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Deliberate release web pages

The PHLS deliberate release web pages at http://www.phls.co.uk/topics_az/deliberate_release/menu.htm contains essential public health, microbiological and clinical information concerning responding to threats from deliberate release of biological agents. It contains specific information relating to release of anthrax, botulism, smallpox, plague, tularaemia, viral haemorrhagic fevers, glanders/melioidosis, and a number of chemical agents (nerve agents, mustard gas, chlorine, hydrogen cyanide, phosgene and ricin). There are also specific pages on 'clinical tips' for practising clinicians and microbiologists, along with clinical pictures for recognition of anthrax and smallpox, and general information on a number of the diseases suitable for members of the public. The web pages have links to a number of relevant international web sites and to the Department of Health Emergency Planning Co-ordination Unit web site at <http://www.doh.gov.uk/epcu/>. The guidance on the PHLS web site has been developed by the Department of Health, the PHLS, and specialists in the relevant fields. It has been placed on the PHLS site so that this, with the EPCU site, functions as a single unified source for information. The guidance is continuously updated, so readers are advised to consult the web site regularly rather than down-loading or printing out the guidance. The dates of updates and additions are on the front page of the site.

An important recent addition to the PHLS site is *Interim guidance for NHS staff on the initial investigation and management of outbreaks and incidents of unusual illnesses*. This has been developed by the PHLS and the Chemical Incident Response Service. It has specific sections for the ambulance service, hospital clinicians, general practitioners, pathologists, local laboratories and public health officials. Its specific address is http://www.phls.co.uk/topics_az/deliberate_release/pdf/unusual_guidelines.pdf

Comments on this and other deliberate release guidance are welcome, and should be sent to DRcomments@phls.org.uk Not all NHS staff are aware of this guidance and *CDR Weekly* readers are encouraged to draw the web-site to the attention of colleagues in all parts of the NHS.

Salmonella Enteritidis outbreak in a London hospital – update

There has been a recent report by a London Hospital of an outbreak of *Salmonella* Enteritidis phage type (PT) 6a with resistance to nalidixic acid (Nx) and low level susceptibility to ciprofloxacin (Cp_L) (1). There were 27 hospital-acquired cases with dates of onset between 10 September and 7 October 2002. The source was thought to be imported raw shell eggs. Imported raw shell eggs were found to be contaminated with *S. Enteritidis* PT6a (Nx, Cp_L) during a national outbreak investigation of *S. Enteritidis* PT14b (2). The PHLS Laboratory of Enteric Pathogens (LEP) confirmed that the pulsed field gel electrophoresis patterns of the egg isolates and the clinical cases in the hospital outbreak were indistinguishable. Cases of infection with *S. Enteritidis* PT6a (Nx, Cp_L) stopped after the use of imported raw shell egg was discontinued.

Since the end of the outbreak of infection with *S. Enteritidis* PT14b, a second cluster of cases has been reported predominantly due to *S. Enteritidis* PT 1, also NxCp_L. The second cluster began on 19 October 2002 and 25 hospital-acquired cases have been identified. The latest date of onset in a confirmed case was 6 November 2002. Following the occurrence of this second outbreak, the hospital has discontinued the use of all raw shell egg and raw poultry on the premises while investigations into the source of the outbreak continue.

During the outbreaks there have been six deaths among patients identified as having salmonella, although none of these deaths have been attributed to salmonella infection. Four of the patients were terminally ill, and two died from serious, but unrelated, conditions.

1. PHLS. Nosocomial outbreak of *Salmonella* Enteritidis PT 6a (Nx, Cp_L). *Commun Dis Rep CDR Wkly* [serial online] 2002 [cited 14 November 2002]; **12**(43): news. Available at <http://www.phls.co.uk/publications/cdr/archive02/News/news4302.html#salmupdate>
2. PHLS. National outbreak of *Salmonella* Enteritidis PT 14b: update. *Commun Dis Rep CDR Wkly* [serial online] 2002 [cited 14 November 2002]; **12**(43): news. Available at <http://www.phls.co.uk/publications/cdr/archive02/News/news4302.html#salmupdate>

Gonococcal Resistance to Antimicrobials Surveillance Programme (GRASP): annual report 2001

The Gonococcal Resistance to Antimicrobials Surveillance Programme (GRASP) 2001 report has now been published (1) and is downloadable on the PHLS website, [click here](#) (pdf format). The report summarises findings from the second year of data collection in which 2666 isolates were analysed, and identifies key changes since the programme began.

GRASP is a collaborative sentinel surveillance initiative established between the PHLS Communicable Disease Surveillance Centre (CDSC), the Genitourinary Infections Reference Laboratory (Bristol), and Imperial College (London) with the aim of determining and monitoring the prevalence of antimicrobial resistance in *Neisseria gonorrhoeae* in England and Wales. The methodology for GRASP has been fully described elsewhere (2,3), but to summarise, during the months of June, July, and August 2001, 24 laboratories in England and Wales submitted all isolates of gonococci to two reference laboratories for antibiotic susceptibility testing. Their associated genitourinary medicine clinics (GUM) collected detailed demographic and behavioural data on all patients diagnosed with *gonorrhoea*. For each GRASP isolate, minimum inhibitory concentrations (MIC) are determined for penicillin, ciprofloxacin, tetracycline, ceftriaxone, and spectinomycin. In the 2001 collection the antimicrobial azithromycin was added to this protocol. The current UK guidelines now recommend the use of penicillin or fluoroquinolones (either ciprofloxacin or ofloxacin) for the treatment of uncomplicated gonococcal infection (4).

In 2001, 3.1% of isolates showed resistance ($\geq 1\text{mg/L}$) to ciprofloxacin, an increase from 2.1% found in

2000. A further 2.6% of isolates showed intermediate resistance to ciprofloxacin (≥ 0.125 to < 1 mg/L). Relatively high prevalence levels of ciprofloxacin resistance (≥ 1 mg/L) were found in the North West (8.6%), and South East regions (5.2%) compared to a low prevalence level of 1.8% in London. These high prevalences and increase in ciprofloxacin resistance are a cause for concern, as it remains the first line therapeutic choice in many clinics.

In 2001, 8.1% of isolates showed some form of resistance to penicillin compared to the 9.2% observed in 2000. High-level plasmid mediated penicillin resistance was greatest in the Eastern (6.2%) and West Midlands regions (5.2%). Chromosomally mediated penicillin resistance was highest in the South East (9.5%), Wales (8.0%), and London (5.3%). Continuing high levels of tetracycline resistance were observed in 2001: 32.5% of isolates showed some form of resistance to tetracycline compared to the 37.6% observed in 2000. All isolates were found to be susceptible to spectinomycin and ceftriaxone. Six isolates (0.3%) were found to be resistant to azithromycin.

The 2001 collection also confirmed previous findings of a non-random distribution of gonorrhoea with young people, homosexual men and some ethnic minorities bearing a disproportionate burden of disease (3). Twenty-nine per cent of *N. gonorrhoeae* diagnoses were found among women and 71% among men. Twenty-five per cent of all gonococcal infections were diagnosed among homo/bisexual men, who accounted for 32% of *gonorrhoea* diagnoses in London and 19% outside of London. Black and ethnic minority groups are also disproportionately affected accounting for 48% of *gonorrhoea* diagnoses in women and 39% in men in the participating clinics.

Almost one third (32%) of patients reporting previous infection with gonorrhoea suggesting continued contact with transmission networks. Forty-two per cent of women and 13% of men had an asymptomatic infection. Furthermore, 31% of individuals presented with a concurrent sexually transmitted infection (STI); 36% of females were concurrently infected with *Chlamydia trachomatis* compared to 17% in males. The substantial geographical clustering of gonococcal disease, the high proportion of asymptomatic and concurrent STI infections, all highlight the importance of sexual health screening among those at risk to prevent onward transmission and the development of serious complications.

These findings illustrate the public health importance of the enhanced antimicrobial resistance surveillance programme. Continued surveillance of the patterns and distribution of gonococcal antimicrobial resistance is required to ensure that prevention and treatment strategies remain responsive to the changing epidemiology of this sexually transmitted infection.

1. GRASP Steering Group. *The Gonococcal Resistance to Antimicrobials Surveillance Programme (GRASP) Year 2001*, report. London: Public Health laboratory Service 2002.
2. GRASP Steering Group. *The Gonococcal Resistance to Antimicrobials Surveillance Programme (GRASP) Year 2000*, report. London: Public Health Laboratory Service 2001.
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Cluster of wound botulism cases in injecting drug users in England – update

Six suspected cases of wound botulism have been reported to the PHLS Food Safety Microbiology Laboratory (FSML) following the public health alert issued on Wednesday, 30 October 2002 (1). All six cases were reported from England and are known to be injecting drug users. To date, the number of suspected cases of wound botulism reported in 2002 in the United Kingdom is now known to be sixteen. Nine of these cases have been laboratory confirmed. *Clostridium botulinum* toxin type A was detected in seven cases, and toxin type B in two. Tests are continuing on two patients; two cases previously reported as suspected cases of wound botulism are now thought to be more compatible with other clinical diagnoses.

All of these cases in 2002 have been reported from England, apart from one case reported from Scotland in February 2002. The most recent cases may be caused by a batch of drugs contaminated with the anaerobic bacterium *C. botulinum*.

An alert has been sent to all CCDCs asking them to inform local clinical services, drug services and

coroners of this cluster, and detailed advice is available on the PHLS website at http://www.phls.org.uk/topics_az/injectingdrugusers/menu.htm. Clinicians and CCDCs are asked to report any suspected cases of wound botulism to Moira Brett at the FSML (tel: 020 8200 4400 ext 4933) or to Sarah O'Brien or Peter Horby at the PHLS Communicable Disease Surveillance Centre (tel: 020 8200 6868 ext 4422/8076). Out of hours, suspected cases should be reported to the CDSC duty doctor on 020 8200 6868.

1. CDSC. Cluster of wound botulism cases in injecting drug users. *Commun Dis Rep CDR Wkly* [serial online] 2002 [cited 14 November 2002]; **12** (44): news. Available at <http://www.phls.org.uk/publications/cdr/archive02/News/news4402.html>

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Outbreak of Introduced *Plasmodium vivax* malaria in northern Queensland, Australia

An outbreak of *Plasmodium vivax* malaria has occurred in northern Queensland affecting ten people including three tourists from overseas, one from each of Canada, Germany, and Ireland. The outbreak occurred in the first two weeks of October 2002 and the source is believed to be a man who travelled to Indonesia in 2001 and Africa in 2002, and who had stayed at the campsite in September 2002. He was diagnosed with *Plasmodium vivax* malaria the day after he left the campsite.

This outbreak occurred in tourists staying at the Noah Beach campsite, which is a campsite in Daintree National Park, Cape Tribulation. It has approximately 20 to 25 sites and operates a self-registration system. Where contact details have been given, the Tropical Public Health Unit in Queensland have contacted campers to inform them of the situation and offer advice. Visitors to the park have included travellers from Holland, Israel, Italy, Sweden, Switzerland, and the United Kingdom. [Dianne Brookes, Tropical Public Health Unit, Queensland – Personal communication]

The individual who is believed to be the source of the infection was staying at the campsite in late September, and mosquitoes will have become infectious from about 8 October 2002 (1). Mosquito trapping revealed a large number of *Anopheles farauti* mosquitoes breeding in two creeks either side of the campsite. These are known to transmit malaria in northern Queensland, but they are short-lived, so it is likely that there are no more infectious mosquitoes present at the campsite (2), assuming there were no further human cases to act as a reservoir. Due to the nature of the campsite people do not often stay longer than a couple of nights, therefore the presence of a human reservoir is unlikely to be sustained. This, together with other considerations is likely to be why there has not been a larger outbreak or why malaria has not become re-established in Australia.

Malaria is not endemic in Australia, which was declared malaria free in 1981 (3). Imported cases do occur, however, and some additional cases have been derived from imported cases. There was a similar outbreak of *Pl. vivax* involving five cases in the Cape Tribulation area in 1986; the source was believed to be a man who had arrived from the Solomon Islands and spent a week in Cape Tribulation (4)

Pl. vivax infection is a prostrating, but not usually a life-threatening form of malaria. It is transmitted by the *Anopheles* mosquito. After a primary infection, some of the hypnozoites remain dormant in the liver cells and can be reactivated many months later, causing a further symptomatic attack (relapse). Health professionals are reminded to take a full travel history in those who present with an unexplained flu-like illness or fever. They should consider malaria in the differential diagnosis in any patients who present with these symptoms within the next year and may have stayed at the Noah Beach campsite in October 2002.

1. Locally acquired *P. vivax* malaria, North Queensland, Australia. In *ProMed Mail* [online]. Boston US: International Society for Infectious Diseases, 2 November 2002 [cited 13 November 2002]. Available at http://www.promedmail.org/pls/askus/f?p=2400:1001:433878::NO::F2400_P1001_BACK_PAGE,F2400_P1001_PUB_MAIL_ID:1010,19696

2. Health authorities confirm two more malaria cases. In *ProMed Mail* [online]. Boston US: International Society for Infectious Diseases, 2 November 2002 [cited 13 November 2002]. Available at http://www.promedmail.org/pls/askus/f?p=2400:1001:433878::NO::F2400_P1001_BACK_PAGE,F2400_P1001_PUB_MAIL_ID:1010,19696

3. World Health Organisation. Synopsis of the world malaria situation in 1981. *Wkly Epidemiol Rec* 1983; **58**: 197-9

4. Walker J. The role of a diagnostic reference laboratory in malaria surveillance. *Communicable Diseases Intelligence* 1996; **20** (13): 302-4

VIM metallo- β -lactamases conferring multi-resistance in *Pseudomonas aeruginosa* from the UK

Resistance to imipenem and meropenem

The carbapenems (imipenem and meropenem) have a broader spectrum than any other antibiotic available, and are invaluable for treating severe infections caused by multi-resistant pathogens. They are stable to prevalent β -lactamases, including the extended-spectrum and AmpC types that cause most of the cephalosporin resistance now seen in gram-negative opportunists. A few bacterial species, notably *Stenotrophomonas maltophilia* and some flavobacteria, do have carbapenem-hydrolysing β -lactamases 'carbapenemases', but these are infrequent pathogens or, in the case of *S. maltophilia*, are usually susceptible to co-trimoxazole. Slowly and inexorably, though, transferable carbapenemases are spreading into major pathogens. The key enzymes are zinc-dependent and belong to two families, IMP and VIM, which probably originated in unidentified environmental organisms (1,2). Other zinc-dependent and independent carbapenemases are known, but show less sign of spread (1,2).

VIM and IMP enzymes internationally

During the past decade IMP and VIM metallo- β -lactamases have become scattered among *Pseudomonas aeruginosa* and *Acinetobacter* spp. in the far east, where they have also been recorded from *Enterobacteriaceae* such as *Enterobacter*, *Citrobacter*, *Klebsiella*, *Serratia*, and *Shigella* spp (2,3). About 1% of *P. aeruginosa* isolates in Korea (6), Taiwan (5) and, Japan (4) now have VIM or IMP enzymes and, although this proportion is tiny, it must be set against near-complete resistance of many producers. Producers are also becoming scattered in southern Europe, with a large outbreak (>200 patients) of VIM-positive *P. aeruginosa* in Thessaloniki, Greece (7). In the United Kingdom (UK), the Antibiotic Resistance Monitoring and Reference Laboratory (ARMRL) has received its first few VIM-positive *P. aeruginosa* isolates. Microbiologists and public health doctors should appreciate the potential threat posed by such organisms.

VIM enzymes in England

During the past 18 months, ARMRL has received six VIM-producing *pseudomonas* from four hospitals in England. One isolate, from Kent, was received during surveillance of bacteraemia isolates commissioned by the British Society for Antimicrobial Chemotherapy (<http://www.bsacsurv.org>). The remainder were referred by hospital laboratories for investigation of resistance – three from two hospitals in London (one of these isolates was a *P. putida*, not *P. aeruginosa*) and two from Lancashire. All were positive for blaVIM by polymerase chain reaction (PCR). They were resistant to carbapenems, cephalosporins, ciprofloxacin and aminoglycosides (except, in one case, amikacin). All were susceptible to polymyxin and several also to aztreonam, a β -lactam that is stable to metallo- β -lactamases, but which may be compromised by other mechanisms that are frequent in *P. aeruginosa*, such as efflux and chromosomal β -lactamase over-production (8). Broad resistance to β -lactams correlates with the broad hydrolytic activity of the VIM enzymes; resistance to aminoglycosides with the fact that VIM enzymes are often encoded by integrons – natural recombination systems that capture and assemble multiple resistance genes behind a single promoter (the starting point for transcription) (1, 3).

Molecular fingerprinting at the Laboratory of Hospital Infection indicated that the two Lancashire isolates (from different patients in the same ward) were indistinguishable by pulsed-field gel electrophoresis of XbaI or SpeI digests of genomic DNA, implying cross-infection. The Kent isolate may also be related to one of those from London, although these were obtained nearly 18 months apart. The blaVIM genes are being sequenced at ARMRL (at least four VIM types occur, with VIM-2 predominant) and their transmissibility is being investigated. One London isolate was from a patient admitted from the United Arab Emirates; the others are still under epidemiological investigation, with review of case notes by the PHLS Communicable Disease Surveillance Centre to identify common factors and risks. The finding of VIM enzymes in *P. aeruginosa* follows the report, earlier this year, of an *Acinetobacter junii* with IMP-1 metallo- β -lactamase in Salford (9).

The present isolates were clearly resistant to imipenem and meropenem, but this behaviour is not universal among IMP and VIM producers. Many gene-positive isolates have been reported to lack phenotypic resistance, probably because resistance requires an impermeable host strain (10). This situation may be advantageous in militating against the rapid accumulation of resistance but is also a

concern, since it means that the blaVIM and blaIMP genes might easily spread undetected, with resistance only being becoming apparent once the strain undergoes a permeability mutation. Silent gene spread has been reported (eg, in some Taiwanese hospitals), where blaIMP and blaVIM genes have disseminated among *Enterobacter* and *Klebsiella* strains that remain ostensibly susceptible to imipenem and meropenem (11,12) but which would require only minor permeability changes to allow expression of resistance.

Action by microbiologists

Without genetic surveillance it is impossible to determine the true prevalence of blaVIM and blaIMP genes among UK isolates and, for the present, investigation is confined to isolates with phenotypic resistance – such as those described here. In the case of *P. aeruginosa*, most carbapenem resistance is due to impermeability or efflux and not to VIM and IMP carbapenemases. Such resistance is of lesser concern since it is never transmissible and since the impermeability is narrow-spectrum, sparing non-carbapenem drugs (13, 14). ARMRL therefore advises diagnostic laboratories to test carbapenem-resistant *P. aeruginosa* isolates with Etest metallo- β -lactamase detection strips (AB Biodisk, Solna, Sweden/Cambridge Diagnostic Services, Cambridge, UK). These contain gradients of imipenem with and without EDTA. Where imipenem is potentiated >8-fold by the EDTA, metallo- β -lactamase production is inferred and the isolate should be sent to ARMRL for investigation. In the case of Enterobacteriaceae and *Acinetobacter* ARMRL is happy to examine all carbapenem-resistant isolates for IMP and VIM genes.

Many infection-control teams screen for antimicrobial-resistant organisms among patients with a recent history of admission to hospitals abroad. This has been particularly pertinent for MRSA but relevant also for other organisms such as multiresistant *Klebsiella spp* (15). Currently it would appear that patients transferred from middle and far east might be a particular source of metallo- β -lactamase producers, but the problem may become more widespread, and a simpler policy of screening all international patient transfers might be preferable. The epidemiology of the current VIM cases awaits clarification, and some do not appear to have had a history of hospitalisation abroad; however index cases who had been abroad may have been missed. By analogy, one outbreak of cephalosporin- and aminoglycoside-resistant *Klebsiella* (15) originated from a patient recently hospitalised in Bahrain and rapidly transferred to several patients on intensive care units in two hospitals before the index case had been identified.

Admitted patients should have a risk assessment, including a review of their history (eg, units attended, care received, antimicrobials prescribed), country's history of antimicrobial resistance, number and type of close contact procedures now required (eg, central venous catheter care) and the risk category of the unit receiving the patient. Certain high-risk units (eg, intensive care units), may wish to screen all admitted patients, as their experience is that cases with unusual histories may be missed (15,16). If multiresistant carbapenemase producers are detected, the situation is serious and, ideally, isolation in a side-room is required. If more than one patient is involved, cohort or targeted nursing in a side room or bay would also be acceptable. Hand hygiene (including alcoholic preparations) should be reinforced. The infection control team may also wish to consider weekly and discharge screening of other patients on an affected ward to confirm that control measures have been successful. Since environmental reservoirs, such as shared equipment, can be relevant to the spread of multiresistant opportunist pathogens (16) practices and decontamination procedures should be audited, reviewed and backed-up by regular educational programmes (16).

Aside from the immediate treatment problem the longer-term concern must be the lack of new agents that might be used against the multiresistant gram-negative organism that can carry VIM and IMP enzymes. Most antibiotic development is presently concentrated versus gram-positive pathogens such as MRSA rather than opportunistic non-fermenters such as *P. aeruginosa*. For further information, contact David Livermore, ARMRL, CPHL; email: DLivermore@phls.nhs.uk.

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14. Livermore DM. Multiple mechanisms of antimicrobial resistance in *Pseudomonas aeruginosa*: our worst nightmare? *Clin Infect Dis* 2002; **34**: 634-40.
15. Cookson B, Johnson AP, Azadian B, Paul J *et al.* International inter and intra-hospital patient spread of a multiple antibiotic-resistant strain of *Klebsiella pneumoniae*. *J Infect Dis* 1995; **171**: 511-3.
16. MacRae MB, Shannon KP, Rayner DM, Kaiser AM, Hoffman PN, French GL. A simultaneous outbreak on a neonatal unit of two strains of multiply antibiotic resistant *Klebsiella pneumoniae* controllable only by ward closure. *J Hosp Infect* 2001; **49**(3): 183-92.

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General outbreaks of foodborne illness, England and Wales: weeks 41-44/02*

Health authority	Organism	Place of outbreak	Month of outbreak	No. ill	Cases positive	Suspect vehicle	Evidence
Lambeth Southwark & Lewisham	S. Enteritidis PT 6A	Hospital	September	13	13	Eggs	M
Sefton	S. Enteritidis PT8	Residential	September	8	2	None	–
Liverpool	S. Enteritidis PT 1	Retailer	October	85	51	Home made mayonnaise	D
Leicestershire	S. Braenderup	Caterer	October	20	2	Curry powder	M
Suffolk	S. Enteritidis PT 4	Reception	October	20	3	Chocolate mousse	D
North Essex	S. Enteritidis PT3	Restaurant	October	5	5	None	–
Suffolk	S. Enteritidis PT 4	Restaurant	October	2	2	None	–
East Sussex	S. Enteritidis PT 6	Reception	September	10	#nk	None	–

* Preliminary data. Final information will be published in the quarterly report.

not known

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; S (statistical): a significant statistical association between consumption of the suspect vehicle(s) and being a case; D (descriptive): other evidence, usually descriptive, reported by local investigators as indicating the suspect vehicle

Salmonella infections: England and Wales, reports to the PHLS (salmonella data set*)

Salmonella infections: England and Wales, reports to the PHLS (salmonella data set*) September 2002

Details of serotypes of the 2203 infections recorded in September are given in the adjacent table. In October 2002, 2013 infections were recorded and preliminary information was received for about eight outbreaks (see table above).

	September 2002
Salmonella (provisional data)	2203
S. Enteritidis (PT4)	590
S. Enteritidis (other PTs)	1029
S. Typhimurium	187
S. Virchow	21
Other (typed)	376

Common gastrointestinal infections, England and Wales: laboratory reports, weeks 40-44/02

	Number of reports received					Total reports	Cumulative total to	
	40/02	41/02	42/02	43/02	44/02	40-44/02	44/02	44/01
Laboratory reports								
<i>Campylobacter</i>	819	1025	1395	542	950	4731	37,965	48,013
<i>Escherichia coli</i> O157*	6	19	15	17	3	60	498	691
Salmonella†	535	594	532	499	441	2601	12,431	14,359
<i>Shigella sonnei</i>	11	13	33	5	22	84	619	767
Rotavirus	19	22	56	19	64	180	13,927	15,742
Norwalk-like virus	30	51	218	28	121	448	2807	1414
<i>Cryptosporidium</i>	47	75	107	63	83	375	2385	2828
<i>Giardia</i>	61	72	115	46	79	379	2714	2871

* Vero cytotoxin producing isolates (data from LEP)

† Data from PHLS LEP

Cyclospora cayetanensis in patients aboard cruise ships: issues of investigation and laboratory diagnosis

During summer 2002, three cases of illness with the protozoan parasite *Cyclospora cayetanensis* were identified in patients returning from the same Caribbean cruise. One of the cases had been originally identified in the primary testing laboratory as *Cryptosporidium* and was referred to the Cryptosporidium Reference Unit (CRU) for confirmation. Differential diagnosis is important, not only for surveillance purposes but also because, unlike *Cryptosporidium*, treatment is available for *C. cayetanensis*: trimethoprim sulphamethoxazole provides rapid, effective anti-parasitic treatment (1). Cyclosporiasis is characterised by diarrhoea, often of abrupt onset. Stools are frequent and sometimes explosive, and other symptoms include anorexia, nausea, vomiting, abdominal bloating and cramps, weight loss, fatigue, low

grade fever, and body aches. Although self-limiting in immunocompetent patients, illness is often prolonged, and the duration of diarrhoea averages five days to 15 weeks in untreated patients (2). Symptoms can be relapsing-remitting, including alternate diarrhoea and constipation (3).

C. cayetanensis can be hard to identify since the oocysts are often unsporulated in relatively fresh stools and may appear to provide little in the way of diagnostic features. Those that can be of help in diagnosis have been well described (4) and good diagnostic pictures have been published (5). Laboratories may find these helpful since *C. cayetanensis* is rare in the UK. Differentiation from *Cryptosporidium* can be facilitated by the use of a calibrated eyepiece graticule to measure presumptive oocysts. Laboratories should also inspect verified positive control material, which admittedly is more readily available for *Cryptosporidium* than *C. cayetanensis*. Additionally, the CRU is happy to receive presumptive isolates, clearly labelled as such, for timely confirmation.

During the incident mentioned above, liaison between the CRU and the port health authority environmental health officers highlighted many concerns not only about *C. cayetanensis* and cruise ships but also about the increasing possibility of imported infection due to an increasing variety of vulnerable foods being imported from potentially risky sources. Although risks to the UK have been evaluated (6), it appears that they may be increasing as the food supply becomes increasingly global. Widespread outbreaks were reported in North America during the 1990's (7) and an outbreak was reported, in Germany in 2000 (8).

In order to provide better tools for evaluation of risk, the CRU is evaluating traditional microscopy-based methods and more recently developed PCR-based tools for the detection and differentiation of *C. cayetanensis* from a variety of sample matrices. This is in collaboration with the Wessex Environmental Microbiology Service and CREH Analytical Limited. In order to support this work, the CRU welcomes any isolates, which should be sent, with relevant patient details to: Cryptosporidium Reference Unit, Swansea PHL, Singleton Hospital, Swansea SA2 8QA; DX 6070300 Swansea 90 SA.

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Typhoid and paratyphoid, England and Wales: laboratory reports, July to September 2002

Organism and Phage type	Number of cases	Infection acquired abroad			Excreters and carriers
		Yes	No	Not Reported	
S.Typhi					
A	2	1	–	1	–
B2	2	2	–	–	–
C1	1	1	–	–	–

D1	1	-	-	1	-
D1-N	1	1	-	6	-
E1	15	9	-	-	-
E2	1	1	-	1	-
E3	1	-	-	1	-
M1	1	-	-	-	-
N	1	1	-	-	-
O	1	1	-	1	-
Untypable	4	3	-	1	-
Untypable Vi-1	3	2	-	1	-
Untypable Vi-2	2	1	-	-	-
Vi-negative	1	1	-	1	-
46	5	4	-	1	-
S.Paratyphi A					
1	12	8	-	4	-
1A	3	2	-	1	-
2	1	1	-	-	-
4	8	5	-	3	--
13	6	6	-	1	--
RDNC	2	1	-	1	-
S.Paratyphi B					
Taunton	1	-	-	1	-

Forty-two cases of *Salmonella* Typhi infection were reported in the third quarter of 2002. Twenty-eight cases were infected abroad (Indian subcontinent 20, Nigeria 2, Afghanistan 1, Ghana 1, Greece 1, Malawi 1, Philippines 1, and abroad (unknown location) 1). In fourteen cases the country of infection was not stated.

Thirty-two cases of *S. Paratyphi A* infection were reported. Twenty-three cases were infected abroad (Indian subcontinent 21 and Nigeria 2). In nine cases the country of infection was not stated.

One case of *S. Paratyphi B* was reported and the country of infection was not stated.



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Daan Mulder Memorial Symposium

The Daan Mulder memorial symposium will take place at the London School of Hygiene and Tropical Medicine on Friday 13 December 2002. The title of the symposium is *The current status of knowledge on the epidemiology, natural history, and control of tuberculosis*. This is the fifth symposium. The attendance fee is £20, which covers lunch and refreshments. For further information contact Glenda Young, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT, tel: 020 7927 2909; fax: 020 7927 2666; email Glenda.young@lshtm.ac.uk.



VTEC 2003

VTEC 2003 will be held at the Edinburgh International Conference Centre from 8 to 11 June 2003. The conference will feature research from around the world, building on the past two conferences in Kyoto, Japan in 2000, and Baltimore US in 1997. The main topic headings are the food chain, epidemiology, the biology of VTEC, and clinical aspects. The call for papers has been issued – **the deadline for the submission of abstracts is 20 December 2002**. The conference fee, if paid before 31 March, is £375, thereafter it is £450. This does not include accommodation. For further details can be obtained from VTEC Conference organisers c/o In Conference Ltd, 10b Broughton Street Lane, Edinburgh EH1 3LY, tel: +44 (0) 131 556 9245, fax: +44 (0) 131 556 9638, email: VTEC@in-conference.org.uk, website: <http://www.VTEC2003.com>.
