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Health Protection Surveillance Centre

25-27 Middle Gardiner St
Dublin 1, Ireland

Ph +353 1 876 5300
Fx +353 1 856 1299
E info@mailx.hse.ie
www.hpsc.ie

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Two Cases of Trichinosis in Polish Nationals living in Ireland.

Introduction

A 27 year old Polish national, presented to the Emergency Department in St. James's Hospital (SJH) on 8th June 2007 with a history of fevers, periorbital swelling, conjunctival injection, myalgia and diarrhoea for the preceding ten days. He had been living in Ireland for the past year but had returned to North West Poland for holidays in April 2007. While there he purchased and consumed lightly smoked pork sausages. He first consumed some of the sausages on 5th or 6th May. He returned to Ireland on 11th May.

On 28th May he began to feel unwell and noticed his eyes began to swell and he developed conjunctivitis. He attended an ophthalmologist for this complaint, which was diagnosed as a viral conjunctivitis. The following day he developed a high-grade fever, diarrhoea and pains in his legs. He also complained of a dry cough. His general practitioner treated him with chloromycetin eye drops and co-amoxiclav for three days with no resolution of symptoms. He was then referred to SJH for further investigation.

The patient indicated that Polish radio had reported an outbreak of trichinosis in the region of Poland that he had visited. As a result of this and based on his clinical presentation and characteristic blood investigations (eosinophilia, elevated muscle enzymes) a presumptive diagnosis of trichinosis was made. Serology was sent for *Trichinella* antibody, and treatment was commenced with mebendazole 200mg tds. His Polish fiancée who had travelled with him to Poland and consumed some of the sausages was also ill with similar symptoms and following appropriate investigations was commenced on mebendazole 200mg tds. On the 27th June, serology results on both patients were strongly positive for *Trichinella* antibody.

Public health investigation

Appropriate contact was made after the first case presentation with the relevant authorities within the Health Protection Surveillance Centre and the Department of Public Health, HSE East. HPSC confirmed the outbreak of trichinosis in North West Poland with the Polish authorities. Preliminary investigations point towards sausages produced from uncooked pork meat as the source of the outbreak. The product is not for sale in Ireland. However, the index case brought some of the sausages with him on his return to Ireland.

In addition to the index case and his fiancée, there were two female members in the Irish household. However, neither had consumed the sausage and it was not eaten by anyone other than the index and his fiancée. None of the sausage was available for testing. The sausage had been placed on the top shelf of the fridge so potential for cross contamination existed. In view of this the household members were advised to seek medical help if they developed symptoms and to contact the Public Health Department, HSE Eastern area.

All A & E consultants, ophthalmologists and general practitioners in the HSE Eastern area were alerted to the possibility of similar cases occurring in individuals entering the country from Poland who may have consumed contaminated product.

An alert was also sent to the Polish community in Ireland through Ireland's Polish language weekly newspaper.

What is trichinosis?

Trichinosis is a zoonotic disease with a worldwide distribution. It is very rare in Ireland. It develops when humans ingest undercooked meat containing the larvae of the roundworm *Trichinella*. The larvae can be found in the meat of certain wild carnivorous (meat-eating) animals but can also occur in domestic pigs. The severity of the infection usually correlates with the number of ingested larvae. The incubation period is 5-45 days. Person-to-person transmission does not occur.¹

Prevention

- Trichinosis is prevented by cooking meat until the juices run clear or all parts reach a temperature of 71°C (160°F).¹
- Freezing pork less than 6 inches thick for 20 days at -15°C (5°F) will kill all common types of *Trichinella* larvae.² However, freezing wild game meats, unlike freezing pork products, is not guaranteed to kill all larvae.
- The EU requires all pig, horse and game meat for human consumption to be inspected before export to prevent distribution of infected meat to consumers.
- Avoid eating illegally imported meat.

A factsheet on trichinosis is available at www.ndsc.ie/php/printerfriendly.php?sPageUrl=http://www.ndsc.ie/hpsc/A-Z/Zoonotic/Trichinellosis/Factsheet/index.html.

McHugh G, Low J, Healy ML, Clarke S, SJH;
Kiely D, Hayes C, HSE E

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Surveillance of Infectious Disease Outbreaks in Ireland, 2005

Introduction

The principal objective of the national outbreak surveillance system is to gain information on the epidemiology of all outbreaks of infectious disease in Ireland. More specific objectives include measuring the burden of illness caused by outbreaks, identifying high-risk groups in the population and estimating the workload involved in the management of outbreaks. The information gathered can be used to inform health professionals on the causes and factors contributing to outbreaks, to target prevention strategies and to monitor the effectiveness of prevention programmes.

Outbreak definition

*An outbreak of infection or foodborne illness may be defined as two or more linked cases of the same illness or the situation where the observed number of cases exceeds the expected number, or a single case of disease caused by a significant pathogen. Outbreaks may be confined to some of the members of one family or may be more widespread and involve cases either locally, nationally or internationally.*¹

Methods

Since 1st January 2004, outbreaks or "unusual clusters or changing patterns of illness" became notifiable under the Amendment to the Infectious Diseases Regulations.² Since that date, medical practitioners and clinical directors of diagnostic laboratories are required to notify to the medical officer of health any unusual clusters or changing patterns of illness, and individual cases thereof, that may be of public health concern.

In addition, since 1st January 2004, all outbreak data are being entered onto the CIDR system database either directly by the Health Service Executive (HSE) area (if that area has gone live onto CIDR) or indirectly by staff in the Health Protection Surveillance Centre (HPSC).

Results

During 2005, 175 outbreaks of infectious disease were notified to HPSC, of which 161 were gastrointestinal/ infectious intestinal disease (IID) outbreaks. The IID outbreaks were responsible for at least 2,591 people becoming ill, and there were 205 reported hospitalisations. The regional distribution of all outbreaks of infectious disease, and specifically IID outbreaks, are detailed in table 1. The highest number of outbreaks was reported from the HSE Eastern area (n=65), although the highest outbreak rate was in HSE SE (7.3/100,000 population). The lowest rate was reported from HSE W (1.8/100,000).

Causative pathogen

The breakdown of IID and non-IID outbreaks by pathogen are outlined in tables 2 and 3 respectively. Continuing the trend observed in previous years, the IID outbreaks in 2005 have been dominated by

Table 1. All outbreaks of ID, total numbers ill in ID outbreaks, and number of IID outbreaks reported by HSE area, 2005

HSE area	No. of outbreaks	Outbreak rate per 100,000 pop.	No. ill all outbreaks	No. of IID outbreaks
E	65	4.6	1,534	61
M	10	4.4	117	9
MW	7	2.1	35	7
NE	13	3.8	125	12
NW	11	5.0	148	10
SE	31	7.3	477	31
S	31	5.3	255	25
W	7	1.8	57	6
Total	175	2.8	2,748	161

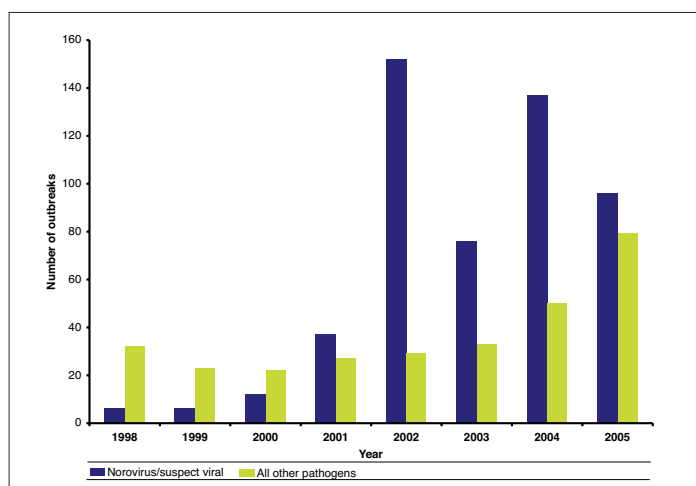


Figure 1. Number of outbreaks by year and by pathogen, 1998-2005 (Data prior to July 2001 provided by FSAI)

norovirus/ suspect viral outbreaks, accounting for 60% of all IID outbreaks reported in 2005 (figure 1). The largest single outbreak reported in 2005 was a norovirus outbreak in a hospital involving 187 people.

After norovirus, the next most commonly reported outbreaks were enterohaemorrhagic *E. coli* (EHEC), *Salmonella enterica*, *Campylobacter* and *Cryptosporidium*.

There were 20 outbreaks of EHEC (19 VTEC) reported in 2005, four general and 16 family outbreaks. The largest VTEC outbreak involved a significant investigation and occurred at a private house/crèche in the Mid West Area. Nine cases were ill and 18 cases in total were confirmed positive for VTEC O157. Two cases with haemolytic uraemic syndrome (HUS) were hospitalised. Results from a case-control study indicated that potential exposure to drinking water from a vulnerable local private group water scheme was a risk factor.³

Seventeen outbreaks of *S. enterica* were reported in 2005, affecting a total of 52 people and resulting in 12 hospitalisations. There were three general and 14 family outbreaks. Three outbreaks were travel related with the Czech Republic, Tunisia and Spain cited as the countries of infection.

The number of outbreaks reported that were attributable to *Campylobacter* rose from one in 2004 to eight in 2005. All eight outbreaks were family outbreaks, and the suspected mode of transmission recorded was foodborne (5), person-to-person (2) and unknown (1).

Table 2. Pathogens associated with IID outbreaks notified in 2005

Disease	No. of outbreaks	No. ill
Noroviral infection	61	1,891
Suspected norovirus	32	297
EHEC	20	53
Salmonella	17	52
Campylobacter	8	17
Cryptosporidiosis	6	49
Norovirus & <i>C. difficile</i>	3	32
<i>C. difficile</i>	2	20
Shigellosis	2	44
Giardiasis	1	3
Rotavirus	1	14
Unknown	8	119
Total	161	2,591

There were six outbreaks of cryptosporidiosis in 2005, four general and two family outbreaks. The largest outbreak reported was a community outbreak affecting 31 people in the Carlow town area. The mode of transmission was

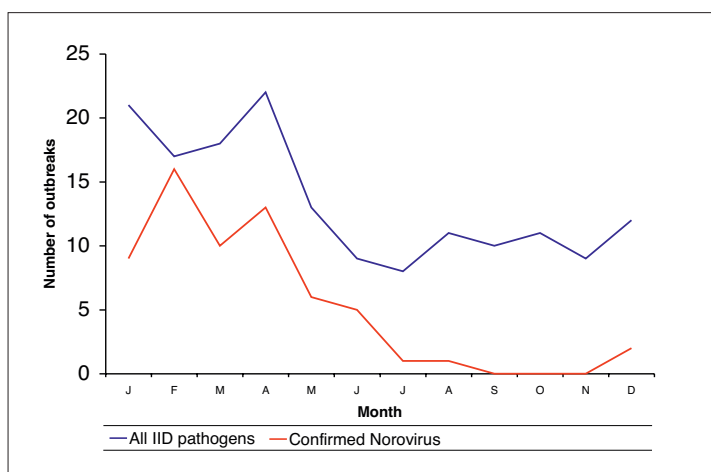


Figure 2. Seasonal distribution of all IID and confirmed norovirus outbreaks, 2005

illness of first case, it is seen that the majority of outbreaks occurred in the first 4 months of the year (figure 2). This peak is attributable to the substantial number of confirmed/suspected norovirus outbreaks that occurred at this time.

Discussion

There was a slight decrease in the overall number of outbreaks reported nationally in 2005, with 161 outbreaks of IID notified, compared to 169 in 2004.

As observed in recent years, viral gastroenteritis, principally caused by norovirus, accounted for the majority of outbreaks reported in 2005 (60% of IID outbreaks confirmed/suspected norovirus). Detailed molecular detection and typing of norovirus isolates was introduced by the National Virus Reference Laboratory (NVRL) in 2003, which has enabled us to study in much greater detail the molecular epidemiology of strains causing outbreaks. These data are routinely submitted to the European network 'DIVINE-NET', which is an extension of the previous network entitled 'Foodborne Viruses in Europe'.⁵ DIVINE-NET aims to merge epidemiological and virological data on outbreaks of viral gastroenteritis, including norovirus, across Europe.

In 2005, travel-associated outbreaks were a significant feature. In total nine outbreaks were travel-associated, including a large norovirus outbreak on a cruise ship that affected 95 individuals.

An outbreak of shigellosis associated with travel to Egypt was identified in June 2005 involving over half the passengers on a flight from Luxor, in Egypt to Dublin. The Egyptian authorities were alerted and appropriate measures were put in place. Subsequent investigations could not conclusively identify the source of infection.⁶

In October 2005, Health Protection Scotland identified an international outbreak of *S. Goldcoast* infection in tourists returning from Majorca. An alert through Enter-Net and the European Commission's Early Warning and Response system (EWRS) led to an international response with active case finding. In total, 148 cases were identified in 10 different countries – including six cases in Ireland. Despite extensive investigations the source of infection was not identified. The outbreak was declared over on the 1st December 2005.

Outbreak data have been entered onto the CIDR system since the beginning of 2004, therefore real time data on outbreaks are available to all CIDR users nationally as they go-live on the system. With the continued national roll-out of CIDR, it is hoped enhanced surveillance data on all outbreaks of infectious disease will be even more timely and complete as users enter their own outbreak data. This will enable epidemiological, microbiological and environmental data relating to the outbreak to be shared locally and nationally, and should greatly assist in the management and control of outbreaks, as well as allowing analysis of the national data to inform future public health policies.

Barbara Foley, Fiona Cloak, Paul McKeown, HPSC

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Table 3. Non-IID outbreaks notified in 2005

Disease	No. of outbreaks	No. ill
Mumps	6	39
HiB	1	2
Influenza A	1	42
Influenza B	1	33
Legionellosis	1	-
Tuberculosis	1	8
Probable varicella	1	9
Scabies	1	4
Suspected streptococcal infection	1	20
Total	14	157

Table 4. IID outbreaks by location, 2005

Location	No. of IID outbreaks
Hospital	59
Residential institution	40
Private house	38
Travel related	9
Community outbreak	4
Hotel	3
Creche	1
Public house	1
Other	3
Not specified	3
Total	161

suspected as waterborne although only low levels of *Cryptosporidium* were found in the town water supplies. A boil water notice was issued and subsequently lifted when sampling confirmed that the town supply was free from *Cryptosporidium*.⁴

Fourteen outbreaks of non-IID/gastroenteric diseases

were notified in 2005, six of which were reported to be outbreaks of mumps (table 3).

Mode of transmission

Similar to previous years, person-to-person spread is the mode of transmission reported for the majority of outbreaks of IID in 2005. Most of these outbreaks were due to norovirus/ suspect viral. Like 2004, the foodborne route was the second most frequently suspected mode of transmission and was identified in over 12 outbreaks in 2005. For many outbreaks more than one mode of transmission was suspected.

Location

As in previous years, the commonest location in which outbreaks occurred in 2005 was the healthcare setting (table 4) with 61% of all reported IID outbreaks occurring in these settings. Nine outbreaks were associated with foreign travel in 2005 compared to only one travel-associated outbreak in 2004. *Salmonella* accounted for three of these outbreaks followed by norovirus (2), EHEC (2), *Cryptosporidium* (1) and *Shigella* (1).

Seasonal distribution

When the IID outbreaks in 2005 are analysed by month of onset of

Introduction

A new variant strain of *C. trachomatis* (vCT) has recently been characterised in Sweden following an unexpected dramatic decrease in the incidence of chlamydial infection between November 2005 and December 2006.¹ This vCT strain has a 377 bp deletion in the cryptic plasmid in the area targeted by both the Roche and Abbott nucleic acid amplification assays for the detection of *C. trachomatis*. As a consequence, the commercial kits manufactured by these two companies generate false negative results for patients infected with this variant strain. The Becton Dickinson ProbeTec (BD ProbeTec) assay is based on a fragment of DNA located elsewhere on the cryptic plasmid and is able to detect this new variant.

The laboratory at St. James's Hospital (SJH), Dublin, currently uses the BD ProbeTec assay to screen clinical samples for the presence of *C. trachomatis*. All positive samples are then confirmed using the Roche COBAS Amplicor assay. The use of this testing algorithm places the laboratory in an ideal position to detect the vCT strain. A retrospective study, involving specimens taken between July and December 2006, failed to detect vCT in our patient population.² Subsequently, a male patient and his female partner were investigated for vCT as their demographic details (one partner was of Swedish origin) and laboratory results (BD ProbeTec assay positive and Roche Amplicor assay negative) were suggestive of a vCT infection. Further molecular analysis was performed on two first void urine samples from the male and one endocervical swab from the female. We now report the first two cases of vCT in Ireland.

Materials and Methods

Template DNA was extracted from first void urine samples using the QIAamp® Viral RNA Mini Kit and from the endocervical swab using the QIAamp® DNA Mini Kit (QIAGEN GmbH, Hilden, Germany). The extracts were amplified with primers that flanked the 377 bp deletion in the cryptic plasmid, as previously described.² The expected sizes of the product were 583 bp (wildtype) or 206 bp (variant) in length. DNA sequencing was performed on the amplified product using the ABI Prism® 3130 xl DNA analyser and the sequences were compared with the sequence of the cryptic plasmid of the variant strain (GenBank accession number: EF121757). Further characterisation of the strains was performed by amplification of the *ompA* gene using the primers CTY-F (5'-ATG AAA AAA CTC TTG AAA TCG -3') and CTY-R (5'-CTC AAC TGT AAC TGC GTA TTT -3'). Sequencing of the *omp1* gene required the use of the additional primers CTSEQ-F (5'-GCT CAA TCT AAA CCT AAA -3') and CTSEQ-R (5'-TTT AGG TTT AGA TTG AGC -3').

Results

Gel electrophoresis initially indicated that all three samples contained vCT, as the PCR product size was ~ 200 bp in all samples as shown in figure 1. The presence of the vCT strain was confirmed by DNA sequencing of the three PCR products and an alignment with the sequence of the cryptic plasmid of vCT (GenBank accession number: EF121757). All three test samples contained *C. trachomatis* which harboured the exact same 377 bp deletion as has been described.¹ Further analysis showed that these vCT strains were genotype E.

Discussion

The newly-discovered vCT strain has a selective advantage over wildtype chlamydial strains as it has remained undetected by the widely used Roche and Abbott assays. Laboratories using either assay will not detect this variant strain and these infections will go undiagnosed allowing the potential for the infection to spread. Active surveillance for vCT is vital to determine the extent of the spread of this strain and should provide essential epidemiological information. The variant strain was recently detected in samples from two patients in Oslo, one being of Swedish origin.³ This is the second report of the variant strain existing outside of Sweden. Interestingly, like the cases in Oslo, one of the patients was also

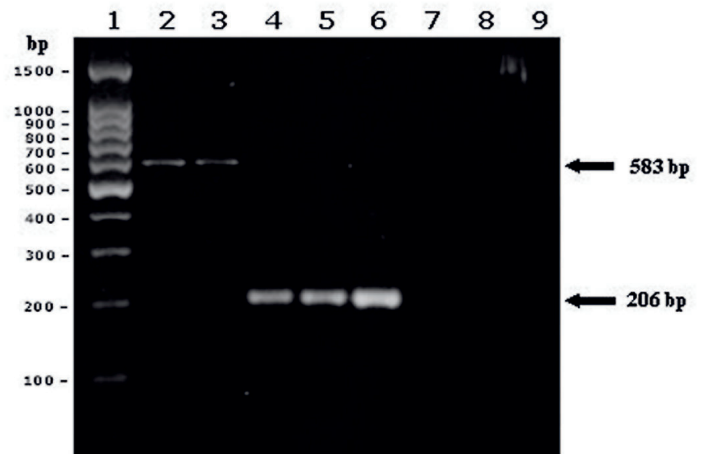


Figure 1: Agarose gel showing amplified product after PCR to amplify a region of the *C. trachomatis* cryptic plasmid. Lane 1: molecular weight marker; Lane 2 and 3: wildtype *C. trachomatis* controls; Lanes 4, 5 and 6: patient samples; Lane 7 and 8: negative controls; Lane 9: non-template control. Block arrows indicate the expected product size of the wildtype (583 bp) and vCT strains (206 bp).

of Swedish origin. This would imply that the strain did originate in Sweden and is now spreading into the rest of Europe. Further analysis of 13 vCT isolates in Örebro county, Sweden, showed that they were all genotype E and had a unique multilocus sequence type.⁴ Characterisation of the vCT strains at SJH also determined them to be genotype E. This suggests that the Irish vCT strains are closely related to the strains characterised in Sweden.

It is of critical importance that all diagnostic laboratories are aware of the emergence of this vCT strain. False negative test results will result in chlamydial infections remaining undiagnosed and this may result in serious complications, such as infertility and pelvic inflammatory disease, for patients in later life. False negative results will also reduce the impact of screening programmes due to the decrease in contact tracing and the provision of appropriate treatment. Roche and Abbott are currently modifying their molecular assays to ensure that the assays will detect both wildtype and variant strains. The emergence of the variant strain is an interesting example of a bacteria evolving to avoid detection in the modern world of molecular detection methods. In order to minimise the risk of a similar situation arising in the future, it is the opinion of the authors that dual target detection in molecular assays to detect *C. trachomatis* infections should now become standard. The development of dual target molecular assays may also be considered for other pathogens, which rely on molecular methods for their detection.

Lynagh Y, Walsh A, (adwalsh@stjames.ie),
and Crowley B, St James's Hospital

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