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## Contents





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# Increase in Viral Meningitis, 2006

This summer there was a marked increase in the number of viral meningitis notifications made to HPSC. By 29th September 2006, 109 cases of viral meningitis had been reported. Most cases (60%) occurred in August. This peak exceeded that seen in the summer of 2001 (figure 1).

Enterovirus was isolated in 67% of cases, echovirus in 2%, and varicella species in 1%. In 30% of cases no specific virus was reported. Although nearly all age groups were affected, 94% of all cases were less than 35 years of age, with the highest number of cases reported in the 0-10 year age group (32%, n=35).

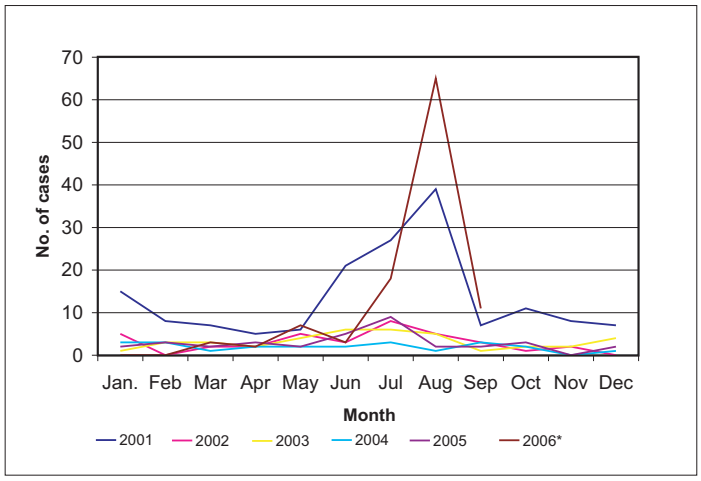


*Figure 1. Number of viral meningitis cases by month, 2001 to 29/09/2006**
*Data source: CIDR*

Crude incidence rates (CIR) varied across the country with the highest CIR reported in the HSE North West at 6.3/100,000 population (n=14), and the lowest in the HSE South at 0.9/100,000 (n=5).

Viral meningitis is relatively common. The typical presentation includes headache, fever, nausea or vomiting, and occasionally a rash. A history of a sore throat or diarrhoea may precede meningitis symptoms. The majority of infections cause mild or inapparent disease, but severe cases may be hospitalised for diagnosis and management of symptoms. There is no specific drug treatment for most cases of viral meningitis. Recovery is usually complete and rapid.

Enteroviruses (such as echovirus or coxsackie virus) are the most common cause of viral meningitis. Enteroviral diagnosis is confirmed by detection of virus in a faecal sample or by detection of viral nucleic acid in CSF by PCR testing. Since November 2005, the National Virus Reference Laboratory have been routinely doing PCR testing for enteroviruses on CSF samples.

In temperate climates such as Ireland, enterovirus meningitis occurs mainly in late summer, and is most common in preschool children. Transmission usually occurs through the faecal-oral route but may also occur via the respiratory route or contact with a contaminated environment. The incubation period is usually 3-6 days.

As enterovirus infection can spread readily from person to person via the faecal-oral route (virus shedding can continue for several weeks after infection) good personal hygiene is important. Emphasis is put on hand hygiene, particularly after using the toilet, before preparing or eating food, after contact with someone who has viral meningitis or a similar illness, and after changing or handling dirty nappies.

**Suzanne Cotter, Patricia Gravey, HPSC; Grainne Tuite, NVRL**

# New Patient Information Leaflets on the HPSC website

Two new patient information leaflets, produced by the Infection Control Sub-Committee of the SARI (Strategy for Control of Antimicrobial Resistance in Ireland) group are now available for download on the HPSC website at www.hpsc.ie

- **MRSA Information for Patients and Visitors**
- **Healthcare-associated Infection: Information for Public and Patients.**

These can be found in the A-Z section under 'MRSA/Factsheets/MRSA Information for Patients and Visitors.

# Salmonella in Ireland, 2005

## Introduction

Salmonellosis is one of the most common zoonotic diseases in humans. At present, over 2,460 serotypes of *Salmonella* have been identified. Two serotypes, however, *S. enterica* serotype Enteritidis and *S. enterica* serotype Typhimurium have accounted for the majority of cases of human salmonellosis in recent years.

Salmonellosis presents clinically as an acute enterocolitis, with sudden onset of headache, abdominal pain, diarrhoea, nausea and occasionally vomiting. Fever is almost always present. Dehydration, especially amongst vulnerable populations such as infants, the immunocompromised and the elderly, may be severe. *S.* Typhi and *S.* Paratyphi can cause enteric fever, a severe systemic life threatening condition, but this is very rare in Ireland and mainly travel-associated.

A wide range of domestic and wild animals, as well as humans can act as the reservoir for this pathogen. Prevention, surveillance and control of *Salmonella* infections is of major public health importance.

## Methods

The National Salmonella Reference Laboratory (NSRL) was established in 2000 in the Department of Medical Microbiology, University College Hospital, Galway. This laboratory accepts *S. enterica* isolates from all clinical and food laboratories for serotyping, phage typing and antimicrobial sensitivity testing.

This report reviews data available from the NSRL and weekly events of salmonellosis extracted from the CIDR (computerised infectious disease reporting system) system for the year 2005. These data enable us to provide an overview of the epidemiology and burden of disease caused by *Salmonella* infections in Ireland today.

## Results

### Demographic information

There were 357 clinical isolates of *S. enterica* referred to NSRL in 2005. The male:female ratio was 0.9:1. The highest number of cases was seen in children under five years of age.

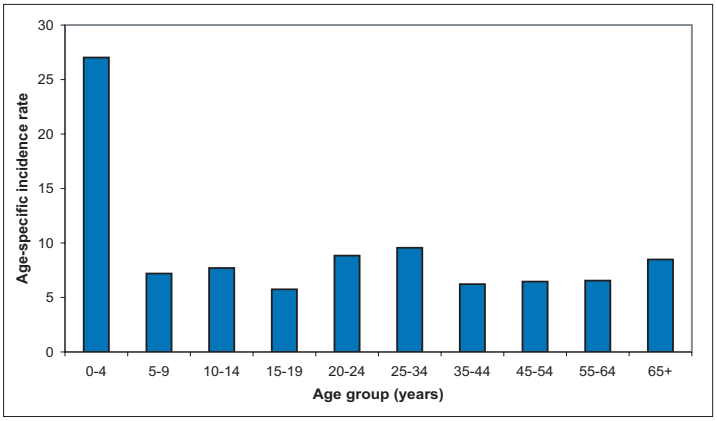


*Figure 1. Age-specific incidence rate of human salmonellosis in Ireland, 2005*

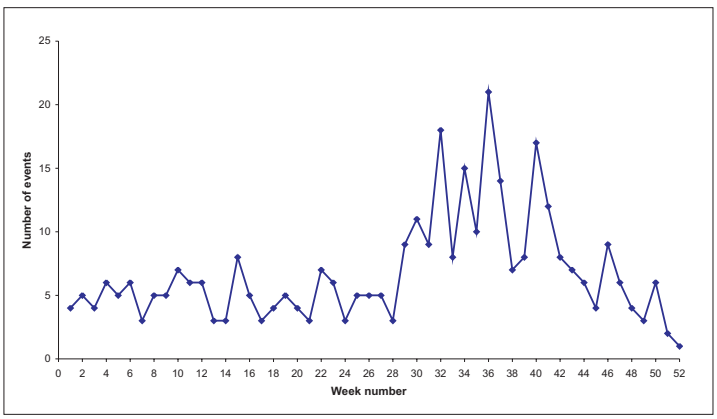


*Figure 2. Number of salmonellosis notifications by week, 2005 (data from CIDR*

*Table 1. Serotypes of S. enterica referred to NSRL, 2000-2005*

| | No. of isolates (%) | | | | | |
|---|---|---|---|---|---|---|
| Serotype | 2000 | 2001 | 2002 | 2003 | 2004 | 2005 |
| *S.* Enteritidis | 239 (36) | 248 (46) | 165 (40) | 205 (42) | 173 (41) | 145 (41) |
| *S.* Typhimurium | 284 (43) | 165 (30) | 140 (34) | 135 (28) | 125 (30) | 85 (24) |
| *S.* Agona | 6 (1) | 2 (0.4) | 5 (1) | 5 (1) | 2 (0.5) | 10 (3) |
| *S.* Virchow | 9 (1) | 16 (3) | 10 (2) | 10 (2) | 10 (2) | 9 (3) |
| *S.* Hadar | 11 (2) | 4 (1) | 6 (1) | 21 (4) | 4 (1) | 8 (2) |
| *S.* Dublin | 12 (2) | 12 (2) | 9 (2) | 5 (1) | 4 (1) | 5 (1) |
| *S.* Kentucky | 15 (2) | 4 (1) | 1 (0.2) | 10 (2) | 7 (2) | 4 (1) |
| *S.* Bredeney | 24 (4) | 11 (2) | 2 (0.5) | 3 (1) | 11 (3) | 3 (1) |
| All others | 65 (10) | 81 (15) | 78 (19) | 92 (19) | 83 (20) | 88 (25) |
| **Total** | **665** | **543** | **416** | **486** | **419** | **357** |

When age-specific incidence rates were calculated (figure 1), the burden of illness in this age group was even more evident.

### Seasonality

Analysis of the number of salmonellosis events notified to HPSC by week in 2005, revealed peaks in incidence from mid-August to early October (figure 2). Seasonal peaks are typically seen each year at this time.

### Serotyping, phage typing and antibiotic susceptibility results from NSRL

*Serotyping*

As has been the trend in recent years, the predominant serotype causing human illness in 2005 was *S.* Enteritidis (n=145), followed by *S.* Typhimurium (n=85). Table 1 depicts the changing shift in the more common serotypes in the past number of years. In 2005, after *S.* Enteritidis and *S.* Typhimurium, the next most commonly isolated serotypes were *S.* Agona (n=10), *S.* Virchow (n=9), *S.* Hadar (n=8) and *S.* Dublin (n=5). There were 5 cases of *S.* Typhi detected in 2005, the same number detected in 2004.

*Phage typing*

The predominant phage types of *S.* Typhimurium and *S.* Enteritidis are summarised in tables 2 and 3. The commonest phage type of *S.* Typhimurium reported in 2005 was DT104 (44%), followed by DT104b (15%). In previous years (1998-2003) PT4 was the predominant phage type of *S.* Enteritidis, but this trend changed in 2004 with PT1 replacing PT4 as the main phage type detected. This trend has continued in 2005 with PT1 accounting for 30% of the isolates followed by PT14b (15%), PT8 (14%), and PT4 (13%).

*Travel-association*

In 2005, 75 out of 357 isolates (21%) reported to NSRL were found to be associated with travel outside of Ireland. The most commonly reported countries were Spain (n=7), Nigeria (n=7), Thailand (n=6), Majorca (n=6) and Tunisia (n=5).

*Table 2. Phage types of S. Typhimurium in human isolates, 2005*

| Phage type | No. of isolates (%) |
|---|---|
| DT104 | 37 (44) |
| DT104b | 13 (15) |
| DT193 | 5 (6) |
| DT12 | 3 (4) |
| DT208 | 1 (1) |
| U310 | 1 (1) |
| Other | 12 (14) |
| No type | 13 (15) |
| **Total** | **85** |

*Antimicrobial resistance*

The antimicrobial susceptibility patterns of the most commonly isolated serotypes in 2005 are presented in table 4. Analysis of the 2005 AMR data again demonstrated high levels of resistance among *S.* Typhimurium, particularly DT104 isolates.

### Clinical notification data

There were 349 cases of salmonellosis notified to HPSC through the weekly notification system in 2005, giving a crude

*Table 3. Phage types of S. Enteritidis in human isolates, 2005*

| Phage type | No. of isolates (%) |
|---|---|
| PT1 | 44 (30) |
| PT14b | 22 (15) |
| PT8 | 20 (14) |
| PT4 | 19 (13) |
| PT21 | 12 (8) |
| PT24var | 4 (3) |
| PT6a | 3 (2) |
| PT6 | 3 (2) |
| Other | 13 (9) |
| No type | 5 (3) |
| **Total** | **145** |

incidence rate of 8.9 per 100,000 population (figure 3).

### Outbreaks

In 2005, there were 17 outbreaks of *S. enterica* notified to HPSC: three general and 14 family outbreaks affecting a total of 52 people. Three of the outbreaks were travel-related with Spain, Tunisia and the Czech Republic cited as the countries of infection. The largest outbreak was a community outbreak caused by *S.* Agona that affected six people and resulted in five hospitalisations.

### Discussion

*Salmonella enterica* continues to be an extremely significant cause of gastroenteritis in Ireland, despite a decrease in the rate of infections due to salmonellosis in 2005 (8.7/100,000) compared to 2004 (10.6/100,000). The highest incidence was reported in HSE South East. A similar incidence rate was reported in Northern Ireland (10.43/100,000) [CDSC, NI, August 2006, personal communication] while higher rates were seen in England and Wales[1] (22.7/100,000 provisional) and Scotland[2] (22.3/100,000).

As noted in previous years males and females were equally affected. All age-groups were affected but the highest incidence was noted in children less than five years of age. It is likely that more specimens are submitted for testing from this age-group so this should be borne in mind when interpreting these data.

Analysis of serotyping revealed that there were 44 different serotypes identified in 2005. *S.* Enteritidis and *S.* Typhimurium were the causative serotypes identified in 65% of cases. The proportion of cases attributable to *S.* Typhimurium continues to decrease with *S.* Enteritidis and other serotypes increasing in their relative importance. The emergence of more unusual serotypes could be attributable to the increase in the number of cases that are associated with foreign travel (21% in 2005). In addition, a significant number of travel-associated typhoid cases are reported each year. Five cases were reported in 2005, one associated with travel to India and two with travel to Pakistan. It is important that travellers are made aware of the measures that can be taken to reduce the risk of developing food-/water-borne illness whilst abroad and especially that typhoid vaccination is given when travelling to endemic countries.

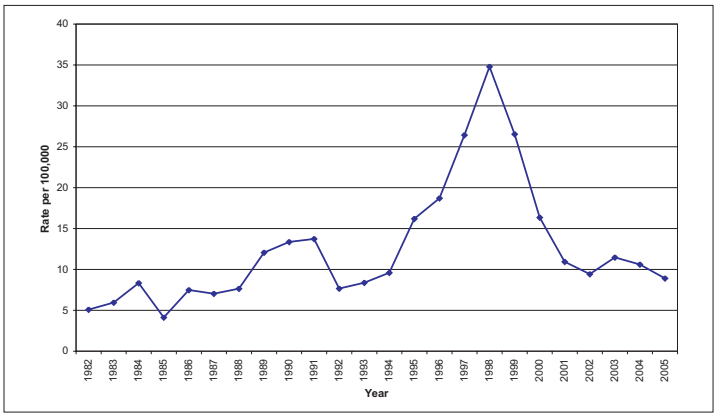


*Figure 3. Crude incidence rate of salmonellosis in Ireland per 100,000 population, 1982 - 2005.*

*Table 4. Antimicrobial susceptibilities of human Salmonella enterica serotypes isolated in Ireland, 2005*

| | % Resistance | | | | | | |
|---|---|---|---|---|---|---|---|
| Serotype (n) | Amp | Chl | Strep | Sulph | Tet | Trim | Nal |
| Enteritidis (145) | 5 | 0 | 0 | 2 | 3 | 2 | 30 |
| Typhimurium (85) | 73 | 64 | 72 | 75 | 79 | 8 | 9 |
| Agona (10) | 40 | 0 | 40 | 0 | 0 | 0 | 10 |
| Virchow (9) | 22 | 0 | 0 | 44 | 33 | 44 | 89 |
| Hadar (8) | 75 | 0 | 100 | 0 | 100 | 0 | 88 |
| Stanley (6) | 67 | 50 | 33 | 50 | 67 | 17 | 0 |
| Typhi (5) | 20 | 20 | 20 | 20 | 20 | 20 | 40 |
| Kentucky (4) | 25 | 0 | 25 | 50 | 50 | 0 | 50 |
| Bredeney (3) | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

*Amp = Ampicillin, Chl = Chloramphenicol, Strep = Streptomycin, Sulph = Sulphonamide, Tet = Tetracycline, Trim = Trimethoprim, Nal = Naladixic acid*

In previous years (1998-2003) PT4 was the predominant phage type of *S.* Enteritidis, but this trend has changed since 2004 with non-PT4 phage types being detected more and more. In particular, the incidence of PT14b and PT8 has increased in recent years, accounting for 3% and 5% respectively of the total phage types detected in 2003 and increasing to 15% and 14% respectively in 2005. This shift in phage type has been observed in many countries in Europe in recent years and could be attributable to factors such as increased travel, global food trade and animal vaccinations.[3 4]

The typing of all human salmonella cases by the NSRL continues to be an extremely powerful discriminatory tool particularly for cluster/outbreak detection and especially for the two most common serovars *S.* Enteritidis and *S.* Typhimurium. In addition, the submission of Irish data to international networks such as Enter-net allows the collation and analysis of serotyping, phage typing and AMR data across international borders and not only allows international outbreaks to be identified but also allows such emerging trends to be identified, monitored and explained.

In September 2005, the NSRL reported an unusual cluster of four human cases of *S.* Agona with a distinctive antibiogram (AS resistant). By November six cases had been identified, five from HSE SE and one with an epidemiological link to HSE SE. In addition the NSRL had identified a non-human (poultry) isolate from a poultry plant in the South East. Despite extensive epidemiological, environmental and microbiological investigations no common source was found. As no further cases were reported, the outbreak was declared over.

In October 2005, an international outbreak of *S.* Goldcoast infection in tourists returning from Majorca was identified by Health Protection Scotland. An alert through Enter-net and the European Commission's Early Warning and Response system (EWRS) led to an international response with active case finding. In total, 148 cases were identified in ten different countries – including six cases in Ireland. Despite extensive trawling, no testable hypothesis about foods, outlets, or other potential sources of infection could be generated. The outbreak was declared over on the 1st December 2005.

Finally analysis of the 2005 AMR data (antimicrobial resistance) of the various *Salmonella* serotypes demonstrated high levels of resistance among *S.* Typhimurium isolates, particularly DT104 isolates. The emergence of MDR (multi-drug resistant) *S.* Typhimurium and DT104 is well documented and constitutes an increasing global public health problem.[5]

**V Jackson, P McKeown, B Foley, HPSC; G Doran, M Cormican, NSRL**

**Acknowledgements**

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# Botulism

The Health Protection Surveillance Centre has recently been notified of a case of foodborne botulism. The case involved a non-national individual, resident in Ireland, who had returned here following a visit home and consumed a homemade pork pie that been improperly stored in his luggage in transit. Type A and B botulinum toxins were identified in the food samples.

On 1st January 2004, botulism became notifiable in Ireland. Prior to this, it was notifiable under the category of food poisoning. Cases of botulism are rare in Ireland. In 2002, there were two confirmed and one suspected case of wound botulism in injecting drug users.[1]

Botulism is more common in other parts of Europe such as Poland and France. The number of cases of foodborne botulism has fallen in Poland in recent years from 328 cases in 1990 to 93 cases in 1998. Most cases were associated with eating home-processed meat products (mainly pork).[2] In France there are around 20-30 cases reported each year with type B representing about 97% of cases.[3] In 2006, two cases of mild botulism were reported in France in drug users who inhaled cocaine.[4]

**Background**

Botulism is caused by the neurotoxin producing, anaerobic spore forming bacteria *Clostridium botulinum*. They are ubiquitous in nature being mainly found in soil, aquatic environments, and the intestinal tracts of animals and humans. The disease is rare but can be life-threatening. There are seven antigenic toxin types; A, B, C, D, E, F, and G. Toxins A, B, E, and rarely F, are the principal causes of botulism in humans.[5]

There are five categories of botulism[5]:

- Foodborne botulism caused by the ingestion of preformed toxin in contaminated food.
- Wound botulism which results from the production of toxin after growth of *C. botulinum* in an infected wound.
- Infant botulism where the infant ingests spores which germinate in the gut where the bacteria reproduce and produce toxin.
- Botulism due to intestinal colonisation in children and adults. This is thought to occur when the normal gut flora is altered by antibiotic therapy or surgical procedures.
- Aerosolised botulinum toxin used as a bioterrorism weapon.

The food traditionally implicated in botulism differs between countries and reflects the eating habits and food preservation methods of the countries concerned. The foods most likely to be implicated are lightly preserved foods such as fermented, salted or smoked fish or meat, and inadequately processed home canned or bottled foods, especially low-acid foods such as vegetables.[6] Outbreaks have been reported associated with home-preserved mushrooms, home-canned asparagus and home-preserved fish.[7 8 9] Infant botulism has been associated with the ingestion of honey and many countries recommend that infants <1year of age should not be given honey.[6] Commercial products are increasingly being implicated in outbreaks as methods of preserving food are changing. Fresh products that are vacuum-packed and refrigerated or heat-treated at inadequate temperatures facilitate the growth of *C. botulinum*.[8] A single case of foodborne botulism should be followed up urgently as it may be the start of a larger outbreak.

**Mode of action.**

The toxin is carried in the bloodstream from the site of infection to peripheral nerve endings where it binds to receptors and blocks the release of acetylcholine. This causes an acute flaccid paralysis affecting the face and head first, then spreading bilaterally down the rest of the body.[5]

**Clinical features**

Early symptoms include fatigue and weakness followed by blurred vision, dry mouth and difficulty swallowing and speaking. In foodborne cases vomiting and diarrhoea or constipation may occur. There is no fever or loss of consciousness. Death can result from respiratory failure. Botulism is underdiagnosed as many of the symptoms can be mistaken for more common clinical entities such as stroke, Myasthenia Gravis and Guillain-Barré syndrome. In infants the illness may have a gradual onset with constipation, followed by weakness, loss of appetite and loss of head control. Sudden death can also occur. Some studies have suggested that botulism may account for an estimated 5% of sudden infant death syndrome.[9]

The incubation period is 12-36 hours. The case fatality rate is approximately 5-10%. Mortality is high if treatment is delayed. Recovery can take months. Person-to-person transmission does not occur.[9]

**Laboratory diagnosis**

Serum and stool specimens, and epidemiologically implicated foods should be tested for botulinum toxin in suspected foodborne botulism. Stool specimens should be cultured for *C. botulinum*. Wound botulism is diagnosed by toxin in serum or by positive wound culture.[9]

**Treatment**

Antitoxin should be administered as soon as the diagnosis is made. **Antitoxin is available from Cherry Orchard Hospital**. Supportive treatment including mechanical ventilation may be required in severe cases and may be required for months. Surgical debridement and antibiotics are required in wound botulism. Vaccination is rarely used and its effectiveness has not been fully evaluated.[6]

**Prevention**

The toxin is destroyed by normal cooking processes. *C. botulinum* will not grow and therefore the toxin will not be formed in acidic foods (pH<4.6). However, the low pH will not inactivate toxins that have already formed. Good food preparation practices especially preservation and hygiene are key factors in the prevention of foodborne botulism.[6]

**Lorraine Hickey, HPSC**

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