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Patient dies of rabies acquired in the Philippines

A man died of rabies at a London hospital this week. He had been bitten during a fight between two dogs in the Philippines six weeks before becoming unwell, and had not sought medical attention following the bite. Both of the dogs involved in the fight had died, alerting the patient to the possibility that he might be at risk of rabies. His initial symptoms were non-specific, progressing to aching and numbness at the site of the wound, difficulty in swallowing and spasms of the face and throat. Although there are no known cases of person to person transmission of rabies other than through corneal grafts, health care staff and other close contacts of the patient have been offered rabies prophylaxis as a precaution.

Rabies is found in animals in many countries, but the United Kingdom (UK) has been entirely free of rabies since the early 1920s. The last human case of rabies acquired in the UK was in 1902. Between 1976, when rabies became a statutorily notifiable disease, and 2000, nine deaths from rabies were reported, seven of which were notified. The death from rabies of one additional patient in 1996 was neither notified nor certified. All of these rabies infections were acquired abroad.

Travellers to rabies endemic countries should be advised to avoid unnecessary contact with animals. If they are bitten or scratched by any warm-blooded animal (including bats) they should wash the wound thoroughly and seek medical attention immediately. Post-exposure prophylaxis is extremely effective in preventing rabies if given immediately. Once clinical signs of rabies develop, however, death is almost inevitable. Recommended post-exposure prophylaxis for high risk exposures is rabies-specific immunoglobulin and rabies vaccine to be given immediately, followed by a further four doses of vaccine 3, 7, 14, and 30 days later. A course of vaccine alone (5 doses) may be recommended for low risk exposures. Individuals who have been immunised in the previous two years should receive vaccine boosters. Both immunoglobulin and vaccine for post-exposure prophylaxis are available from the PHLS.

Certain occupational groups are eligible for pre-exposure immunisation and this is also warranted for travellers who will be living in rabies endemic countries or unable to get immediate medical attention if bitten while travelling, for example those on long trekking holidays in resource poor countries. Further details are available in the *Memorandum on rabies* available at www.doh.gov.uk/memorandumonrabies/.

Meningococcal disease associated with Hajj – update

The outbreak of W135 meningococcal disease among Hajj pilgrims and their contacts (1,2) is continuing, although at a lower rate than in the early weeks following Hajj 2001. From the start of Hajj (4 March 2001) up to 9 May a total of 41 cases of invasive W135 disease with a (sub)type compatible with last year's outbreak strain (2a P1.2,1.5) have been reported in the United Kingdom (UK). In 29 of these the strain's (sub)type was confirmed as 2a P1.2,1.5. Eight of the cases have been in Hajj pilgrims, 19 had contact with a pilgrim, and for ten no Hajj association could be identified (for seven cases information is not yet available). The median age of the pilgrim cases is 40 years, compared to 2 years amongst the other cases. A high case fatality rate of 27% (11 cases died, including six children aged less than 14 years) has been observed. Most cases have been reported from the north of England and London, which may reflect the distribution of Muslim populations in the UK. No cases have been reported from Wales, Scotland, or Northern Ireland. The attack rate amongst pilgrims this year is 38 per 100,000, similar to the rate following last year's Hajj (35 per 100,000), despite the change in vaccination guidelines to recommend quadrivalent (QV) vaccine for UK Hajj (and Umrah) pilgrims (3,4). This is likely to relate to QV vaccine coverage estimated to be below 47%. No vaccine failures have been reported yet. Most pilgrim contacts of non-pilgrim cases for whom information on vaccination status was available, were vaccinated with A/C vaccine in 2001 - none were vaccinated with the recommended QV vaccine.

Sustained transmission of the outbreak strain, mainly, but not exclusively amongst Muslims is expected, based on the pattern following last year's outbreak (3). To improve outcome in suspected meningococcal disease start of treatment in primary care with intra venous benzylpenicilline is

recommended (5). To enhance early recognition, health care workers and others should be vigilant of ongoing increased risk of disease in the Muslim population.

The PHLS Communicable Disease Surveillance Centre will continue to monitor invasive W135 meningococcal disease, and would like to be informed of new cases. Data should be collected on the [standard reporting form](#) (fax 020 8200 7868; email to Mary Ramsay, mramsay@phls.org.uk or Susan Hahné, susan.hahne@phls.wales.nhs.uk).

1. CDSC. Meningococcal infection in pilgrims returning from Hajj. *Commun Dis Rep CDR Wkly* [serial online] 2001 [cited 9 May 2001]; **11** (12): news. Available from <www.phls.co.uk/publications/CDR%20Weekly/archive/news1201.html#meningococcal>

2. CDSC. Meningococcal infection associated with Hajj 2001 - update. *Commun Dis Rep CDR Wkly* [serial online] 2001 [cited 9 May 2001]; **11** (14): news. Available from <www.phls.co.uk/publications/CDR%20Weekly/archive/news1401.html#meningococcal>

3. CDSC Meningococcal infection and the Hajj. *Commun Dis Rep CDR Wkly* [serial online] 2001 [cited 9 May 2001]; **11** (2): news. Available from <www.phls.co.uk/publications/CDR%20Weekly/archive/news0201.html#hajj>

4. Department of Health, United Kingdom. Immunisation for pilgrims travelling to Saudi Arabia for Hajj or Umrah. *CMO's urgent communication Cem/Cmo/2001/03*. [cited 9 May 2001] Available from <www.doh.gov.uk/cmo/cmo01_03.htm>

5. CDSC. Control of meningococcal disease: Guidance for consultants in communicable disease control. *Commun Dis Rep CDR Review* 1995; **5** (13):189-95.

Antiretroviral drugs and HIV

With an increasing number of HIV infected people on antiretroviral drugs in the United Kingdom (UK) the issue of emerging anti-retroviral resistance is potentially of increasing interest. In a short report in last week's British Medical Journal the UK Collaborating Group on Monitoring the Transmission of HIV Drug Resistance reported an increase in the prevalence of genetic markers associated with HIV-1 drug resistance in people newly infected with the virus since 1994 (1). In the UK, the prevalence of at least some level of drug resistance in primary infections rose to over 20% in the year 2000. This was predominantly amongst men who have sex with men (1). Some caution is needed in coming to firm conclusions. The sample size was small and the rise does not quite meet conventional statistical significance. Also, as an accompanying leading article points out, antiviral resistance for HIV is not entirely straightforward (2). Genotypic markers do not entirely equal actual resistance and it is not clear how competent these resistant viruses are at replicating – without selection pressure from therapy they may be overwhelmed by drug susceptible viruses (2). A rising prevalence of gene markers among new infections is not, however, something that can be viewed with any degree of complacency. Overall the lesson from United States and Europe, where antiviral therapies for HIV have been available for more than ten years, is that emergence of drug resistant virus variants in those receiving therapy with virological failure is common. Since some of these variants are cross-resistant to more than one drug (of the 15 or so currently available drugs) this may severely limit subsequent treatment options for such individuals. There is also the increasing evidence that HIV drug resistant strains can be transmitted (1,3), with the possibility that subsequent first line therapy may be compromised.

There is another reason for concern about HIV-1 drug resistance in the future. Antiretrovirals are increasingly being used in resource poor countries. The welcome resolution of a court case in South Africa has overcome patent restrictions and should now allow greater availability of antiretrovirals (and other medicines) in South Africa through generic substitution for many drugs (not just those concerned with HIV), greater competition in public procurement of drugs, improved quality of drugs, and more rational use of medicines (4).

Expanding access to essential medicines in resource poor settings – including drugs that tackle HIV disease – requires three critical elements. Firstly, lower prices for medicines but hopefully without prejudicing drug investment by the pharmaceutical industry, especially in drugs most applicable to diseases that predominate in countries poor in resources (such as malaria, tuberculosis (TB), schistosomiasis). Secondly, funding for essential medicines needs to be substantially increased. This means more domestic funding as well as more international monies from bilateral and multilateral donors. For HIV/AIDS, it is essential that any additional funding increases spending on prevention activities as well as on improved care and treatment. Thirdly, commitments and actions to build reliable healthcare and drug supply, delivery, and control systems are needed.

This last element is crucial. Successful examples of effective healthcare programmes and reliable supply systems do exist in South Africa and some other parts of Africa. A good model is provided by some national essential drug programmes and TB control programmes such as those in Tanzania and Uganda. These are, however, exceptions and there are good reasons for believing that many antiretrovirals will be used outside controlled systems. Perhaps resulting in increasing levels of drug resistance in HIV infections acquired in Africa.

These potential developments are of growing relevance to Europe including the UK. The number of heterosexually acquired infections diagnosed in Europe but reported as being acquired abroad in a high prevalence country has increased in recent years (5). Thus may lead to increased levels of antiviral resistant HIV infections appearing in Europe. With increasing numbers of heterosexually acquired infections within the UK, and a significant proportion of these being acquired in Africa, it is possible that imported drug resistant variants will also spread within the community (6). The inclusion of resistance testing within the UK Register of HIV Seroconverters study is an essential component of ongoing surveillance, and the EU-funded CASCADE initiative aims to co-ordinate such data from

throughout Europe. This approach should also be extended to other HIV surveillance programmes, and linked to virus subtype information, which will allow an assessment of the population dynamics of spread of resistance, particularly with regard to imported infections.

There is, therefore, an increasing need to ensure that antiviral therapies instituted in the developing world are prescribed appropriately, and that surveillance for emergence of drug resistance in all treated (and untreated) populations is undertaken both here and abroad.

1. UK Collaborative Group on Transmission of HIV resistance in Primary Infections. Analysis of prevalence of HIV-1 drug resistance in primary infections in the United Kingdom. *BMJ* 2001; **322**: 1087-8.

2. Little SJ. Is transmitted drug resistance in HIV on the rise? *BMJ* 2001; **322**: 1074-5.

3. Yerly S, Kaiser L, Race E, Bru J-P, Clavel F, Perrin L. Transmission of antiretroviral-drug-resistant HIV-1 variants. *Lancet* 1999; **354**: 729-33.

4. World Health Organization. WHO statement on outcome of South African court case. Statement WHO/08, 19 April 2001. Available online at <www.who.int/inf-pr-2001/en/state2001-08.html>.

5. European Centre for the Epidemiological Monitoring of AIDS. *HIV/AIDS surveillance in Europe. Mid-year report 2000*. No. 63. 2000. Saint Maurice: Institute de Veille Sanitaire, 2000. Available online at <www.ceses.org/AidsSurv/rapport_n63_2000/euro_hiv63full.pdf>

6. CDSC. AIDS and HIV Infections in the UK. *Commun Dis Rep CDR Wkly* [serial online] 2001 [cited 11 May 2001] : **11** (17): HIV/STIs. Available online at <www.phls.co.uk/publications/CDR%20Weekly/pages/hiv.html#AIDS>

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General outbreaks of foodborne illness, England and Wales: laboratory reports, weeks 15-18/01

Health authority	Organism	Place of outbreak	Month of outbreak	Number ill	Cases positive	Suspect vehicle	Evidence
Hull	<i>Salmonella typhimurium</i> DT104	Private dwelling	April	2	2	None	-
Herefordshire	SRSV	Public house	March	38	1	Various	D

D (descriptive): other evidence, usually descriptive, reported by local investigations as indicating the suspect vehicle;

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

Details of serotypes of the 650 salmonella infections recorded in March 2001 are given in the table below. In April 2001, 660 salmonella infections were recorded and preliminary information was received about one outbreak (see table above).

* figures quoted from the PHLS salmonella data set are for isolates confirmed and typed by PHLS Laboratory of Enteric Pathogens (LEP)

	March 2001
Salmonella (provisional total)	650
S. enteritidis (PT4)	137
S. enteritidis (other PTs)	196
S. typhimurium	97
S. virchow	28
Other (typed)	192

Common gastrointestinal infections, England and Wales: laboratory reports, weeks 15-18/01

	Number of reports received				Total reports 15-18/01	Cumulative reports	
	15/01	16/01	17/01	18/01		2001	2000
Laboratory reports							
<i>Campylobacter</i>	516	401	942	933	2792	15504	14034
<i>Escherichia coli</i> O157*	4	11	7	3	25	101	104
<i>Shigella sonnei</i>	7	5	17	32	61	261	202
Rotavirus	544	548	697	788	2577	8657	11388
SRSV	13	127	73	23	236	753	955
<i>Cryptosporidium</i>	14	19	42	56	131	910	1075
<i>Giardia</i>	39	18	37	92	186	1047	1209

* Vero cytotoxin producing isolates (data from LEP)

Typhoid and paratyphoid, England and Wales: laboratory reports, January to March 2001

Organism and phage type*	Number of cases	Infection acquired abroad			Excretors (E) and carriers (C)
		Yes	No	Not reported	
S. typhi					
A	3	2	–	1	–
E1	19	14	–	5	–
O	2	1	–	1	–
Untypable	2	2	–	–	–
Other PTs**	9	4	–	5	–
Total	35	23	–	12	–
S. paratyphi A					
1	17	11	–	6	–
3	2	1	–	1	–
4	6	5	–	1	–
13	8	5	–	3	–
Other PTs#	2	1	–	1	–
Total	35	23	–	12	–

* all isolates were confirmed and phage typed by LEP; ** C1, C10, D2, E7, T, 40, Degraded, Untypable Vi-1, Vi negative, 51 (one each); # 1A, RDNC (one each)

Thirty-four cases of *Salmonella typhi* infection were reported in the first quarter of 2001. Twenty-two cases were known to have been infected abroad (Indian subcontinent 20, Kenya 1, Nigeria 1). One patient had two phage types (T/Degraded) isolated from stool.

Thirty-five cases of *S. paratyphi A* infection were reported. Twenty-three cases were known to have been infected abroad (Indian subcontinent 22, not named 1). In 12 cases the country of infection was not stated.

Food poisoning caused by shellfish contaminated by marine algal toxins

There are some 4000 species of marine algae worldwide and about 2% produce toxins. Toxins are generally produced in the warmer summer months and are then concentrated in filter feeding bivalves such as mussels and scallops. Concentration up the food chain can occur in gastropods, crustacea, and fish. Consumption of contaminated shellfish and fish can result in illness. The major shellfish poisoning syndromes are diarrhetic shellfish poisoning (DSP), paralytic shellfish poisoning (PSP), amnesic shellfish poisoning (ASP), and neurotoxic shellfish poisoning (NSP). Cases of DSP and PSP have been reported in the United Kingdom (UK). The symptoms of the shellfish poisonings include gastrointestinal disease, but high levels of some toxins also cause neurological symptoms (box). High levels of ASP produce permanent loss of short term memory and death, PSP paralysis and death, and NSP convulsions.

Box Symptoms of food poisoning caused by shellfish contaminated by marine algal toxins

Amnesic shellfish poisoning	Onset: 15 mins to ~38 hrs	Duration: few days
Low levels of toxins: diarrhoea, abdominal cramps, nausea, vomiting, headache		
High levels of toxins: diarrhoea, abdominal cramps, nausea, vomiting, headache, disorientation, permanent loss of short term memory, death		
Diarrhetic shellfish poisoning	Onset: few mins to few hrs	Duration: 1 to 3 days
Diarrhoea, abdominal cramps, nausea, vomiting, chills		
Paralytic shellfish poisoning	Onset: ~15 mins	Duration: up to 3 days
Low levels of toxins: nausea, vomiting, parasthesia in lips and extremities, muscle weakness		
High levels of toxins: nausea, vomiting, parasthesia in lips and extremities, paralysis, death		
Neurotoxic shellfish poisoning	Onset: ~1 hr	Duration: 8 hrs to 3 days
Vomiting, abdominal pain, nausea, fatigue, numbness in mouth and extremities		
Severe cases: seizures, breathlessness, tachycardia		
Azaspiracid	Onset: 12 to 24 hrs	Duration: 5 days+
Severe diarrhoea and vomiting, abdominal pain, vomiting		

One of the most recently discovered algal toxins is azaspiracid. The first reported outbreak of food poisoning caused by azaspiracid was in the Netherlands in 1995, and outbreaks occurred in Northern Ireland in 1997, and France and Italy in 1998. In all these incidents the causative food was mussels from the west coast of Ireland. Symptoms are severe diarrhoea and vomiting with abdominal pain and nausea. The onset is 12 to 24 hours after consumption, and symptoms persist for up to five days. Preliminary studies have shown that azaspiracid can cause necrosis in the intestine, thymus, and liver. The structure of azaspiracid was determined in 1998, and at least eight different forms have subsequently been identified, of differing toxicity.

At least five outbreaks of food poisoning caused by imported mussels contaminated by azaspiracid were investigated by the Food Safety Microbiology Laboratory (FSML) in the summer of 2000. Three separate incidents were reported in a restaurant in Sheffield between 22 July and 5 August. Other outbreaks were reported in Warrington, Aylesbury, and the Isle of Wight. The symptoms were severe diarrhoea and vomiting, with nausea and abdominal pain. The onset was 12 to 24 hours after consumption and the duration was at least one to two days. All cases had eaten imported frozen mussels harvested from two locations off the west coast of Ireland. These are the first reported cases of illness caused by azaspiracid in England and Wales.

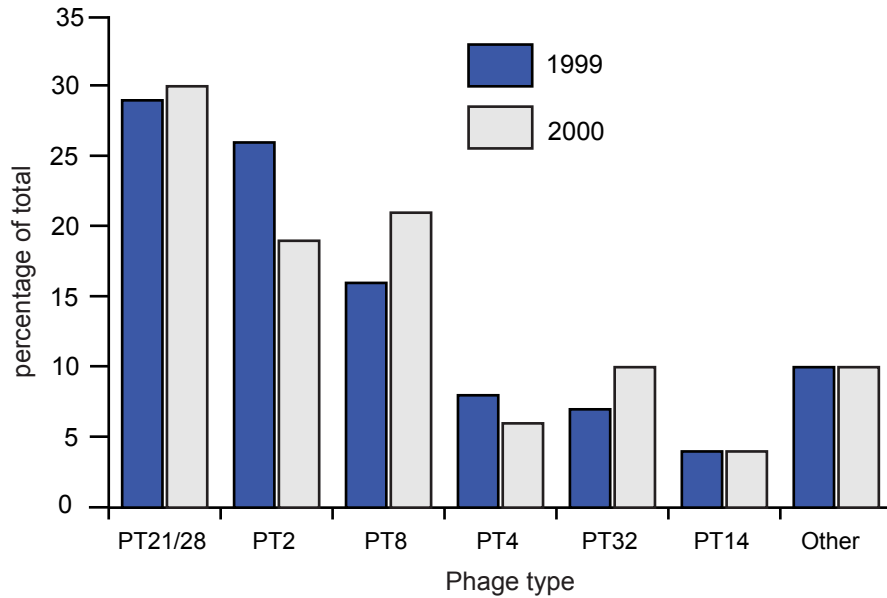
All the marine algal toxins are heat stable and toxicity is not removed by normal cooking or processing. There are no known antidotes so the only way to prevent human illness is to prevent toxic shellfish entering the food chain by monitoring shellfish in the environment. This is organised by the Food Standards Agency. ASP, DSP and PSP are regularly detected in UK shellfish. Azaspiracid was detected for the first time in shellfish off the southern coastline of England in the summer of 2000. Shellfish are usually contaminated by algal toxins in the warmer months but azaspiracid persists for long periods (up to eight months) and during winter.

A diagnosis of food poisoning is confirmed by the detection of the toxin in food samples. The FSML provides a service for testing shellfish and other foods for marine algal toxins. Samples should arrive chilled or frozen and be of at least 150g of flesh. For further information and advice please contact Moira Brett, Obi Mpamugo, or Vina Mithani, tel: 020 8200 4400 ext 4116.

Vero cytotoxin-producing *Escherichia coli* O157: 1999 and 2000

The number of isolations of Vero cytotoxin-producing *Escherichia coli* O157 (VTEC O157) confirmed by the PHLS Laboratory of Enteric Pathogens from human infections in England and Wales fell from 1084 in 1999 to 896 in 2000. The majority of strains in both years were VT2 (78% and 72% respectively); VT1+2 strains comprised 21% and 27% respectively and VT1 strains accounted for less than 1%. The strains belonged to 21 (1999) and 16 (2000) different phage types (PTs). The proportions of the six most common types are shown in the figure. PT21/28 was the most commonly reported type in both years, representing about 29% of isolates, having replaced PT2 as the most common type in 1999. There were at least ten general outbreaks caused by PT21/28 in 2000. Reports of PT2 have fallen steadily since 1996 (1) and in 2000 it was the third most common PT. Reports of PT8 increased in 2000, with it becoming the second most common type and contributing to the higher proportion of VT1+2 strains found.

Figure Predominant phage types of VTEC O157 from England and Wales: 1999 and 2000



Data from Scotland for 1999 and 2000 (2) show that the PTs most commonly reported were the same as those in England and Wales but the proportions were very different. For both years PT21/28 was reported twice as frequently in Scotland (57% and 67% respectively) but isolations of PT2 (9% and 7%) and PT8 (11% and 7%) were about half those in England and Wales. Both sets of data showed an increase in PT32 in 2000.

1. Vero cytotoxin-producing *Escherichia coli* O157: phage types reported in 1998. *Commun Dis Rep CDR Wkly* 1999; **9**: 33.
2. SCIEH. Gastrointestinal infections. *SCIEH Weekly Report* 2001; **35**: 118.

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