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Outbreak of Vero cytotoxin-producing *E. coli* (O157 VTEC) associated with a producing butcher in Leeds

West Yorkshire Health Protection Unit is currently involved in investigating an ongoing outbreak of Vero cytotoxin-producing *E. coli* (O157 VTEC) connected with a producing butcher's shop in Armley, Leeds.

Twenty-five cases of presumptive *E. coli* O157 have been isolated since the beginning of July, with a further case positive on latex testing and awaiting confirmation. A small number of further possible cases are still being investigated. Eleven of the 25 confirmed cases have been hospitalized, although the most have now returned home and all are recovering.

Onset of illness was between 23 June 2006 and 3 July 2006 for the confirmed cases. The HPA Laboratory of Enteric Pathogens (LEP) has to date confirmed 16 isolates (12 human, four food) as VTEC O157. The human isolates are phage type (PT) 4 and 21/28, and the food isolates were all PT4. All 16 isolates possess genes for Vero cytotoxin (VT) 2.

Early on in the investigation, a common link was noted with cooked meats purchased from a producing butcher's in Armley, Leeds. Investigations by Leeds City Council Environmental Health Officers revealed the butcher sold both raw and cooked meats in the shop and also supplied cooked meats to another 18 suppliers, including delicatessens and sandwich shops, across the Leeds area. The butcher also sold pre-cooked meats, sandwiches and pre-prepared salads from a stall in Leeds market. Food and environmental samples were taken from both the shop and the market stall and the butcher concerned agreed to voluntary closure and recalled cooked meat from all the outlets supplied. The HPA Food Water and Environment Laboratory in Leeds isolated presumptive *E. coli* O157 in a cooked meat sample taken from the market stall. These isolates were confirmed by LEP as VTEC O157 PT4.

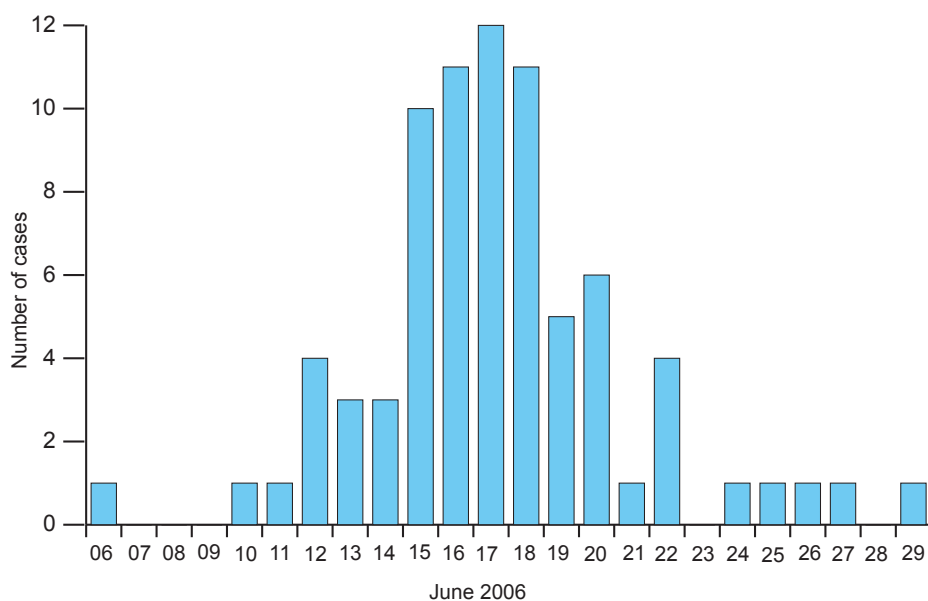
Wide-spread media publicity led to a number of other possible cases coming forward and investigations are ongoing. Direct links with consumed food from either the butcher's shop or one of the supplied outlets have been made for 16 of the 25 confirmed cases to date.

National increase in *Salmonella* Ajiobo infection. England & Wales, June 2006: update

Since 20 June 2006 the Health Protection Agency (HPA) Laboratory of Enteric Pathogens (LEP) have received and provisionally confirmed 119 human cases of *Salmonella* Ajiobo infection in residents of England and Wales. There are 103 primary UK-acquired cases of fully sensitive *S.* Ajiobo infection: seven cases who reported recent travel outside the United Kingdom (UK) prior to illness, five who were infected with strains resistant to the LEP panel of antimicrobial drugs, and four who reported recent contact with other individuals with gastrointestinal symptoms have been excluded.

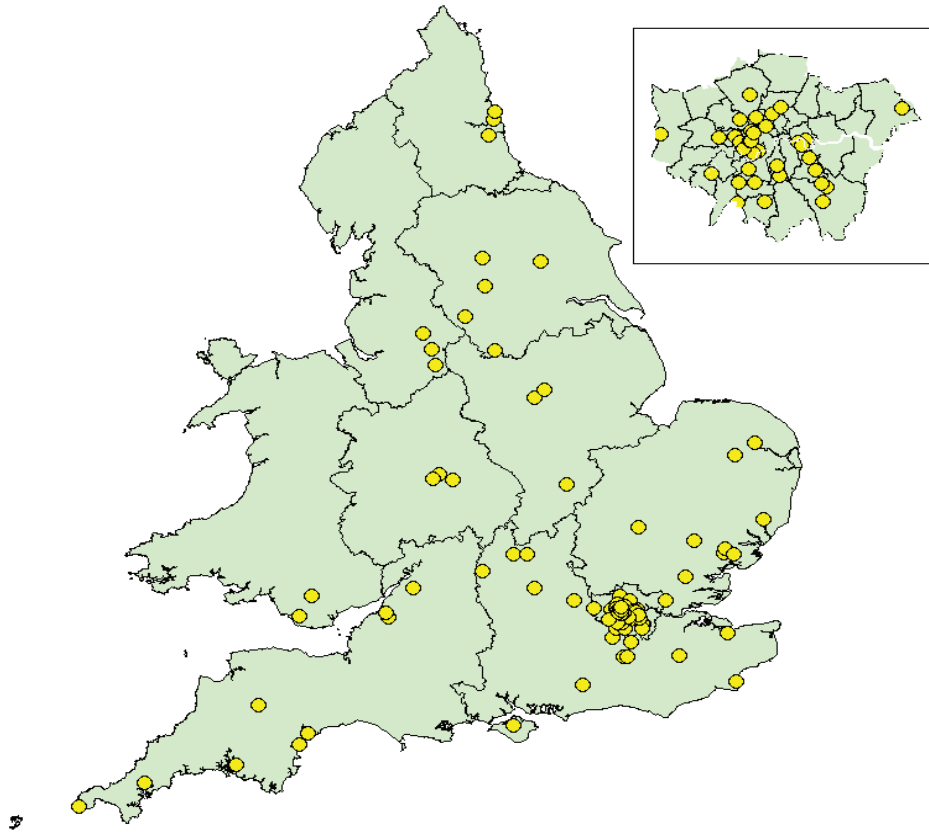
The onset of illness for the majority of cases was between 6 and 29 June (figure 1), with one case reporting an onset of illness on 21 April. The epidemic curve currently suggests a point source exposure over a number of days.

Figure 1. Epidemic curve (N=78)



For those cases where age is known (N=101), most cases are adults aged between 20 and 59 years (80%), although patients' age ranges from 4 months to 84 years. Females are overrepresented (59/103; 57%). Six out of 59 patients (10%) are known to have been admitted to hospital as a result of their illness. Most cases have been reported from laboratories in London (43/103; 42%) and the south east of England (18/103; 17%) but cases have been reported from all NHS regions (figure 2).

Figure 2. GIS map based on patients' postcode (n=87)



Epidemiological and microbiological investigations into the cause of the outbreak continue.

Treatment outcome results for tuberculosis cases reported in 2003

The results of treatment outcome monitoring (TOM) for tuberculosis cases reported in 2003 to Enhanced Tuberculosis Surveillance in England, Wales, and Northern Ireland have been finalised. Information on outcome relates to the patient's status 12 months after commencing treatment or notification. Monitoring the completion of treatment in all tuberculosis cases is a key tool in assessing the effectiveness of the national tuberculosis control effort. The definitions used are in line with European and World Health Organization (WHO) recommendations, but adapted to the United Kingdom (UK) context. For more background information please see the first TOM report [1].

There were 6837 tuberculosis cases reported in 2003, of which 120 were subsequently found not to have tuberculosis. Of the remaining 6717, 90% had an outcome reported compared with 85% in 2002 and 79% in 2001. Initial analysis of the 6018 cases with a reported outcome revealed:

- 79% (4746/6018) of all tuberculosis cases and 79% (835/1061) of new infectious pulmonary cases completed treatment.
- Treatment completion was higher for foreign-born cases 82% (3233/3961) compared to cases born in the UK 74% (1200/1624).
- 7% (440/6018) of cases died, 4% (215/6018) of cases were still on initially planned treatment and 1% (60/6018) had their treatment stopped.
- 9% (557/6018) of cases had an outcome reported as either unknown, lost to follow-up or transferred out.

Although a higher proportion of cases had an outcome returned, the proportion completing treatment among this cohort (79%) was very similar to previous years (78% in 2002, 79% in 2001). WHO recommends at least 85% of new infectious pulmonary cases should successfully complete treatment. A substantial proportion of UK born cases derive from elderly populations aged over 65 years, in whom the risk of death from tuberculosis or other causes is much higher.

We would like to acknowledge the efforts made by Local and Regional Services in coordinating outcome returns and the clinicians and nurses who participate in TOM. The tuberculosis pages on the HPA web site have been updated with the 2003 outcome data including a breakdown by regional level and can be viewed at:

http://www.hpa.org.uk/infections/topics_az/tb/epidemiology/tables.htm#tom;

http://www.hpa.org.uk/infections/topics_az/tb/epidemiology/figures/figures_menu.htm

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Important changes to the childhood immunisation programme: CMO Letter

The Chief Medical Officer for England has published a new letter about changes to the routine childhood immunisation programme [1]. The letter is directly available for download from the Department of Health website at:

<<http://www.dh.gov.uk/assetRoot/04/13/71/75/04137175.pdf>>

From 4 September 2006, the following changes will be introduced:-

- Pneumococcal vaccine will be introduced to the routine childhood immunisation programme, and the schedule for MenC and Hib vaccines will be modified.
- The new routine schedule given in Annex 1, Table 1 of the Letter will be introduced. This schedule requires an additional immunisation visit at 12 months of age.
- A pneumococcal vaccination catch-up programme will be carried out for children aged under two years.

The Joint Committee on Vaccination and Immunisation has endorsed these changes.

The detailed vaccination schedule can also be seen on the HPA website at

<http://www.hpa.org.uk/infections/topics_az/vaccination/new_sched_sept2006.htm>, and details of the pneumococcal vaccine at

<http://www.hpa.org.uk/infections/topics_az/pneumococcal/vaccine/vaccine.htm>.

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Inspector of Microbiology webpages

The Inspector of Microbiology and Infection Control now has bespoke webpages on the Department of Health website. They can be accessed at:

<<http://www.dh.gov.uk/PolicyAndGuidance/HealthAndSocialCareTopics/MicrobiologyAndInfectionControl/fs/en>>.

Current items include a directory of microbiology laboratories in England, a request to share best practice in reducing MRSA, and health protection outputs of NHS microbiology laboratories.

Enteric

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Preliminary information has been received about the following outbreaks.

Health Protection Unit	Organism	Location of food prepared or served	Month of outbreak	Number ill	Cases positive	Suspect vehicle	Evidence
Bedfordshire & Hertfordshire	<i>S. Enteritidis</i> PT13A	Function	June	30	6	–	–
West Yorkshire	<i>E. coli</i> O157	Retailer	June	6	6	–	–

M (microbiological): identification of an organism of the same type from cases and in the suspect vehicle, or vehicle ingredient(s), or detection of toxin in faeces or food; D (descriptive): other evidence, usually descriptive, reported by local investigators as indicating the suspect vehicle or food; S (statistical): a significant statistical association between consumption of the suspect vehicle(s) and being a case.

Salmonella infections (faecal specimens), England and Wales, reports to the HPA (salmonella data set): May 2006

Details of the 6771 Salmonella infections recorded in May 2006 are given in the table below. In June 2006, 731 Salmonella infections were recorded and preliminary information was received one outbreak (see General outbreaks table above).

	May 2006
S. Enteritidis (PT4)	141
S. Enteritidis (other PTs)	211
S. Typhimurium	76
S. Virchow	33
Others (typed)	310
Total Salmonella (provisional data)*	771

*Figures quoted from the Health Protection Agency salmonella data set are for isolates confirmed and typed by Laboratory of Enteric Pathogens (LEP).

Common gastrointestinal infections, England and Wales, laboratory reports: weeks 23-26/06

Laboratory reports	Number of reports received				Total reports 23-26/06	Cumulative total to	
	23/06	24/06	25/06	26/06		26/06	26/05
<i>Campylobacter</i>	1149	1165	1001	657	3972	17,791	20,564
<i>Escherichia coli</i> O157*	20	18	29	43	110	296	245
<i>Salmonella</i> †	201	134	201	119	655	3644	3765
<i>Shigella sonnei</i>	9	6	6	7	28	253	453
Rotavirus	73	52	52	27	204	11,887	12,475
Norovirus	36	23	11	10	80	2990	2234
Cryptosporidium	27	45	30	17	119	976	975
Giardia	31	34	40	23	128	1068	1239

*Vero cytotoxin-producing isolates (data from Health Protection Agency's Laboratory of Enteric Pathogens (LEP)).

† Data from Health Protection Agency's Laboratory of Enteric Pathogens.

Less common gastrointestinal infections, England and Wales laboratory reports: 14-26/06

Laboratory reports	Total reports	Cumulative total to	Cumulative total to
	14-26/04	26/2004	26/2003
Adenovirus	3	11	14
Astrovirus	5	36	108
Sapovirus	1	3	17
<i>Shigella flexneri</i>	83	157	165
<i>Plesiomonas</i>	5	17	14
<i>Vibrio</i>	8	11	14
<i>Yersinia</i>	3	7	14
<i>Entamoeba histolytica</i>	20	42	45
<i>Blastocystis hominis</i>	37	85	202
<i>Dientamoeba fragilis</i>	17	40	82

Vero cytotoxin-producing *Escherichia coli* O157: 2005

In 2005, 950 isolations of Vero cytotoxin-producing *Escherichia coli* O157 (VTEC O157) were confirmed by the HPA's Laboratory of Enteric Pathogens (LEP) from human infections in England and Wales. This was a 36% increase compared with the 699 isolates in 2004 [1] and was the fifth highest total recorded since 1997 (1097). The distribution of VTEC O157 according to the sender's health region is shown in table 1 where the data are compared to 2004. The overall increase was attributed in part to confirmation of 180 isolates from Wales most of which were associated with a large outbreak of infection [2].

Table 1. VTEC O157 isolations in different health regions 2005

Region/Country	Total 2005	% total England and Wales 2005	Total 2004	% total England and Wales 2004
North East	60	6.3	53	7.6
North West	134	14.1	110	15.7
Yorkshire and the Humber	145	15.3	95	13.6
East Midlands	45	4.7	45	6.4
West Midlands	54	5.7	80	11.4
East of England	71	7.5	70	10
London	77	8.1	37	5.3
South East	83	8.7	88	12.6
South West	101	10.6	100	14.3
England (Total)	770	-	678	-
Wales	180	18.9	21	3
England and Wales (Total)	950	-	699	-

Approximately 74% of strains in 2005 had VT2 genes only and 35% had both VT1 and VT2; five isolates were VT1 only. The strains belonged to 19 designated phage types (PTs), but 77% belonged to PTs 21/28, 8, and 2. Table 2 compares the data with those from 2004 [1], for the most frequently isolated types .

Table 2. Predominant phage types of VTEC O157 from human infections in England and Wales : 2005 and 2004

Rank	Phage type	2005: % of total	2004: % of total (rank)
1	21/28	44	29 (1)
2	8	22	23 (2)
3	2	11	22 (3)
4	32	5	5 (4)
5	4	4	4 (5)
6	34	3	3 (6)
	Other	11	14

The most prevalent types were the same as in 2004. The large increase in the proportion of PT21/28 strains reflected the association of this type with the south Wales outbreak [2]. Despite a national increase in PT8 isolates between October and December 2005 compared with 2004 (3), the overall proportion of this type did not change since 2004. In contrast, there was a continuing decline in PT2 that has accompanied the predominance of PT21/28 since 1999 [4]. Approximately 3% of strains reacted with the typing phages, but did not conform to a designated type (RDNC). Provisionally, there were 13 general outbreaks of infection in 2005 of which nine were caused by PT21/28; there were single outbreaks caused by PT1, PT2, PT8, and an RDNC strain.

In Scotland, isolations of *E.coli* O157 reported to Health Protection Scotland [5] fell by 18%, from 209 in 2004 to 172 in 2005. Strains of PT21/28 accounted for 44% of Scottish isolates, compared to 58% in 2004 (the lowest proportion since 1998). PT8 (23%) and PT2 (11%) were next most common, both showing marked changes in frequency over 2004 (12% and 17% respectively), but 35% of Scottish PT8 cases in 2005 were imported. Provisionally, PT21/28 was identified in seven of the 13 general outbreaks in Scotland in 2005.

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Surveillance of waterborne disease in England and Wales: summary of 2005

Seven waterborne outbreaks of infectious intestinal disease were reported to the Health Protection Agency's Centre for Infections (CFI) in 2005 (table 1), six of which were due to cryptosporidium. The largest two outbreaks were associated with public drinking water supplies, while the remaining five were linked to recreational water exposure.

The strength of association between water exposure and human disease was determined according to microbiological test results from cases, microbiological examination of water samples, and descriptive and analytical epidemiology [1].

Table 1. Outbreaks of infectious intestinal disease associated with water in England and Wales: 2005

Outbreak reference number	Organism	Region	Month	Total affected (lab confirmed cases)	Suspected source of outbreak	Association between water exposure & illness
05/410	Cryptosporidium	South West	May	3 (2)	Descriptive epidemiology indicated a possible link between human illness and use of a public swimming pool.	Possible
05/514	Cryptosporidium	East	July	15 (9)	Outbreak occurred at an animal park. Descriptive epidemiology suggested an association with a children's paddling pool. Faecal indicator organisms were detected in pool water.	Probable
05/789	Giardia	North West	July	16 (16)	Descriptive epidemiology linked the outbreak to an inadequately chlorinated swimming pool.	Probable
05/623	Cryptosporidium	London	September to December	>129 (129)	Outbreak linked to several swimming pools. Samples from one pool contained oocysts. Descriptive epidemiology indicated a strong association between human illness and this pool.	Strongly associated with one pool; possibly linked to additional pools
05/554	Cryptosporidium	South East	August to November	88 61 <i>C. hominis</i> 1 hospital case	Outbreak linked to several swimming pools, one of which tested positive for oocysts. Descriptive epidemiology suggested a possible association between human illness and pool water exposure.	Possible
05/552	Cryptosporidium	South	September	140 (140)	Outbreak associated	Strong

		East	to November	5 hospital cases	with a public drinking water supply. Oocysts and faecal indicators were detected in treated water. Analytical epidemiology showed a dose-response relationship between illness and drinking un- boiled tap water.	
05/790	Cryptosporidium	Wales	September to January	232 (232) 218 <i>C. hominis</i>	Outbreak associated with consumption of mains water. Oocysts were detected in treated water. Case-control study showed a dose- response relationship between illness and consumption of tap water.	Strong

The first waterborne outbreak reported to CFI in 2005 involved three children who had been swimming in a public leisure centre pool in the South West region, on the same day. Two cases were laboratory-confirmed as cryptosporidiosis. Although cryptosporidium oocysts were not detected in pool water samples, descriptive epidemiology pointed to a possible association between illness and water exposure. Following the outbreak, the system was chlorinated and the frequency of filter backwashing was increased from weekly to twice weekly.

In July 2005 an outbreak affecting fifteen children was linked to a wildlife park paddling pool in the East of England region. All cases were under four years of age and nine tested positive for cryptosporidium. Pool water showed increased turbidity and contained faecal indicator bacteria, although samples were not tested for cryptosporidium. Coupled with descriptive epidemiological evidence, this is indicative of a probable association between illness and water exposure. The pool was subsequently closed and its filter changed.

An outbreak associated with a swimming pool in the North West region involved sixteen laboratory-confirmed cases of giardiasis; seven out of ten primary cases had been swimming in the pool. Although water samples did not contain cysts or faecal indicators, free chlorine levels were consistently insufficient. Together with descriptive epidemiology this indicates that human illness was probably associated with pool water exposure. As control measures, the pool was closed and its filter changed. An independent inspector conducted a complete assessment of pool water disinfection and maintenance systems, following which regular monitoring visits were undertaken by an Environmental Health officer.

In September an outbreak involving 129 laboratory-confirmed cases of cryptosporidiosis was linked to several swimming pools in London. Oocysts were detected in water samples taken from one pool. Coupled with descriptive epidemiology, this suggests that human illness was strongly associated with this pool and possibly linked to additional pools. Following detection of the outbreak, continual flocculation was introduced at the contaminated pool and filter backwashing was re-scheduled to take place overnight, to allow recirculation of water through the filters before pool use. Posters are now displayed advising children not to swim for two weeks after a period of diarrhoeal illness.

A second outbreak linked to various swimming pools occurred between August and November in the South East involving 88 cases, one of whom was hospitalised. Typing data confirmed 61 cases of *C. hominis* and seven of *C. parvum*. Water samples taken from one pool tested positive for cryptosporidium oocysts. Descriptive epidemiology suggested a possible association between human illness and water exposure. The oocyst-contaminated pool was temporarily closed and underwent six cycles of treatment. This pool was not re-opened until water samples tested negative for oocysts.

The second largest waterborne outbreak in 2005 was associated with a public water supply in the South East region, resulting in 140 laboratory-confirmed cases of cryptosporidiosis, five of whom were hospitalised. Of 76 primary cases (all *C. hominis*), approximately two-thirds were resident in an area served by a single mains supply. The implicated supply, derived from a river and a borehole, abstracts water 1.5 miles downstream of a sewage treatment plant discharge point and is subject to 24-hour cryptosporidium monitoring. During the outbreak treated water from both the water

treatment plant and the post-treatment service reservoir tested positive for oocysts, with respective maximum counts of 0.35 and 0.118 oocysts per 10 litres. The results of a case-control study revealed a strong dose-response relationship between drinking un-boiled tap water and the risk of human illness, with an odds ratio of 1.29 per glass consumed (95% CI: 1.29 to 1.48; $p < 0.001$). Immunocompromised people were advised to boil their tap water and water was supplied from an alternative source.

The largest waterborne outbreak in 2005 was associated with mains drinking water in north west Wales, between September and January [2]. Of 232 laboratory-confirmed cases, 218 were identified as *C. hominis*. The implicated source water, derived from an upland reservoir, was treated by micro-straining, pressurized sand filtration and chlorination. Continuous cryptosporidium-monitoring, in place from the beginning of November, detected oocysts in treated water. The highest oocyst count was 0.076/10 litres. Significant rainfall events occurred prior to and during the outbreak period, but are not unusual for the time of year; sewage effluent in the reservoir's catchment is hypothesized as the source of contamination. *C. hominis* was confirmed in sewage effluent, raw water, and treated water in distribution.

Epidemiological analysis confirmed a strong association between illness and consumption of un-boiled tap water, which was 6.1 times more likely among cases than controls (OR=6.1, 95% CI 1.8 to 23.8). A dose-response relationship was found with an increasing risk of disease with higher consumption of un-boiled tap water. A boil water notice was issued in areas supplied by the upland reservoir and to immunocompromised individuals across north west Wales. Ultraviolet treatment was installed at the water treatment works.

Legionnaires' Disease

Legionnaires' disease is an uncommon form of pneumonia caused by bacteria of the genus *Legionella*, most commonly the species *Legionella pneumophila*. Legionella bacteria naturally occur in warm aquatic habitats and are able to colonise water distribution systems. Infection occurs by inhalation of aerosols of, and occasionally aspiration of, water from a source colonised with legionellae. Outbreaks are commonly linked to wet cooling systems (cooling towers and evaporative condensers), hot and cold water systems or spa pools (also known as hot tubs and whirlpool spas), although a variety of other sources have been implicated including cutting fluids, clinical humidifiers, natural warm spas/hot springs, potting compost, humidifiers associated with food display cabinets, indoor fountains, aerobic effluent treatment lagoons, and air scrubbers.

An outbreak of legionnaires' disease is defined as two or more cases linked by area (residence, work, or locations visited) and with sufficiently close dates of onset (within six months for hospital and community acquired infections), for which there is strong epidemiological evidence of a common source of infection, with or without supporting microbiology. Individual cases are confirmed by clinical or radiological evidence of pneumonia along with positive microbiological diagnosis (culture, four-fold rise in serum antibodies or antigen detection in urine), or are taken as presumptive cases (single high titres in serum antibodies, PCR, or other methods). There may be considerable time lag between first and second outbreak cases (up to two years for travel-associated outbreaks), and as a result several outbreaks beginning in 2004 were recognised in 2005. For the purpose of this report, these are recorded as 2005 outbreaks.

In 2005, ten outbreaks of legionnaires' disease not associated with travel outside the UK (involving at least two English or Welsh cases) were reported to CFI, resulting in 36 cases and 4 deaths. The largest of these occurred in London between July and August 2005 and involved 12 cases. In two outbreaks a water distribution system was identified as the source of infection. The first of these occurred in London between July 2004 and February 2005 (recognised in 2005), and the second in Wales from August 2004 to June 2005, each involving two cases and one death. Cooling towers were implicated in two further instances in the West Midlands and the Yorkshire and Humber region, involving two and four cases respectively. In the remaining six outbreaks, which occurred in various regions across England, the source of infection was not identified.

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