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Rapid communications

NATIONWIDE OUTBREAK OF STEC O157 INFECTION IN THE NETHERLANDS, DECEMBER 2008-JANUARY 2009: CONTINUOUS RISK OF CONSUMING RAW BEEF PRODUCTS

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The Netherlands experienced a nationwide outbreak of Shiga toxin-producing *Escherichia coli* (STEC) O157 with onset of symptoms from the end of December 2008 until the end of January 2009. A total of 20 laboratory-confirmed cases were linked to the outbreak strain, serotype O157: H-, stx1, stx2, eae and e-hly positive. The investigation into the source of this outbreak is still ongoing, but evidence so far suggests that infection occurred as a result of consuming contaminated raw meat (steak tartare).

Introduction

Shiga toxin-producing *Escherichia coli* (STEC) can cause bloody diarrhoea which progresses to a life-threatening condition known as the haemolytic uraemic syndrome (HUS) in 2-15% of the cases, particularly children [1,2]. STEC O157 is the serogroup most commonly identified in outbreaks.

STEC is found in the guts of many animals, but ruminants and cattle in particular are believed to be the main reservoir of human pathogenic STEC. Although infection can occur through direct contact with animals, most human infections are probably foodborne; water or food products (undercooked beef, raw milk, vegetables such as lettuce and alfalfa) contaminated with manure have often been linked to common source outbreaks. Person-to-person transmission can also occur [3,4].

An enhanced laboratory-based surveillance for STEC O157 was implemented in the Netherlands in 1999 and STEC non-O157 serogroups were added to this surveillance in 2007 [5]. Since 1999, STEC has been notifiable by law. All STEC-positive isolates identified in laboratories across the country are sent to the National Institute of Public Health and the Environment (RIVM). Since the start of the surveillance the number of STEC O157 infections reported annually ranged from 35 to 57 between 1999 and 2006, increased to 83 cases in 2007 as a result of a national outbreak [6], and dropped to 46 sporadic cases in 2008.

In week 4 of 2009, an increase to nearly half the predicted annual number of STEC O157 cases (n=20) was noted thanks

to the intensive surveillance system. This prompted a further epidemiological and microbiological investigation, the results of which are presented here.

Methods

An outbreak investigation was initiated on 29 January 2009 in response to laboratory confirmation of a nationwide outbreak of STEC O157. An outbreak case was defined as a person diagnosed with a laboratory-confirmed STEC O157 infection since 10 December 2008 and a pulsed-field gel electrophoresis (PFGE) profile belonging to the outbreak cluster. Municipal Health Authorities in the Netherlands routinely follow up laboratory-confirmed STEC cases using a standardised questionnaire to collect information on clinical symptoms and exposure to possible risk factors in the week preceding onset of illness. Due to the dispersed distribution of cases within the Netherlands suspicion was raised that the cause could be a common food source or supplier; Municipal Health Authorities were requested to pay particular attention to the completeness of responses to questions pertaining to food history and location of purchase in their follow up of laboratory-confirmed cases.

All STEC positive isolates sent in to the RIVM are tested for genes encoding Shiga toxin type 1 and type 2 (*stx*₁ and *stx*₂), the *E. coli* attaching-and-effacing gene (*eae*) and the enterohaemolysin encoding EHEC-*hly* gene (*e-hly*). DNA fingerprints are generated by PFGE, using *Xba*I as the restriction enzyme. The fingerprints are processed with BioNumerics® (Applied Maths, Kortrijk, Belgium; Dendrogram type=UPGMA, Similarity coefficient=Dice) [4].

Statistical analysis

Analysis of food exposures was conducted using a case-case study design to compare laboratory-confirmed STEC O157 outbreak cases with sporadic cases of STEC O157 reported in the enhanced surveillance in 2008. Food items that were reported to have been definitely or possibly consumed were compared with items that were reported to have not been consumed. An attack rate and odds ratio for each food item was calculated using STATA 10. Individuals

who did not provide information on a food item were excluded from analysis of this particular food. Three possible secondary cases, defined as members of the same family as a case and with onset of symptoms later than that of the first family member, were also excluded from the analysis.

Food tracing

A trace back of suspected food items was initiated. The Food and Consumer Products Safety Authority (VWA) collected samples of any available left-over meat products from patients' homes for testing for STEC O157. The VWA also investigated the supermarkets and producers of various meat products mentioned by the cases.

International cooperation

The Netherlands is member of the European food and waterborne diseases and zoonoses surveillance network (formerly ENTERNET) administered by the European Centre for Disease Prevention and Control, which covers, amongst others, STEC infections. Using this network, in week 6 a request was made to all member countries to provide details of any occurrences of STEC O157 with a similar PFGE pattern.

Results

Between 27 December 2008 and 22 January 2009, 20 cases of STEC O157 (including three secondary cases) were attributed to the outbreak strain in the Netherlands (Figure 1). One additional STEC O157 case, with symptom onset on 13 December 2008, was possibly associated with the outbreak strain in accordance with the PFGE case definition and three isolates are pending PFGE typing to determine whether they are related to the outbreak.

Cases were spread throughout the Netherlands and were aged between 6 and 76 years of age (median age 41), with an equal number of males and females.

The outbreak strain was characterised as serotype O157:H-, and stx1, stx2, eae and e-hly positive and, all isolates, with the exception of one, were sorbitol-nonfermenting (Figure 2). This exact PFGE pattern has been seen on only two occasions in the Netherlands, both in 2005.

Sixteen of the 20 outbreak-related cases (80%) completed the questionnaire, three of which were secondary cases. Seven cases were hospitalised and none developed HUS. The questionnaire was

also returned by 36 non-outbreak cases of STEC O157 with onset of symptoms in 2008. These cases represented 78% of the total

FIGURE 2

Pulsed-field gel electrophoresis (PFGE) pattern of the outbreak strain (middle three lanes) in the national outbreak of Shiga toxin-producing *Escherichia coli* (STEC) O157 in the Netherlands, December 2008 - January 2009

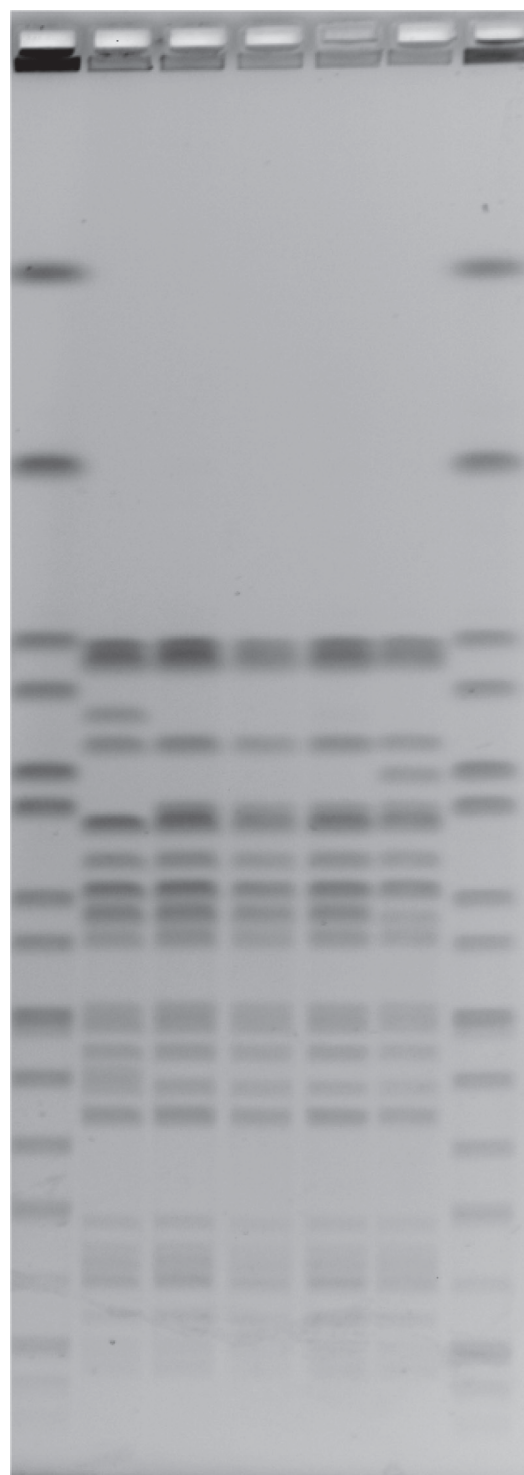
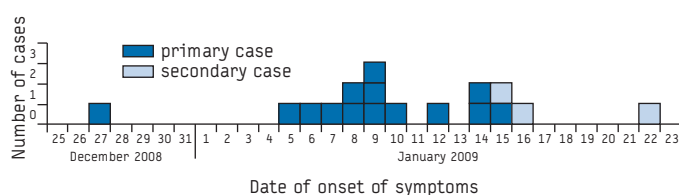


FIGURE 1

Distribution of confirmed cases of the outbreak strain of Shiga toxin-producing *Escherichia coli* (STEC) O157 in the Netherlands, December 2008 - January 2009, by date of onset of symptoms (n=17)*



* Date of onset was unknown for three of the twenty outbreak-related cases

number of STEC O157 cases reported in 2008, ranged in age from 1 to 65 years and 67% were women. Two food items were frequently reported to have been consumed by outbreak cases: minced meat and steak tartare. There appears to be a link between consumption of steak tartare and STEC O157 infection: 83% of the 13 primary outbreak cases who provided the relevant information reported eating steak tartare in the week before illness compared to 18% of the sporadic 2008 cases (OR 16.3; $p < 0.001$, Table).

Many of the cases purchased food at more than one supermarket or store, and the precise location where individual items were purchased was not recorded. The supermarket or butcher where the implicated steak tartare was bought is therefore unknown.

Results of food tracing

All left over food samples collected by the VWA tested negative for STEC O157. However, the food trace-back investigation is still ongoing and it is possible that the STEC cases pending PFGE results may be linked to the outbreak strain. Even if the contaminated batch of meat has been long-since consumed it is still worthwhile to further investigate the production flow in supermarkets, producers of steak tartare and possibly also slaughterhouses to try and gain information to support the epidemiological link.

International response

Nine countries responded to the information request (Belgium, Germany, Finland, Ireland, Norway, England and Wales, Scotland, United States and Denmark), none of which reported current STEC O157 cases with PFGE profiles related to the outbreak strain. Finland reported two strains isolated in 1998 and 2004 with identical PFGE patterns, both patients had reported travel to Turkey.

Conclusions

This is not the first time that a nationwide outbreak of STEC O157 has occurred in the Netherlands. In 2005, an outbreak was linked to steak tartare [3] and in 2007 an outbreak was associated with lettuce [4]. Based on the case-case study it seems probable that the outbreak reported here was also caused by steak tartare; steak tartare consumption was strongly and significantly associated with being an outbreak case and could explain the majority of the cases. The age distribution of cases seems also consistent with these findings; 28% of the non-outbreak cases of STEC O157 infection in 2008 were five years old or younger but the youngest outbreak-related patient was six years old which is not surprising as very young children are less likely to eat raw meat products.

TABLE

Risk factors for outbreak of Shiga toxin-producing *Escherichia coli* (STEC) O157 in the Netherlands, December 2008 – January 2009

	Outbreak case (n=13)	Non-outbreak case (n=36)	Univariate odds ratio	
Food item	n (%)	n (%)	OR (95%CI)	p-value
Minced meat	10 (77)	21 (60)	2.2 (0.4-14.5)	0.23
Steak tartare	10 (83)	6 (18)	16.3 (3.2-238.6)	<0.001

Note: Percentages were calculated taking as denominator the number of persons who provided the relevant answer

The incubation period of STEC O157 is generally considered to be 1-10 days. Thirteen of the 14 primary cases with known date of symptom onset became ill within 11 days of each other. Raw meat can become contaminated during slaughter, and by cutting and mixing the meat a point source contamination can result in the contamination of a large batch of meat. Hygienic slaughter processes are imperative but contamination of carcasses cannot be completely avoided. This outbreak is another sign that despite control measures and legislation, raw meat products continue to pose a risk for the health of the general population. Raising consumer awareness in relation to consumption of raw meat is still needed [1,7,8].

Tracing of meat products continues to be a difficult task because insufficient detail is collected in the routine questionnaires about the precise type of meat, such as whether it is beef or veal, pre-packed or fresh from a butcher. Although questionnaire data is very useful, in our investigation several cases mentioned shopping at more than one supermarket chain without distinguishing which items were purchased where. This made it difficult to trace back the place of production and purchase of the implicated steak tartare. Obtaining supermarket receipts from cases could assist with the trace back, particularly in investigations in which one supermarket chain is frequented or when it is unclear what products were purchased where. This could improve the efficiency of the food trace back, but it is also time-consuming when more than one supermarket is involved and there is no protocol in place. It would also be useful to collect detailed information about steak tartare in the routine questionnaire. It is also apparent that we still do not have a good insight into the production chain of steak tartare, despite two large outbreaks in recent years.

Even though the current outbreak was confined to the Netherlands, international trade in meat and vegetable products makes it important to raise the alert internationally and rapidly and accurately trace suspected food items. However, effective prevention of future outbreaks caused by consumption of steak tartare may be very difficult.

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Rapid communications

A SWIMMING POOL-RELATED OUTBREAK OF PHARYNGOCONJUNCTIVAL FEVER IN CHILDREN DUE TO ADENOVIRUS TYPE 4, GIPUZKOA, SPAIN, 2008

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An outbreak of pharyngoconjunctival fever affecting 59 children was detected in a municipality of northern Spain in July 2008. The outbreak was related to insufficient doses of water disinfectant in the municipal swimming pool. Adenovirus was detected in the pharyngeal swabs of five of six affected children and the four strains that were sequenced were all Adenovirus type 4.

Introduction

Pharyngoconjunctival fever can be caused by both picornaviruses and adenoviruses. The latter are divided into six species (A-F) with 51 known serotypes to date [1]. Humans are the reservoir for this virus and although frequently asymptomatic, adenovirus infections can affect the upper and lower respiratory tract and the eye and can also cause gastroenteritis and cystitis. In addition, adenoviruses are excreted in the respiratory and intestinal mucosa, in the latter sometimes for prolonged periods of time. The incubation time varies from two to 14 days, and transmission occurs through respiratory secretions, person-to-person contact, aerosolised viruses and fomites, as well as the faecal-oral route.

Although the first recorded outbreaks of pharyngoconjunctival fever associated with adenovirus transmission in a swimming pool were reported more than 50 years ago [2], this type of outbreak has been reported only rarely in the past few decades [3-5]. On 3 July 2008, the Epidemiological Surveillance Service of Gipuzkoa was notified of an unusually high number of children with fever, pharyngitis and/or conjunctivitis, who had consulted the paediatrician at the health centre of a municipality in the Goierri region. The present study describes the epidemiological, environmental and virological investigations that were performed to study this outbreak, and the control measures established.

Methods

Cases were defined as all individuals under the age of 15 years consulting the health centre of the affected town between 15 June and 11 August with conjunctivitis and/or pharyngitis with enlarged cervical lymph nodes. According to the census from 2006, the town had 9,141 inhabitants of whom 1,347 were under 15 years-old. The possible occurrence of cases of pharyngoconjunctival fever in the neighbouring towns and a referral hospital was monitored. Affected patients were interviewed in order to record

the following variables: place of residence, age, sex, date of symptom onset, symptoms, presence of complications, swimming pool use and other potential exposures. For each clinical picture, (pharyngoconjunctivitis, pharyngitis without conjunctivitis, and conjunctivitis without pharyngitis), two cases were studied in order to identify viral aetiology, using pharyngeal swabs with viral transport medium (ViralPack, Biomedics, Spain). For adenovirus detection, a real-time polymerase chain reaction (PCR) method was used that amplified a fragment of the hexon gene [6], and the amplicons obtained were sequenced to characterise the adenovirus type. The pH value and the concentration of disinfectant in the water of the four basins of the public swimming pool were determined (Test Cloro and pH 1.11174.0001, Merck, Germany), the automatic pH regulation and disinfectant dosing pump system was inspected and the incident log book was reviewed. Disinfection was performed through bromination in the small inner children's pool and through chlorination in the remaining pools.

Results

Between 16 June and 11 August, 59 children were diagnosed with pharyngoconjunctival fever and met the case definition. Forty-three of the children (73%) had recently used the municipal swimming pool, which was considered the source of infection (primary cases). Fifteen (25%) of the children had been in close contact with a primary case (secondary cases). The very first case that occurred had not visited the swimming pool and was therefore considered sporadic. The epidemic curve confirmed an outbreak with an epidemic pattern characterised by an accumulation of primary cases, consistent with the hypothesis of a persistent common source, and more isolated secondary cases, resulting from person-to-person transmission mainly in a family environment (Figure).

All affected infants and children lived in the area where the swimming pool is situated. They were 34 (58%) boys and 25 (42%) girls. Ten percent of affected individuals were under the age of one year, 29% were between one and four years-old, 59% between five and 13 years-old and one case was 14 years-old (2%). It must be noted that the case definition only included children under the age of 15 years; an estimation of the secondary attack rate among older children and adults in the families or contacts was

therefore not possible. Forty-three percent had fever, pharyngitis and conjunctivitis, 29% had fever and pharyngitis and 28% had conjunctivitis with or without fever. All affected children were diagnosed by pediatricians and three required a consultation with an ophthalmologist due to keratoconjunctivitis. There were no hospitalisations and all cases received symptomatic treatment. Adenovirus was detected in five of the six pharyngeal swabs collected (two children with pharyngitis, one with conjunctivitis and two with pharyngitis and conjunctivitis), and the virus was identified as Adenovirus type 4 in the four amplicons that were sequenced.

After the swimming pool opened (on 7 June) for the summer season, there were numerous electrical system failures causing intermittent failure of the water circulation and bromine dosing pumps. The disinfectant concentrations registered on 3 July (the day the outbreak was reported) were insufficient in the small children's pool (0.45 mg/l of total bromine) and were adequate in the remaining pools. On the same day, swimming in the small pool was forbidden. Once the disinfection system was repaired and normal disinfection concentrations (bromine and pH) were confirmed, swimming was again permitted.

Discussion

Swimming pool-related outbreaks of viral infection are highly uncommon. The most frequently involved viruses are adenovirus, norovirus, hepatitis A virus and echovirus, in this order [7]. The outbreak reported in the present study was due to an adenovirus type 4 most probably transmitted through the water of the inner children's pool. The abrupt onset of this outbreak generated a certain alarm in the health sector and in the population, but after the system breakdown was repaired and control measures were established, the incidence of infection decreased sharply. Due to logistic problems, no water samples were taken from the swimming pool for virological analysis, which would have allowed the aetiology of the outbreak to be unequivocally confirmed.

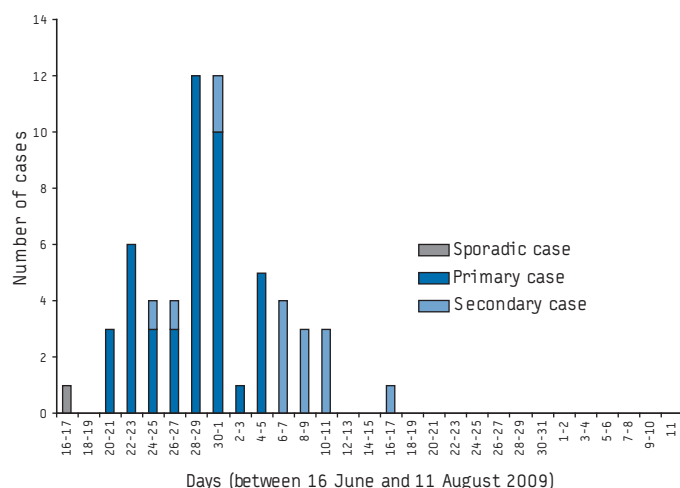
Adenoviruses are non-enveloped viruses, unusually resistant to physical and chemical agents, which gives them prolonged survival capacity [1]. Recently, these viruses have been observed to be prevalent in rivers, coastal water, swimming pools and water supplies worldwide [8,9]. Adenoviruses have also been detected in swimming pool water in the context of outbreaks of pharyngoconjunctival fever [3,4,10]. Transmission of this virus can occur both through intake of swimming pool water or through direct contact between the water and the conjunctival mucosa or upper respiratory tract [9]. The clinical presentation of cases in this outbreak was consistent with pharyngoconjunctival fever, as reported in other swimming pool-related outbreaks of non-enteric adenovirus infection [2-5,10-12]. Adenovirus type 3 has been most frequently found in these outbreaks [2,3,5,11,12], and to a lesser extent, type 7 [13,14] and type 4 [10]. Adenovirus type 4, the only member of human adenovirus species E, is one of the major causes of adenoviral conjunctivitis and the type considered to be responsible for the outbreak reported here. Clinical manifestation of this virus type varies, ranging from pharyngoconjunctival fever to keratoconjunctivitis, unlike conjunctivitis caused by serotypes 3 and 7, which tend to be milder [15].

It is obvious that the electrical problems at the swimming pool must have affected the disinfectant regulation system severely. However, the record books do not report any disinfection problem. We consider it disputable whether the measurements between the start of the electrical problems and the beginning of our *in situ* study were performed correctly. We strongly believe that strict adherence to the existing regulation would have avoided the outbreak.

The reports published to date would seem to indicate that swimming pool-related outbreaks of adenovirus infection have become exceptional in the last few decades. However, the outbreak reported in the present study reveals that these infections continue to pose a risk to swimming pool users when recommended control guidelines are not strictly observed. Adequate standards of hygiene and disinfection must be maintained in these installations to prevent transmission of adenoviruses and other microorganisms, and early investigations could decrease the number of cases

FIGURE

Cases of pharyngoconjunctival fever, by date of disease onset, Gipuzkoa, Spain, June-August* 2009 (n=59)



*Active surveillance was ongoing until 11 August

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Rapid communications

NOSOCOMIAL MEASLES CLUSTER IN DENMARK FOLLOWING AN IMPORTED CASE, DECEMBER 2008-JANUARY 2009

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A cluster of six confirmed cases with identical measles virus genotype was reported in Denmark between December 2008 and January 2009. Transmission occurred among unvaccinated children aged 15-23 months admitted to the same hospital as a 36-month-old unvaccinated girl diagnosed with measles following travel. The findings highlight the importance of vaccination before travelling and adherence to the routine vaccination schedule.

Introduction

In Europe, recommendations regarding vaccination against measles vary, with the first dose of measles, mumps and rubella (MMR) vaccine recommended at around 13 months of age (range 9-23 months) [1]. According to the Danish routine immunisation schedule, MMR is recommended at the age of 15 months (first dose) and 4 years (second dose) [2]. A study aimed at estimating the age of children vaccinated between 2001 and 2005 showed a frequent delay in administering the first dose of the MMR vaccine in Denmark (Table) [3]. Since 1999, coverage with the first dose of MMR vaccine has been 89-90%, and coverage with the second dose 86-88% [3].

With 12 cases of measles reported in 2008 the national incidence rate in Denmark has reached 0.2 per 100,000 inhabitants [4].

Between 1 December 2008 and 30 January 2009, a cluster of six confirmed cases of measles occurred among children aged 15 to 36 months admitted to the paediatric department of Hvidovre Hospital in Denmark. The index case had recently returned from Africa. None of the six children had been vaccinated with the first dose of MMR, although it is recommended at the age of 15 months. They were admitted to hospital for reasons other than measles.

The paediatric department in Hvidovre hospital is an enclosed area consisting of four sections with 50 beds, including the emergency room, and a playing area.

Cases were confirmed by a positive measles IgM antibody test and considered nosocomial if the patient had been exposed to measles during hospital stay. The case definition and classification used was based on the European Commission Decision of 28 April 2008 [5].

Outbreak description

The first patient was a three-year-old girl who had returned from a four-week holiday in East Africa. She was admitted to

Hvidovre Hospital on 1 December 2008, two days after returning to Denmark, with symptoms of gastroenteritis. On admission rash was noticed. The following day the maculopapular rash spread from the face downwards on the trunk, and measles was suspected and confirmed. On day four the patient recovered and was discharged.

The second case was a 15-month-old boy admitted to the same hospital on 18 December. He had an earlier history of asthmatic bronchitis and was admitted to hospital due to fever despite of antibiotic treatment for pneumonia. He stayed in the paediatric emergency room, in the paediatric ward and in the playing area. He developed a rash on 21 December, and measles was suspected on 22 December. He recovered gradually and was discharged after eight days of hospitalisation.

The next four cases were 16 to 26 months of age, and were admitted to the hospital for various illnesses around 20 December, before measles was suspected in the 15-month-old boy described above. These four patients were readmitted on 7 and 8 January 2009. They developed rash and measles was confirmed. After a few days they recovered and were discharged. The exact sequence of events is shown in the Figure below.

All six cases were confirmed by a positive test for measles IgM antibody. The epidemiological link between the index case and the second case was not verified, however, viral isolates obtained

TABLE

Percentage of vaccinated with the first dose of measles mumps rubella (MMR) vaccine, by age, Denmark, 2001-2005

Year	< 18 months	18-23 months	24-35 months	> 36 months
2001	76	16	5	3
2002	74	18	6	2
2003	74	18	6	2
2004	75	19	5	1
2005	77	18	5	-

Source: Valentiner-Branth P, Glismann S, Christiansen AH, Andersen PH, Simonsen JB. MMR-vaccination: Coverage by end 2007. EPI-NEWS 2008;36 [3]

populations. The outbreak-cases were all above the age in which it is recommended to receive MMR vaccination in Denmark.

None of the cases reported in this outbreak had been vaccinated against measles. For three children the reasons for non-vaccination were chronic illnesses that do not contraindicate MMR vaccination. One child had not received the vaccine due to egg allergy which is no longer considered a contraindication to MMR vaccination. Two children were not vaccinated without any apparent reason.

As shown in the Table, in Denmark the proportion of children who receive vaccination with delay is considerable thus expanding the susceptible window between declining maternal antibodies and protection from the first dose of MMR. These missed or postponed vaccinations without obvious medical reason constitute an area where efforts to improve compliance with guidelines are recommended. More accurate knowledge of the parents about immunisation safety, effectiveness and timing may increase timely vaccinations. Improvement of the vaccination coverage is required to reach the 95% needed to eliminate measles virus transmission in the population [9].

With 12 cases reported in 2008, most physicians rarely diagnose measles. The outbreak described in this paper illustrates the difficulties in management of measles in a health care system where the disease is uncommon, and the suspicion is not necessarily aroused on admission, with the risk of transmission of measles virus to vulnerable patients. Also in 2008 an outbreak involving five cases of measles occurred in Denmark following one imported case [10]. Health-care providers should continuously be aware of symptoms of measles and include measles in differential diagnoses for febrile rash illnesses particularly in patients with recent travel to measles-endemic areas.

To protect infants from contracting measles when travelling and to prevent from transmission in Denmark, the MMR vaccine is recommended to infants from nine months of age before travelling to measles-endemic areas, and to all non-immune individuals of all age groups before travel and in general [11]. Young adults may have low levels of measles immunity as they were born too early to have been part of the two-dose MMR vaccination program (introduced in Denmark in 1987) and have grown up in a period when exposure to wild measles virus was declining.

An epidemiological survey made by the Surveillance Community Network for Vaccine Preventable Infectious Diseases (EUVAC.NET) including national surveillance data from 32 European countries reported 12,132 cases of measles during the period 2006-2007 [12]. Most cases were among unvaccinated or incompletely vaccinated children. This epidemiological report shows that major parts of Europe still have problems in maintaining herd immunity with a vaccination coverage of 95%, especially in minority communities contributing with a larger risk of cluster outbreaks [13,14]. Several outbreaks have been reported in 2008 [4]. The World Health Organization (WHO) measles elimination plan for Europe by 2010 [9] demands continued focus and enhanced international vigilance to achieve success.

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Rapid communications

SALMONELLOSIS CASES CAUSED BY A RARE *SALMONELLA* ENTERITIDIS PT6C ASSOCIATED WITH TRAVEL TO BULGARIA, JUNE-JULY 2008

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In June 2008 an outbreak of gastroenteritis was registered in Sunny Beach resort situated on the Black Sea coast in Bulgaria, affecting 14 employees of a hotel, five of whom tested positive for *Salmonella* Enteritidis. During June-July 2008 four sporadic *S. Enteritidis* cases were also reported and two of them were foreign tourists. In the same period *S. Enteritidis* cases connected with travel to Bulgaria were reported to the European Centre for Disease Prevention and Control (ECDC) from Finland, United Kingdom, Sweden, Germany and Norway. We describe a study performed to find out relatedness between Bulgarian and Finnish *S. Enteritidis* isolates using phage typing (PT) and pulse-field gel electrophoresis (PFGE). Fifteen *S. Enteritidis* isolates from Bulgaria and 195 from Finland (including 28 from travellers to Bulgaria) were phage typed. Within Bulgarian isolates four different PTs were found and PT6c with eight strains was predominant. Nineteen out of 28 strains isolated from the Finns visiting Bulgaria belonged also to PT6c. PFGE typing (with one enzyme) of all *S. Enteritidis* PT6c strains (8 Bulgarian and 19 Finnish isolates) showed indistinguishable PFGE profile. The typing results thus demonstrated a link between Bulgarian and Finnish *S. Enteritidis* isolates. We conclude that *S. Enteritidis* PT6c was the cause of a salmonellosis outbreak in Sunny Beach and was exported to Finland, and likely to the United Kingdom, Norway, Sweden and Germany.

Introduction

Background

Salmonella has long been recognised as an important food-borne pathogen which can cause symptoms in humans ranging from self-limited enteric infections to enteric fever. In the European Union (EU), serovars *Salmonella* Enteritidis and *Salmonella* Typhimurium are the most frequent causes of gastroenteritis in humans. In 2006, more than 160,000 cases of salmonellosis were reported in the EU resulting in an annual incidence of 34.6 cases per 100,000 population [1]. In 2001-2007, the annual number of salmonellosis cases in Bulgaria has varied between 800-1000 (incidence 9.3-15.4/100,000). Most of the Bulgarian cases have been sporadic. However, a few outbreaks have also emerged every year due to consumption of contaminated eggs and/or dairy products. Approximately 70% of the strains isolated in Bulgaria are of serovar *S. Enteritidis*.

Cases in Bulgaria

In the beginning of June 2008, a salmonellosis outbreak caused by *S. Enteritidis* occurred among personnel of a hotel in Sunny Beach resort situated on the Black Sea coast, in Nessebar municipality, Burgas region, Bulgaria. In all, 14 persons with symptoms of fever ($\leq 39.5^{\circ}\text{C}$), vomiting, abdominal pain and diarrhoea were reported of whom seven sought medical care in the local hospital. At the same time, during June and July 2008, four sporadic *S. Enteritidis* cases were also reported in the Burgas region two of whom were foreign tourists including an eight-year-old Finnish girl.

Cases in other countries

Finland: In June - July 2008, the Gastrointestinal Infection Unit of the National Institute for Health and Welfare (THL) in Helsinki identified 195 *S. Enteritidis* cases: 28 of them were connected to a trip to Bulgaria, including 19 that were of the phage type PT6c.

United Kingdom: On 8 July 2008 the Health Protection Agency reported an increased number of *S. Enteritidis* cases with an unusual phage type PT6c to the European Centre for Disease Prevention and Control (ECDC). Twelve patients were followed up and it turned out that all had been on holiday in Bulgaria preceding their illness.

Sweden: On 10 July 2008 Sweden reported to ECDC 29 *Salmonella* cases among travellers returning from Bulgaria during June and July. In Sweden, *Salmonella* strains related to travels abroad are not routinely serotyped. Nevertheless, 10 strains that were serotyped were all *S. Enteritidis*, and six of these cases were traced back to hotels in Nessebar and Sunny Beach. The 10 *S. Enteritidis* strains were also phage typed but the strains were not PT6c.

Germany reported to ECDC two *S. Enteritidis* cases linked to Bulgaria. One of these travellers had stayed in hotel at Nessebar from 23 to 30 May 2008 and had symptoms of salmonellosis starting from 28 May. More information about the other case was not available.

Norway reported a total of 76 salmonellosis cases in 2008 linked with travelling to Bulgaria, of which 48 isolates had been identified as *S. Enteritidis*. Of these isolates, eight were phage typed and four of them were of PT6c.

Aims of the study

Effective epidemiological surveillance of salmonellosis requires accurate subtyping of the strains in order to trace the potential sources of infection and the geographical distribution of different *Salmonella* serovars. A number of different phenotypic and genotypic methods have been used in microbiology laboratories for subtyping. Phage typing (PT) and pulsed-field gel electrophoresis (PFGE) are currently the only internationally standardised typing methods for *S. Enteritidis*. In order to find out relatedness between Bulgarian and Finnish *S. Enteritidis* isolates potentially associated with an outbreak occurring in Bulgaria, we initiated an investigation of those strains by phage typing and PFGE.

Methods

Surveillance

Salmonellosis is one of the notifiable communicable diseases in Bulgaria. The surveillance of salmonellosis in the country is laboratory-based. The primary diagnostics is performed by the regional clinical microbiology laboratories that are legally required to record and report all cases discovered in their regions. They send all outbreak-associated and some sporadic *Salmonella* strains to the National Reference Laboratory for Enteric Pathogens for confirmation, serotyping and antimicrobial susceptibility testing.

Outbreak investigation

Following notification of the outbreak in a hotel in Sunny Beach resort, field epidemiological investigation was performed including interviews with cases and contact persons, and active case-finding among hotel personnel. Stool samples were taken from 14 symptomatic employees of the hotel and 100 asymptomatic

contacts identified among personnel and families of cases, and were cultured by standard methods for *Salmonella*.

Collection and laboratory investigation of food samples

Food samples taken from five dishes prepared in the hotel restaurant and suspected based on the interviews (scrambled eggs with chopped peppers and tomatoes, chicken soup, chicken goulash, fish fried in egg and bread-crumbs, chicken giblets with rice) were tested for salmonellosis. Additionally, mash potatoes and two kinds of eggs, disinfected and not disinfected, were also examined in the microbiology laboratory at the Regional Inspectorate of Public Health Protection and Control in Burgas. ISO standard 6579 was used for the investigation of those food samples.

Pheno- and genotyping of the isolates

Subtyping of the *S. Enteritidis* strains was conducted at the National Reference Centre for *Salmonella* in Finland. *S. Enteritidis* isolates from Burgas region available at the National Reference Laboratory of Bulgaria (n=15) and all *S. Enteritidis* strains isolated in Finland during June and July 2008 (n=195) were examined. Isolates were phage typed using the method described in Ward et al. [2]. In addition, the 15 Bulgarian strains and the 19 Finnish PT6c isolates were analysed for genetic relatedness by PFGE using XbaI according to the Centers for Disease Control and Prevention (CDC) PulseNet protocol [3]. The PFGE patterns were named using international standardised nomenclature of PulseNet Europe.

Results

In the outbreak investigation, five of the 14 symptomatic persons among the hotel personnel and eight of the 100 contacts tested positive for *S. Enteritidis*. The food samples were all negative for *S. Enteritidis*. Nevertheless, scrambled eggs with chopped peppers and tomatoes were suspected as the most likely source of the

TABLE

Phage types and PFGE types of *S. Enteritidis* strains isolated in Bulgaria from stool specimens of 16 patients, June – July 2008

Date of receipt in NCIPD	Patient's nationality	National region	Occupation (Link to a Sunny Beach hotel)	Phage type	PFGE type
14/05/2008	Bulgarian	Burgas	other (sporadic)	1	SENTXB.0001
20/06/2008	Bulgarian	Burgas	other (sporadic)	6c	SENTXB.0010
20/06/2008	Bulgarian	Burgas	other (sporadic)	6c	SENTXB.0010
20/06/2008	Bulgarian	Burgas	other (sporadic)	6c	SENTXB.0010
20/06/2008	Bulgarian	Burgas	other (sporadic)	6c	SENTXB.0010
20/06/2008	Bulgarian	Burgas	other (sporadic)	4	SENTXB.0001
20/06/2008	Bulgarian	Burgas	other (sporadic)	4	SENTXB.0001
24/06/2008	Bulgarian	Burgas	personnel	6c	SENTXB.0010
24/06/2008	Bulgarian	Burgas	personnel	6c	SENTXB.0010
24/06/2008	Bulgarian	Burgas	personnel	6c	SENTXB.0010
24/06/2008	Bulgarian	Burgas	personnel	6c	SENTXB.0010
02/07/2008	Bulgarian	Burgas	other (sporadic)	1	SENTXB.0001
02/07/2008	Bulgarian	Burgas	other (sporadic)	21	SENTXB.0001
02/07/2008	Bulgarian	Burgas	other (sporadic)	4	SENTXB.0001
02/07/2008	Bulgarian	Burgas	other (sporadic)	1	SENTXB.0001
25/07/2008	Finnish		tourist	6c	SENTXB.0010

Abbreviations: NCIPD: National Center of Infectious and Parasitic Diseases, Sofia, Bulgaria, PFGE: pulse-field gel electrophoresis

outbreak, since all persons affected reported having eaten this dish before onset of symptoms.

As a result of subtyping performed at the National Reference Centre in Finland, within the 15 Bulgarian *S. Enteritidis* strains, four different PTs were found: PT1, PT4, PT6c and PT21 (Table). The predominant PT was PT6c: eight out of 15 strains belonged to this phage type.

Among the 195 *S. Enteritidis* strains isolated in Finland, 15 PTs were found, the most common being PT21 with 54 strains. The cases were associated with trips to 25 countries, most commonly to Greece (34 cases), Bulgaria (28 cases), Turkey (27 cases) and Estonia (18 cases). The 28 strains isolated from Finns who visited Bulgaria in June or July were identified as PT6c (19 cases), PT6 (1 case), PT13a (2 cases), PT14b (2 cases) and PT22 (4 cases). PT6c strains were found only in samples of patients returning from Bulgaria.

PFGE analysis showed that the 15 *S. Enteritidis* strains from Bulgaria and the 19 strains from Finland could be assigned to only two characteristic PFGE patterns (SENTXB.0001 and SENTXB.0010) which were obtained after *Xba*I digestion (Figure). The *S. Enteritidis* PT6c strains (8 Bulgarian and 19 Finnish isolates) were indistinguishable from each other by this analysis and were classified as SENTXB.0010.

Discussion

Numerous reports of salmonellosis associated with foreign travel and caused by different *Salmonella* serovars have been published [4,5,6,7,8]. In this study, we report multinational cases of salmonellosis caused by *S. Enteritidis* PT6c associated with travel to Bulgaria.

S. Enteritidis PT6c is a rare phage type. None of the *S. Enteritidis* strains isolated from 978 patients and typed at the National Reference Centre in Finland in 2007 and 2008 (by June) belonged to this PT (A. Siitonen, unpublished data). Also, to our knowledge, there are no previously published reports on outbreaks caused by this phage type. Several European countries, namely

Austria, Norway, Hungary, Ireland, Finland, United Kingdom, Germany and Sweden reported to ECDC sporadic *S. Enteritidis* cases in 2008 among tourists returning from Bulgaria. United Kingdom, Finland and Norway found *S. Enteritidis* PT6c among *Salmonella* strains isolated from the samples taken from their citizens returning from Bulgaria.

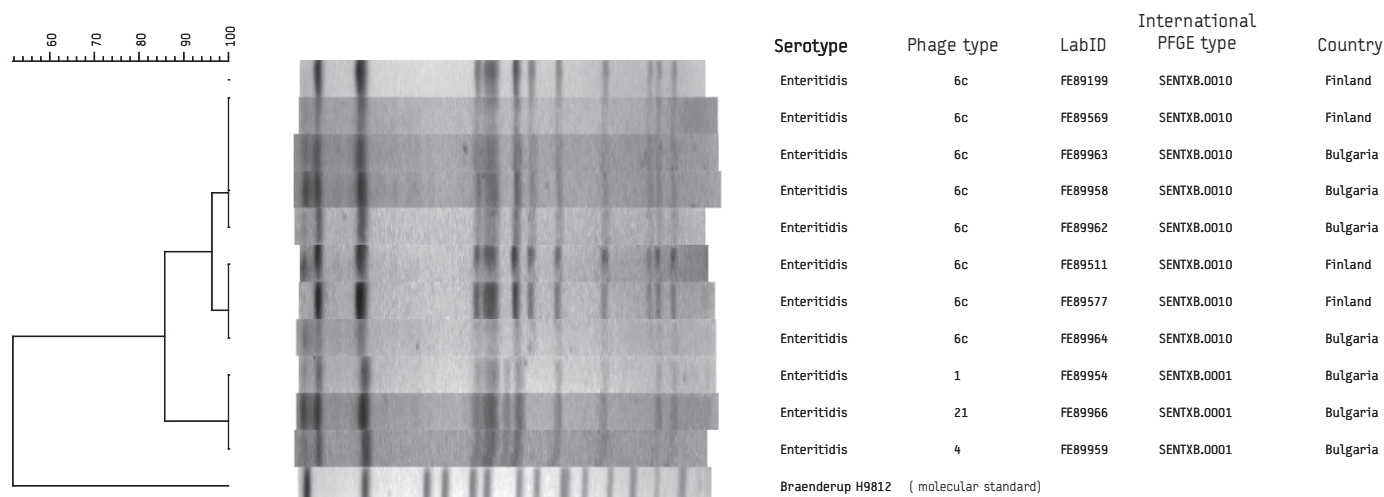
In our study, 27 strains (8 Bulgarian and 19 Finnish) proved to be *S. Enteritidis* PT6c. The facts that i) the Bulgarian and Finnish isolates were of the same phage type and ii) among the Finnish *S. Enteritidis* strains, phage type PT6c was only found in isolates from patients who returned from Bulgaria, indicate an epidemiological link between them. Among Bulgarian *S. Enteritidis* PT6c isolates, four strains were taken from cases associated with *S. Enteritidis* outbreak in a hotel in Sunny Beach resort. *S. Enteritidis* PT6c was also isolated from an eight- year-old Finnish girl who had stayed in another hotel in Sunny Beach resort (Table). In the hotels of this resort, many Finns and people of other nationalities spent their summer holidays in 2008. After July, PT6c was still found in Finnish *S. Enteritidis* cases returning from Bulgaria in August (n=4), September (n=9) and October (n=1) but no cases were detected in November.

The finding that all tested Bulgarian and Finnish *S. Enteritidis* PT6c isolates had the same PFGE profile (SENTXB.0010) also suggests that these strains could have the same origin and be epidemiologically linked. However, this PFGE profile can be found in *S. Enteritidis* strains of several PTs, including common phage types PT4 [9], PT1 and PT21 (A. Siitonen, unpublished). This emphasises the importance and applicability of phage typing over genotyping in epidemiological surveillance of salmonellosis.

Tourism is the fastest growing industry worldwide. The globalisation leads to faster spreading of infectious diseases including salmonellosis and requires us to consider them from a global perspective. International networks worldwide and collaboration of the health authorities are essential for an effective control of salmonellosis.

FIGURE

Cluster analysis based on the PFGE profiles of *S. Enteritidis* isolates originating from Bulgaria and Finland, June-July 2008



In conclusion, the alert system administered by ECDC, the effective collaboration between EU countries and the use of internationally standardised subtyping methods such as phage typing and PFGE, enabled us to establish an international clustered record of *Salmonella* infections caused by a rare *S. Enteritidis* PT6c and its association with travelling to Bulgaria. We believe that *S. Enteritidis* PT6c was the cause of outbreaks of salmonellosis in resorts situated at the Bulgarian Black Sea coast and was exported to Finland and most likely to the United Kingdom, Norway, Sweden and Germany. In this study, the importance of a multinational approach for the determination of potential sources of salmonellosis and its geographical distribution was demonstrated.

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Surveillance and outbreak reports

EPIDEMIOLOGY OF HUMAN CRYPTOSPORIDIOSIS IN IRELAND, 2004-2006: ANALYSIS OF NATIONAL NOTIFICATION DATA

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Cryptosporidium is a protozoal parasite which is of public health interest primarily due to its frequent association with drinking water. Since cryptosporidiosis became a notifiable human disease in 2004 in Ireland, evidence has been growing as to the national burden of illness caused by this pathogen. Nationally, crude incidence rates of between 8.7 and 13.4 per 100,000 were reported annually in the period 2004-2006. Rural areas reported more cases, with regional incidence rates as high as 31.4/100,000 per year. Over this time period, there has consistently been a peak in the number of notifications in springtime, contrasting with the reported seasonal distribution of cases elsewhere in Europe. Outbreak surveillance data suggest that drinking water is an important transmission route for general outbreaks, with person-to-person spread more common in family outbreaks. *Cryptosporidium* is an important gastrointestinal pathogen in Ireland, with much still to be learned about its epidemiology here.

Introduction

Cryptosporidium is a protozoal parasite which first came to prominence as a cause of chronic diarrhoea in acquired immunodeficiency syndrome (AIDS) patients in the United States in the early eighties, but is now recognised as a major cause of gastroenteritis in immunocompetent patients, leading to a spectrum of disease from asymptomatic shedding to watery non-bloody diarrhoea, sometimes accompanied by abdominal pain, nausea, anorexia, fever and weight loss.

A key feature of *Cryptosporidium* oocysts is their relative resistance to chlorination, and as a result, it has gained notoriety as a public health issue due to its association with municipal drinking water supplies and public swimming pools, both of which have been implicated as the vehicle of transmission in a number of outbreaks in developed countries [1-4].

Awareness of cryptosporidiosis as a cause of gastrointestinal illness has risen in Ireland in 2007 due in part to a large waterborne outbreak in a city in the west of Ireland [5], which resulted in an extended boil water notice for several months. Evidence as to the national burden of illness caused by this pathogen, however, has been growing since 2004, when cryptosporidiosis became a notifiable human disease. This paper outlines current knowledge of the national epidemiology of human cryptosporidiosis in Ireland, drawing on recent disease notification and outbreak surveillance data.

Materials and methods

Human cryptosporidiosis has been subject to mandatory notification in Ireland since 1 January 2004. As for all notifiable diseases, basic demographic data is reported routinely on all cases. The case definition adopted since 2004 is based on the European Union (EU) case definition [6]. Notification data are maintained in the Computerised Infectious Disease Reporting (CIDR) system, a central national repository for all infectious disease notification data in Ireland. The notification data used in this report is based on information retrieved from CIDR on cases of cryptosporidiosis reported from 2004 to 2006, as of 5 December 2007.

Reporting of infectious disease outbreaks has been mandatory in Ireland since 1 January 2004, and data on outbreaks of cryptosporidiosis between 2004 and 2006 were retrieved from CIDR for that time period. Prior to 2004, outbreak data had been collected on a non-statutory basis from July 2001 by the Health Protection Surveillance Center (HPSC). In this paper, we present data collected on outbreaks reported between July 2001 and December 2006.

The administration of public health activities in Ireland is divided into eight regional departments, referred to as Health Service Executive (HSE) areas. Regional incidence rates were calculated as crude incidence rates per 100,000 population using Central Statistics Office (CSO) population data from the 2006 census as denominator. For age-specific incidence rates, seven cases were omitted from the analyses, as the variable 'age' was not available.

Results and discussion

Incidence

In the three years from 2004 to 2006, between 367 and 568 cases of cryptosporidiosis were notified annually, resulting in a crude incidence rate (CIR) of between 8.7 and 13.4 per 100,000 population (Table 1).

To put this in perspective relative to other causes of intestinal infectious disease in Ireland, the reported incidence of cryptosporidiosis is similar to that of salmonellosis in the same time period. A recent study [7] compared the incidence of cryptosporidiosis in 16 countries in Europe in 2005, and reported an overall crude incidence rate of 1.9 per 100,000 in these countries, with Ireland having the highest CIR of the 16 countries included in the study. Even bearing in mind that comparison between surveillance data from different countries is difficult due to variation in diagnostic, investigative, and surveillance practices,

all of which influence reporting in each of the countries, it is clear that cryptosporidiosis is an important cause of gastrointestinal illness in Ireland.

Seasonal distribution

Between 2004 and 2006 in Ireland, there was a consistent pattern in the seasonal distribution of notifications (Figure 1), with the highest numbers of cases reported from April to June. Overall, 55% of cases occurred during the second quarter of the year in these three years.

This contrasts strongly with the seasonal distribution of cases reported in the United Kingdom (UK), Sweden and Germany [7], where the highest numbers of cases in 2005 occurred in autumn, and with Spain, where a seasonal peak was observed in June, suggesting that the epidemiology of cryptosporidiosis in Ireland differs appreciably from the current epidemiology of cryptosporidiosis in these countries. Spring peaks in incidence coincide with peak calving and lambing activities, and are believed to be associated primarily with transmission from animal sources [8-9]. Prior to the introduction of the 1999 UK water regulations, there had been both a spring and an autumn peak in the number of cases, whereas in the recent years a significant reduction in the number of spring cases has been noted and attributed to the effectiveness of these regulations [10].

It appears that the seasonal distribution of cases in Ireland more closely reflects that reported for New Zealand, which also displays a pronounced spring peak, albeit in addition to a smaller autumn peak [11].

Age-sex distribution

Figure 2 shows the mean annual age-specific incidence rate in Ireland 2004-2006. Notifications for children predominated with over three quarters of all reported cases being less than 10 years of age. There were more male (n=729) than female (n=634) cases notified.

It is widely accepted that there is a degree of bias in reporting of illness in young children for many diseases, as parents are more likely to seek medical attention and health personnel more

likely to take specimens for children than for adults. Moreover, higher incidences among younger children may reflect a lack of immunity as many older people may have already had exposure to *Cryptosporidium* during their lifetime.

However, selective criteria based on age are also commonly applied to samples for *Cryptosporidium* testing in diagnostic laboratories (a selection criterion probably not as frequently applied when testing for other common gastrointestinal pathogens such as *Salmonella* or *Campylobacter*), and this could have a significant impact on the reported age distribution. For example, although the HPSC report on waterborne cryptosporidiosis [12] recommends that all stool specimens received by laboratories from symptomatic individuals be tested for *Cryptosporidium*, it acknowledges that where resources are limited an age threshold of ten years can be applied, although this threshold is generally not employed during outbreaks. The effect on the reported incidence of disease in Ireland will be dependent on the number of laboratories that have opted to apply an age threshold as a selective criterion when examining specimens for *Cryptosporidium*. Anecdotally, we are aware that many laboratories do not test routinely for *Cryptosporidium* in adults, and a laboratory survey to investigate this further is underway. The recent outbreak in the west of Ireland provides some evidence of the potential effect of an age threshold selective criterion [5]. It was reported that more than 40% of cases in the outbreak were older than 15 years of age. On this basis, it is possible that in areas served by diagnostic laboratories where an age-threshold such as this is applied, around 40% of cases could remain undetected.

Regional distribution

There was a marked difference between the reported incidence of cryptosporidiosis in the HSE-East (which includes the capital city Dublin and thus has a larger proportion of urban dwellers than other HSE areas) and other more rural areas of Ireland (Table 1). The highest average crude incidence rate in 2004-2006 was reported in the HSE-West (22.5/100,000), which had a particularly high incidence in 2005 (31.4/100,000), followed by HSE-Midlands (18.2/100,000), where data were influenced by the occurrence of a community outbreak in 2004, which resulted in a higher than average incidence in that year.

TABLE 1

Crude incidence rates (CIR) of cryptosporidiosis by Health Service Executive (HSE) area and year, Ireland, 2004-2006

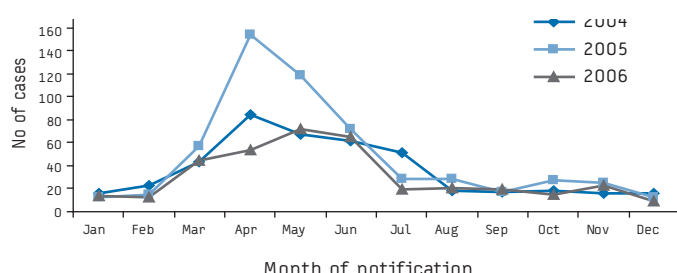
HSE area	CIR (95% confidence interval)			
	2004	2005	2006	Average 2004-2006
East	1.5 (0.9-2.2)	2.5 (1.7-3.3)	0.5 (0.1-0.8)	1.5 (0.9-2.1)
Midland	24.6 (18.5-30.8)	14.3 (9.6-19.0)	15.5 (10.6-20.4)	18.2 (12.9-23.4)
Mid-West	12.5 (8.8-16.1)	15.5 (11.5-19.6)	15.5 (11.5-19.6)	14.5 (10.6-18.4)
North-East	7.6 (4.9-10.3)	15.7 (11.8-19.7)	7.1 (4.5-9.7)	10.2 (7.0-13.3)
North-West	16.9 (11.6-22.1)	18.1 (12.7-23.6)	12.7 (8.1-17.2)	15.9 (10.8-21.0)
South-East	17.4 (13.6-21.2)	21.3 (17.1-25.5)	13.2 (9.9-16.6)	17.3 (13.5-21.1)
South	11.9 (9.2-14.6)	16.9 (13.7-20.1)	11.9 (9.2-14.6)	13.6 (10.7-16.5)
West	18.6 (14.4-22.7)	31.4 (26.0-36.8)	17.4 (13.4-21.4)	22.5 (17.9-27.0)
Ireland	10.2 (9.2-11.1)	13.4 (12.3-14.5)	8.7 (7.8-9.5)	10.7 (9.6-11.7)

Data source: Computerised Infectious Disease Reporting (CIDR), Health Protection Surveillance Centre (HPSC), Ireland

Contact with farm animals and visiting farms are known risk factors for sporadic cryptosporidiosis. Living in an area with poorer water treatment has also been reported as a risk factor for cryptosporidiosis. Moreover, a high proportion of rural dwellers in Ireland are served by private wells, many of which would not have barriers against *Cryptosporidium*. We believe that the lower incidence reported from the HSE-East may reflect at least in part a true difference in risk between urban and rural dwellers.

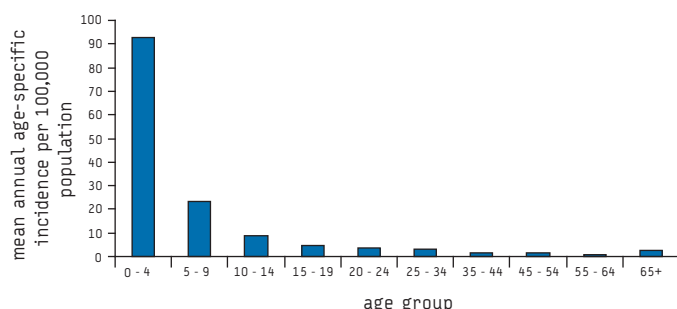
There was also a noticeable difference between the age distribution of cases in different HSE areas. For example, the HSE-East reported a higher proportion of adult cases older than 15 years of age among their relatively small number of cases - almost three quarters of cases reported in the period 2004-2006 (Table 2). Interestingly, several of these cases were reported as travel-associated. In contrast, only 5-8% of cases in the HSE-West and HSE-Mid West during the same period were above the age of 15 years. Some of this variation is likely to be due to surveillance bias as discussed above.

FIGURE 1
Seasonal distribution of cryptosporidiosis cases, Ireland, 2004-2006



Data source: Computerised Infectious Disease Reporting (CIDR), Health Protection Surveillance Centre (HPSC), Ireland

FIGURE 2
Mean annual age-specific incidence rates of cryptosporidiosis, Ireland, 2004-2006



Data source: Computerised Infectious Disease Reporting (CIDR), Health Protection Surveillance Centre (HPSC), Ireland

Transmission routes for cryptosporidiosis in Ireland

Outbreak surveillance data provide important information on disease transmission routes. Between July 2001 and December 2006, 23 outbreaks of cryptosporidiosis were reported to the HPSC, including nine family outbreaks and 14 general outbreaks (outbreaks involving cases who were not part of the same family) (Figure 3). Water was reported as a suspected source for 14 outbreaks - drinking water for twelve outbreaks (11 general and one family outbreak) and recreational water for two family outbreaks. For general outbreaks, drinking water was the most common suspected transmission route, while for family outbreaks person-to-person transmission appears more important (Figure 3). For one waterborne outbreak during this time period, there was analytical evidence demonstrating a statistically significant increase in the likelihood of disease in those who consumed tap water [13].

Some of the best available evidence internationally on the epidemiology of *Cryptosporidium* is from the United Kingdom [3,8,14-19]. Evidence has been gathered through a combination of outbreak surveillance, case control studies and speciation of positive human specimens from routine human surveillance. The most commonly reported outbreak transmission routes in England and Wales have been public water supplies and swimming pools [3,14,19]. Swimming pools have not been reported as a location for outbreaks for cryptosporidiosis in Ireland during this time period, although there were cases reported associated with an outbreak linked to a swimming pool in Spain in 2003 [20].

Since surveillance for human cryptosporidiosis began in 2004 following the revision of the list of notifiable diseases [21], much has been learned about the epidemiology of human cryptosporidiosis in Ireland. There remain, however, a number of issues on which further data would be advantageous. In the United Kingdom, speciation of human isolates has proved invaluable in elucidating the epidemiology of infection in conjunction with case control studies and other surveillance data [19]. In the time period 2004-2006 in Ireland, typing of positive human specimens was only rarely undertaken except in the event of outbreaks. A small number of hospital laboratories in Ireland have started to have *Cryptosporidium*-positive specimens typed on a routine basis

TABLE 2
Age distribution of cryptosporidiosis notifications, Ireland, 2004-2006, by Health Service Executive (HSE) area

HSE-area	<5 yrs	5-14 yrs	15+ yrs	Total
East	13 (19.4%)	5 (7.5%)	49 (73.1%)	67
Midland	99 (72.3%)	25 (18.2%)	13 (9.5%)	137
Mid-West	103 (66.0%)	41 (26.3%)	12 (7.7%)	156
North-East	68 (56.7%)	24 (20.0%)	28 (23.3%)	120
North-West	62 (55.9%)	33 (29.7%)	16 (14.4%)	111
South-East	134 (56.1%)	34 (14.2%)	71 (29.7%)	239
South	152 (60.1%)	59 (23.3%)	42 (16.6%)	253
West	208 (75.4%)	53 (19.2%)	15 (5.4%)	276
Total	839 (61.7%)	274 (20.2%)	246 (18.1%)	1359

Note: includes only cases where information on age was available
Data source: Computerised Infectious Disease Reporting (CIDR), Health Protection Surveillance Centre (HPSC), Ireland

since 2007, and the results of these studies will provide the first systematic evidence of the relative importance of different species in this country. Provisional results from these studies suggest that *C. parvum* is more common than *C. hominis* among sporadic cases in Ireland (unpublished data). A research study by Zintl *et al* [22] concurs with this.

Another issue that needs to be assessed quantitatively is the relative importance of travel-associated infection. In the United Kingdom, international travel is believed to play an important part in the epidemiology of *Cryptosporidium* in autumn months. The available data at national level in Ireland on 'country of infection' is limited but has been improving over time. Given that a number of community outbreaks have been reported in Ireland, it is likely that indigenous cases form the majority of cases, however, this would be important to verify, and hopefully can be achieved with time.

Increasingly, circumstantial evidence from outbreak surveillance data in Ireland suggests that drinking water and person-to-person spread are important transmission routes during outbreaks. Elsewhere, personal risk factors for sporadic cryptosporidiosis have variously included factors such as international travel, contact with cattle, visiting farms, contact with another person with diarrhoea, swimming in a public swimming pool, freshwater swimming, having a chronic medical condition, and drinking unboiled tap water [15-16, 23-26]. Socio-economic risk factors such as living in an area which has a high proportion of individuals of higher socio-economic status, living in an area with a high rate of manure application to land, or living in an area with poorer water treatment, were reported by Lake *et al* [27]. A key advantage of the Hunter study was that analyses of the case control study were undertaken separately for *C. parvum* and *C. hominis* cases permitting determination of the species-specific risk factors [15]. The only factor which significantly

increased the risk of *C. parvum* infection was touching or handling farm animals, while international travel, spending time sleeping or sitting on the ground and nappy-changing contact with a child less than five years of age were associated with *C. hominis* infection. No studies have been published in Ireland on the risk factors for sporadic cryptosporidiosis. Further research on this topic would be very valuable, in particular in the light of the seasonal distribution of cases and the likely difference in epidemiology that this suggests.

Acknowledgements

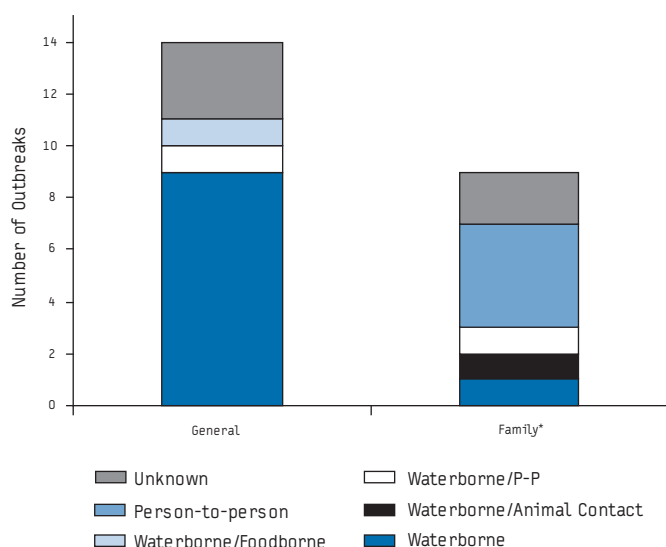
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FIGURE 3

Outbreaks of cryptosporidiosis in Ireland, July 2001 – December 2006, by suspected transmission route (n=23)



*Note: recreational water was suspected in two of the three family outbreaks where water was believed to have played a role in transmission
Data source: Computerised Infectious Disease Reporting (CIDR), Health Protection Surveillance Centre (HPSC), Ireland

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