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CDR WEEKLY



NEWS

ENTERIC

RESPIRATORY

IMMUNISATION

HIV/STIs

BACTERAEMIA

ZOOSES

TRAVEL HEALTH

PRIMARY CARE

NEW

DIARY

BACK ISSUES

SEARCH

Main stories this week:

Vancomycin-resistant *Staphylococcus aureus* reported in the United States

Communicable Disease and Public Health published

Updated this week:

General outbreaks of foodborne illness, England and Wales: laboratory reports, weeks 23-26/02

Salmonella infections (faecal specimens), England and Wales: reports to the PHLS

Common gastrointestinal infections, England and Wales: laboratory reports, weeks 23-26/02

Less common gastrointestinal infections, England and Wales: laboratory reports, weeks 14-26/02

Up and coming 'new types' of Salmonella in England and Wales

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NEWS

ENTERIC

RESPIRATORY

IMMUNISATION

HIV/STIs

BACTERAEMIA

ZOOSES

TRAVEL HEALTH

PRIMARY CARE 

DIARY

BACK ISSUES

SEARCH

News

Last updated: 11 July 2002

Next update due: 18 July 2002

Contents

[Vancomycin-resistant *Staphylococcus aureus* reported in the United States](#)[Communicable Disease and Public Health published](#)[Top |](#)

Vancomycin-resistant *Staphylococcus aureus* reported in the United States

Since the first reports of glycopeptide-resistant enterococci (GRE) in 1987, concern has been expressed about enterococcal van genes, which encode vancomycin resistance, reaching methicillin-resistant *Staphylococcus aureus* (MRSA). This fear has now been realised, with the reported isolation of a vancomycin-resistant *S. aureus* (VRSA) (vancomycin minimum inhibitory concentration [MIC] >128 mg/L; teicoplanin MIC 32 mg/L) from the tip of a dialysis catheter and from a chronic foot ulcer of a patient in Michigan, in the United States (US). Glycopeptide-resistant *Enterococcus faecalis* (genotype not specified) was also isolated from the ulcer (1).

This high-level, *vanA*-mediated resistance contrasts with the low-level 'intermediate' vancomycin resistance recently seen in the United Kingdom (UK) (2). Isolates of *S. aureus* with intermediate vancomycin resistance (VISA) (MICs 8 mg/L), were first reported from Japan in 1997 (3) and about two dozen strains have been reported worldwide. Their resistance mechanism is uncertain, but involves a thickening of the cell wall, and is distinct from anything observed in GRE. The Michigan VRSA is the first clinical *S. aureus* isolate confirmed to be truly resistant to vancomycin by the American National Committee for Clinical Laboratory Standards (NCCLS) susceptibility testing criterion (MIC >16 mg/L) and, importantly, is the first clinical *S. aureus* isolate proven to have acquired enterococcal vancomycin resistance genes.

Transfer of van determinants from enterococci to *S. aureus* has long been predicted, with such a transfer (to a methicillin-susceptible *S. aureus* strain) having been demonstrated *in vitro* (4). Enterococci and staphylococci have several antimicrobial resistance genes in common, such as genes for high-level aminoglycoside resistance and β -lactamase production, suggesting that intergeneric exchange does occur. It is puzzling that transfer of enterococcal van genes to *S. aureus* has not occurred previously, or has never been detected, in the clinical situation. This may be due to the different ecological niches preferred by the species, although both may colonise the skin of hospital patients and must presumably 'meet' fairly often, or it may be that enterococcal plasmids or transposons carrying glycopeptide resistance are unable to transfer efficiently to, or be maintained stably in, staphylococci (5).

In enterococci, one type of vancomycin resistance (VanA), is conferred by a variety of complex, but related, genetic elements, which are believed to be derived from a progenitor transposon, Tn1546 (6). Whether the glycopeptide-resistant *E. faecalis* from the foot ulcer that yielded the Michigan VRSA also had the *vanA* genotype was not reported. If it did (as is implied in the report) (1), then it is important that further studies are undertaken to compare the *vanA* elements in the MRSA and the *E. faecalis*, to confirm whether direct exchange has occurred.

Enterococci have several means of exchanging genetic material, including broad host-range plasmids, narrow host-range plasmids, and conjugative transposons. One system merits particular mention since it affords strains of *E. faecalis* a highly efficient system for exchanging plasmids, including those determining antibiotic resistance. This is based on the production of short peptide 'sex' pheromones by recipient strains that cause donor cells carrying suitable 'responsive' plasmids to aggregate and form clumps with recipient cells, thereby bringing about the cell-to-cell contact necessary for conjugative

DNA transfer. These pheromones are not produced by most *E. faecium* strains, which are the main enterococcal host for vancomycin resistance, although rare strains of this species may carry responsive plasmids (5). A pheromone-like peptide, *staph-cAM373* is, however, produced by many *S. aureus* (7), and causes *E. faecalis* strains carrying appropriate responsive plasmids to clump (8). This provides a theoretical route by which *S. aureus* might acquire glycopeptide resistance from enterococci. Indeed, an *E. faecalis* strain carrying a *vanA* plasmid that was responsive to staphylococcal pheromone, *staph-cAM373*, was recently described (8).

It has long seemed inevitable that one of the forms of glycopeptide resistance seen in enterococci would emerge in MRSA, as has now happened. For once, the overused epithet 'superbug' seems appropriate. VRSA have a daunting combination of pathogenic potential and resistance. Despite this, the Michigan report, although alarming, will probably not generate the same degree of consternation as it might have less than five years ago. There are now several newly licensed anti gram-positive agents, including linezolid (an oxazolidinone), and quinupristin-dalfopristin (a streptogramin A/B combination). The latter two agents both have excellent inhibitory activity against almost all *S. aureus* strains (*ie* stop the strains growing and replicating), although neither actually kills the bacteria. The Michigan VRSA strain remained susceptible to these and to several 'old' antibiotics, including chloramphenicol, tetracyclines, and co-trimoxazole. At the time of reporting, the patient was clinically stable, and was responding – as an out-patient – to 'aggressive wound-care' and systemic co-trimoxazole (1). Nevertheless, it should be cautioned that resistance to new antibiotics can arise in MRSA, and also that not all MRSA strains have susceptibility to older agents; the EMRSA-17 strain seen in the UK, for example, is very multi-resistant.

Prompt recognition of vancomycin-resistant *S. aureus* is extremely important in the clinical laboratory. Quantitative confirmation of suspected resistance should be performed at the primary laboratory (*eg* by Etest), whenever possible, as rigorous infection control procedures must be implemented rapidly to prevent spread of the resistant strain. The strain should then be submitted to a reference laboratory for independent confirmation and, if appropriate, for detailed genotypic analysis. For further information contact Neil Woodford, Resistance Mechanisms Section, PHLS Antibiotic Resistance Monitoring and Reference Laboratory; tel 020 8200 4400 ext 4255; email: nwoodford@phls.org.uk.

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[Top](#) |

Communicable Disease and Public Health published

Communicable Disease and Public Health (CDPH), Volume 5, Number 2 has recently been published.

The first editorial in this issue refers to the projections for United Kingdom (UK) residents infected with

HIV up to 2005, the first article in the journal. There is now a year on year increase of more than 2000 in the number of people living with HIV. Other editorials discuss the bioterrorism threat of smallpox, and the implications of subsuming the Public Health Laboratory Service into a new Health Protection Agency with a wider remit. Scientific articles in the journal discuss intestinal infections associated with fresh fruit and vegetables, the surveillance of cryptosporidiosis, aspects of influenza vaccination and diagnosis, needle disposal by diabetics, scabies control in homes for the elderly, and the consumption of raw milk on farms. There are guidelines on the investigation of legionnaires' disease and the use by general practitioners of antibiotic sensitivity testing. Finally, a short review explains why there are as yet no laboratory diagnostic tests for vCJD infection applicable to the living as opposed to the dead. There is discussion of the PHLS 'Investors in People' initiative and an article on the management of work related stress.

CDPH, which is published quarterly, is a PHLS journal reflecting the breadth of activity by the public health and medical microbiology communities in UK. It is expected that publication will continue under the aegis of the Health Protection Agency in 2003.

[Back to top](#)

- NEWS
- ENTERIC
- RESPIRATORY
- IMMUNISATION
- HIV/STIs
- BACTERAEMIA
- ZOONOSES
- TRAVEL HEALTH
- PRIMARY CARE NEW
- DIARY
- BACK ISSUES
- SEARCH

Enteric

Last updated: 11 July 2002
Next update due: 15 August 2002

Contents

- [General outbreaks of foodborne illness, England and Wales: laboratory reports, weeks 23-26/02](#)
- [Salmonella infections \(faecal specimens\), England and Wales: reports to the PHLS](#)
- [Common gastrointestinal infections, England and Wales: laboratory reports, weeks 23-26/02](#)
- [Less common gastrointestinal infections, England and Wales: laboratory reports, weeks 14-26/02](#)

[Up and coming 'new types' of Salmonella in England and Wales](#)

[Next](#) | [Top](#) |

General outbreaks of foodborne illness, England and Wales: laboratory reports, weeks 23-26/02*

Health authority	Organism	Place of outbreak	Month of outbreak	No. ill	Cases positive	Suspect vehicle	Evidence
Camden and Islington	<i>Salmonella</i> Enteritidis PT4	Hotel	June	>7	>7	Scrambled eggs	D
North and Mid Hants	S. Enteritidis PT4	Restaurant	June	4	4	None	–
Camden and Islington	S. Enteritidis PT6	Hospital	June	2	2	None	–
Kent	S. Enteritidis PT6	Nursery	June	5	5	None	–
Warwickshire	S. Enteritidis PT8	Restaurant	June	>16	>16	None	–

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

[Next](#) | [Top](#) | [PDF](#) | [Archive](#)

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

Details of serotypes of the 819 salmonella infections recorded in May 2002 are given in the table below. In June 2002, 894 salmonella infections were recorded and preliminary information was received about five outbreaks.

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

	May 2002
Salmonella (provisional total)	819
S. Enteritidis (PT4)	171
S. Enteritidis (other PTs)	328
S. Typhimurium	102
S. Virchow	24
Other (typed)	194

[Next](#) | [Top](#) |

Common gastrointestinal infections, England and Wales: laboratory reports, weeks 23-26/02

Laboratory reports	Number of reports received				Total reports	Cumulative total to	
	23/02	24/02	25/02	26/02	23-26/02	26/02	26/01
Campylobacter	558	964	1729	1249	4500	20615	25379
Escherichia coli O157*	10	15	6	14	45	159	233
Salmonella	154	237	263	298	952	4279	5258
Shigella sonnei	6	12	13	39	70	340	454
Rotavirus	296	258	841	210	1605	12591	12804
Norwalk-like virus	49	129	58	50	286	1340	1057
Cryptosporidium	35	42	64	43	184	1238	1198
Giardia	28	43	81	75	227	1501	1520

* Vero cytotoxin producing isolates (data from LEP)

[Next](#) | [Top](#) |

Less common gastrointestinal infections, England and Wales: laboratory reports, weeks 14-26/02

Laboratory reports	Total reports	Cumulative reports	
	14-26/02*	26/02	26/01
Adenovirus	14	23	103
Astrovirus	51	69	92
Calicivirus	5	20	14
<i>Shigella boydii</i>	24	34	30
<i>Shigella dysenteriae</i>	10	17	18
<i>Shigella flexneri</i>	62	110	85
Aeromonas	34	59	74
Plesiomonas	4	8	15

Vibrio	18	29	22
Yersinia	10	18	16
<i>Entamoeba histolytica</i>	42	78	136
<i>Blastocystis hominis</i>	99	178	160
<i>Dientamoeba fragilis</i>	61	100	110
<i>Taenia</i> spp	15	28	39
<i>Trichostrongylus</i>	–	–	1
<i>Trichuris trichura</i>	47	79	41

* provisional data

[Next](#) | [Top](#) |

Up and coming ‘new types’ of Salmonella in England and Wales

Following the major epidemics of *Salmonella* Enteritidis infection and the emergence of the multiresistant strain of *S. Typhimurium* definitive phage type (DT) 104 during the past 20 years salmonella appears to have reached another crossroads.

In 2001, for the first time since 1982, the number of isolates of *S. Enteritidis* phage type (PT) 4 represented less than half of all the *S. Enteritidis* isolated from humans: 54% of more than 11,000 human isolates examined belonged to other *S. Enteritidis* phage types (1). Moreover, in addition to the established phage types such as PT1 and PT6, new types, such as *S. Enteritidis* PT44, which was first defined in 1998, are increasing in importance. PT44 has been linked to travel to the Canary Islands and infections have occurred following vacations on Gran Canaria, Tenerife, Lanzarote, and Fuerteventura islands. *S. Enteritidis* PT5c first made an impact in 2001 with over 350 cases reported in England and Wales (2). Twenty-eight per cent of those infected had travelled abroad, particularly to Tenerife. This phage type was responsible for three outbreaks in 2001 where egg mayonnaise was the vehicle of infection. The Veterinary Laboratories Agency (of England and Wales) has not seen this particular phage type in poultry (*R Davies, personal communication*) suggesting that the outbreaks have resulted from imported eggs. *S. Enteritidis* PT14b is also associated with foreign travel and was responsible for over 390 human infections in 2001. An interesting anaerogenic strain of PT14b was isolated from people returning from Greece and the Greek Islands in 2001 and this strain is continuing to cause infections today.

The multiresistant strain of *S. Typhimurium* DT104 has continued to evolve with new multiresistant strains being isolated. For example, DT12 resistant to ampicillin, chloramphenicol, streptomycin, sulphonamides, spectinomycin, tetracyclines, and trimethoprim was responsible for a small outbreak possibly due to infected milk. In addition the new provisional phage types of U302 and U310 have also evolved from DT104. When first isolated in 1997, U310 was resistant to ampicillin, chloramphenicol, streptomycin, sulphonamides, spectinomycin, and tetracyclines. The strains currently isolated are predominantly from pigs and are more commonly resistant to tetracyclines alone (3).

A disturbing new strain that is emerging is derived from *S. Paratyphi* B variant Java. This is resistant to a wide range of antibiotics – ampicillin, streptomycin, sulphonamides, trimethoprim, and furazolidone predominate. Only eight human cases of this type occurred in England and Wales in 2001, but the PHLS Laboratory of Enteric Pathogens reported on 41 isolates from chicken and chicken products.

In addition to the changes in salmonella types described above, changes in sources of infection have also occurred with foods such as lettuce (4), halva (5) and peanuts being implicated in recent outbreaks.

Clearly salmonella continues to play an important role in human disease and the changes both in the infecting organisms and the various sources of infection require continued vigilance to maintain our epidemiological awareness of this widespread pathogen.

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[Back to top](#)