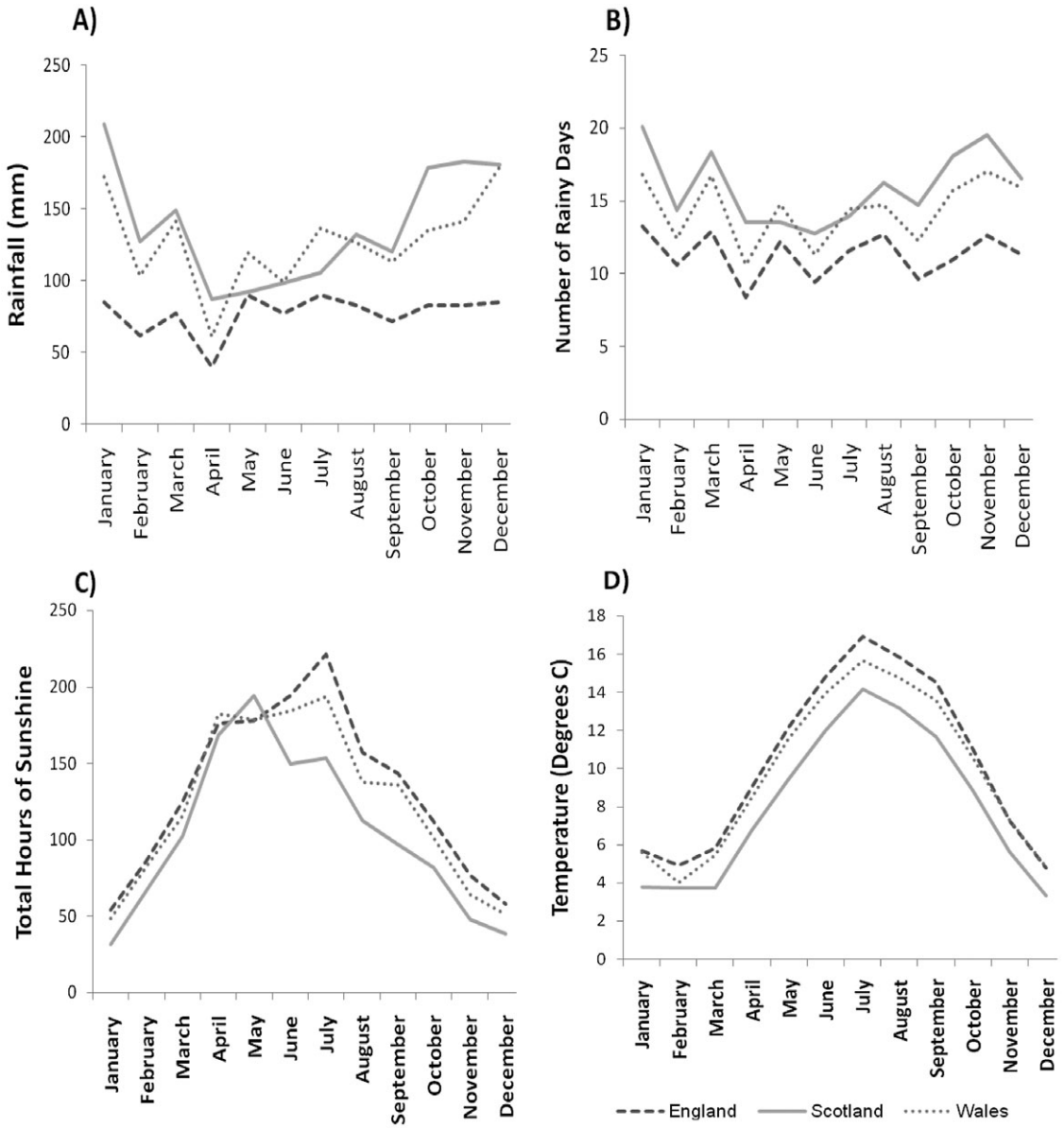

Fig. 3. Average data for England, Scotland and Wales from 2006 to 2008, showing (A) average rainfall (mm), (B) number of rainy days (≥1.0 mm), (C) average total hours of sunshine (h) and (D) mean monthly temperature (°C) (Met Office-1, 2009; Met Office-2, 2009; Met Office-3, 2009; Met Office-4, 2009; Met Office-5, 2009; Met Office-6, 2009; Met Office-7, 2009; Met Office-8, 2009; Met Office-9, 2009; Met Office-10, 2009; Met Office-11, 2009; Met Office-12, 2009).

Other possible factors could be the differences in handling, cooking and eating food. In the summer there are generally more outdoor activities taking place such as barbeques and picnics and there is published evidence for the involvement of zoo/farm visits, school trips, and summer scout camps in infections/outbreaks (Heuvelink *et al*., 2002; Ogden *et al*., 2002). Furthermore, since there is a greater prevalence and shedding of *E. coli* O157:H7 in the summer, it is tempting to speculate that a greater number of these bacteria may also enter slaughterhouses.

Are weather differences a factor?

The mean weather statistics for Scotland from 2006–2008 shows an average annual rainfall of 1657 mm (Fig. 3A) with 192 rainy days (Fig. 3B), with an annual average sunshine of 1246 h (Fig. 3C) and the mean monthly temperature being 8°C (Fig. 3D). A comparison of these averages to the mean data of England shows clear differences. The mean weather statistics for England from the same period shows an average annual rainfall of 921 mm (Fig. 3A), with 136 rainy days (Fig. 3B) and an annual

average sunshine of 1581 h (Fig. 3C) and the mean monthly temperature being 10°C (Fig. 3D). Looking at the data for the last 3 years, it can be suggested that Scotland has a greater amount of rainfall, less sunshine and a lower temperature, on average, than England and Wales.

Ogden and colleagues (2002) investigated the long-term survival of *E. coli* O157:H7 in a field following an outbreak of illness connected with a scout camp. Prior to the camp, approximately 300 sheep had been grazing in the field; subsequent testing of 28 sheep revealed that 17 were positive for *E. coli* O157:H7. The widespread contamination of the area was due to the heavy rainfall: *E. coli* O157:H7 contaminated areas included the soil, sheep faeces, standing water and the scout climbing frames at the field (Ogden *et al*., 2002). Since this outbreak was a result of the heavy rainfall and flooding in the localized area, could the higher amounts of rainfall in Scotland be a factor in the elevated levels of *E. coli* O157:H7 compared with England and Wales?

As Fig. 3A, B and D shows, Scotland has cooler and wetter summers than England and Wales. There is greater faecal shedding of *E. coli* O157:H7 in the warmer months (spring and summer) (Hancock *et al*., 1997), which is mirrored by the increased infection rates in the summer months (Perencevich *et al*., 2008).

Cattle population and number of private water supplies

As mentioned before, cattle are asymptomatic carriers of the organism, with a greater prevalence of stx-positive cows in spring and summer. They therefore have the potential to excrete the organism in their faeces or in abattoirs during slaughtering. In 2006, the estimated cattle population according to the Department for Environment, Food and Rural Affairs (DEFRA) in England and Wales was 5 763 910, and Scotland had an estimated cattle population of 1 417 910. England and Wales obviously had a greater population. However, when the rate of the cattle population is examined per 100 000 of the human population, Scotland has a higher cattle-to-person ratio than England and Wales; Scotland had 27 830 cattle per 100 000 of the population whereas England and Wales had only 10 793 cattle per 100 000 of the population (DEFRA, 2010).

The same is true for the private water supplies available in the UK. Private water supplies have been regarded as a source for *E. coli* O157:H7 infections (Said *et al*., 2003). Private water supplies are located in rural and remote parts of the country; however, some can exist in urban settings being used in industrial processes.

The drinking water inspectorate describes a private water supply as one that is not provided by a water company, and includes wells, boreholes, springs, streams, rivers, lakes or ponds (DWI-3, 2009). There are approximately 50 000 private water supplies in England and Wales and approximately 20 000 private water supplies in Scotland (DWI-1, 2001). According to the Drinking Water Inspectorate (DWI), England and Wales has approximately 93 private water supplies available per 100 000 of the population (DWI-2, 2008) whereas Scotland (DWI-1, 2001) has 393 private water supplies available per 100 000 of the population. Therefore, the increased usage of private water supplies, cattle population and difference in weather conditions, especially the worsened weather conditions during the summer, may explain why Scotland has a greater incidence of *E. coli* O157:H7 when compared with England and Wales.

Conclusion

Escherichia coli O157:H7 is a bacterial pathogen that has been shown to tolerate diverse environmental conditions and while it is usually found in the gastrointestinal tract of cattle it can also survive in a variety of different settings such as soil and water. The bacterium can infect people by getting into the food chain via contaminated meats, vegetables and water supplies. It can also infect people who have increased contact with grounds where animals such as cattle and sheep have previously grazed. Individuals who become infected with *E. coli* O157:H7 can develop acute gastroenteritis, severe bloody diarrhoea and also life-threatening complications such as HUS. As mentioned earlier, *E. coli* O157:H7 is responsible for approximately 1% of all laboratory-confirmed cases of food poisoning in England and Wales, which is a relatively small amount in relation to the size of the population. However, in Scotland, *E. coli* O157:H7 cases account for 3% of all the laboratory-confirmed′ cases of food poisoning. When the size of the population is taken into account and the rate of *E. coli* O157:H7 confirmed cases per 100 000 population is examined the rate for Scotland is higher than that observed in England and Wales.

The *E. coli* O157:H7 phage types appear to be common throughout the UK, with the distribution showing no marked geographical variations, implying that other factors are likely to be involved in the discrepancy in incidence rates. This review has also shown that the prevalence of *E. coli* O157:H7 is largely, seasonally dependent, with a greater faecal shedding of this organism being reported in the warmer seasons. This is directly mirrored by increased bacterial infections also occurring during these seasons. A relationship between the type of stx toxin and season also exists. Weather differences may be a contributing factor to *E. coli* O157:H7 geographical variations. Scotland on average has greater rainfall, less sunshine and lower temperatures, than England and Wales. It has been reported that greater rainfall can con-

taminate the surrounding areas and infect individuals subsequently using these areas. Therefore, weather differences, especially in the summer, may be a contributing factor to the difference in *E. coli* infection rates. Private water supplies also pose a risk for infections, with Scotland having a greater amount of private water supplies available per 100 000.

It is therefore suggested that the greater cattle population, the weather conditions and the increased number of private water supplies may all contribute to why there is a difference in *E. coli* O157:H7 infection rates between Scotland, England and Wales. Further studies are required to strengthen this hypothesis.

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