Illness associated with seafood

A C Scoging

Summary

Seafood is a common vehicle for the transmission of infections and intoxications. The main concerns in the UK are scombroid fish poisoning, viral infections (associated with bivalve molluscs) and bacterial infections (associated with crustaceans). Paralytic shellfish poisoning (PSP) and red whelk poisoning occur sporadically, and ciguatera has started to appear associated with imported fish. The appearance in coastal waters of marine algae associated with diarrhetic shellfish poisoning (DSP) has increased the likelihood of outbreaks of this toxic syndrome. The dinoflagellates causing neurotoxic shellfish poisoning and amnesic shellfish poisoning have not been detected in UK fishing waters but the fluctuating distribution of algal populations makes their appearance a possibility. The Food Hygiene Laboratory and the Torry research station of the Ministry of Agriculture, Fisheries and Food provide a reference service for scombrototoxin, ciguatera, DSP, PSP and red whelk poisoning in the UK.

Introduction

Fish, shellfish and their products are responsible for a significant proportion of foodborne diseases worldwide. The percentage of outbreaks associated with seafood depends on local climate and dietary customs. Seafood is involved in an estimated 11% of foodborne outbreaks in the USA, 20% in Australia and over 70% in Japan which has a greater tradition of eating raw seafood. Seafood has a good reputation in the UK and is the vehicle of infection in only 2% of all reported foodborne outbreaks. However, patterns of seafood consumption are changing – more fish are being eaten due to growing awareness of their beneficial dietary effects and tastes are widening to include more raw fish dishes and exotic species. London’s Billingsgate fish market currently sells over 150 different species imported from all over the world.

The major illnesses transmitted by fish and shellfish fall into three main categories: allergies, infectious diseases and toxic syndromes. Allergic reactions to fish and shellfish are not uncommon and some individuals may react to the preservatives and flavour and colour enhancers added to them. Further information on allergy to seafoods can be found elsewhere. This review focuses on the infections and intoxications associated with seafood.

Infections

Bacterial diseases

Diseases caused by bacteria constitute a large proportion of fish and shellfish-borne illnesses. Table I compares the outbreaks associated with bacteria in the UK and USA, divided into the following three categories.

1. Micro-organisms in the natural marine environment

Vibrio species: the most serious threat to human health is posed by the vibrios. These bacteria are native to warm coastal waters and estuaries and may also be found in inland freshwater. V. parahaemolyticus, V. fluvialis and V. alginolyticus are indigenous to UK coastal waters. Of the 11 pathogenic Vibrio species, six have been associated with seafood transmitted illness: V. cholerae, V. parahaemolyticus, V. vulnificus, V. mimicus, V. hollisae and V. furnissi.
Outbreaks of endemic cholera, caused by V. cholerae O1, continue to occur worldwide; many having food as the vehicle of transmission. In Italy, in 1974, mussels and clams and other partially cooked bivalves were implicated in an outbreak involving 278 people of whom 25 died. All recent outbreaks in the USA have involved inadequately cooked or mishandled crustaceans such as crabs or shrimps. There are at least 80 strains of non O1 V. cholerae widely distributed in the environment. Most of those isolated from seafood are incapable of producing the cholera toxin but can cause gastrointestinal illness. Two outbreaks have been associated with eating raw oysters.

V. paraaeromolyticus occupies coastal waters worldwide and shows an increase in numbers in summer months. Most isolates obtained from seafood and marine environments are avirulent but the percentage of isolates capable of producing gastroenteritis varies geographically. V. paraaeromolyticus causes 45-70% of the total bacterial food poisoning outbreaks in Japan. The first UK outbreak was recorded in 1974 when 12 holidaymakers became ill after eating dressed crab. V. vulnificus has been chrestened the "terror of the deep" due to the severity of the illness it produces. The organism invades the intestinal tract causing a primary septicemia. This progressive disease has a mortality rate of around 50%. V. vulnificus exists naturally in the Atlantic and Pacific oceans and the Gulf of Mexico. Sixty-two cases were reported in Florida between 1981 and 1987, many following the consumption of raw oysters.

Aeromonas hydrophila: aeromonads are indigenous in sea water and may have been introduced through sewage contamination. A. hydrophila has been implicated in recent cases of gastroenteritis following the consumption of contaminated shellfish. In 1986, 472 cases of gastroenteritis were associated with frozen raw oysters which had been stored at -72°C for 18 months, highlighting the survival properties of A. hydrophila under extreme conditions. The occurrence of A. hydrophila in shellfish-growing waters has yet to be fully investigated and its role as a foodborne pathogen is unclear.

Plesiomonas shigelloides: P. shigelloides has been the cause of several recently reported cases of gastroenteritis in different parts of the world. Common food vehicles for this organism have been raw oysters, mussels, salt mackerel and cuttlefish.

Clostridium botulinum: the first case of botulism resulting from fish consumption was described in 1818. C. botulinum had not been discovered as an organism at that time and the disease was attributed to a "toxic fatty acid" thought to develop in sausages during storage. The link between seafood and botulism became well established in areas such as Japan and Alaska, where fish is traditionally eaten raw or lightly cooked. Botulism outbreaks in North America and the UK have mainly involved preserved products. The most recent recorded outbreak in the UK involving seafood occurred in 1978: four deaths followed the consumption of salmon from a pierced tin.

Of the eight existing types of C. botulinum, type E is most frequently isolated from aquatic environments. These strains are non-proteolytic and are capable of growth down to 3-5°C. Botulism arises when bacterial growth and the subsequent production of neurotoxin occurs in food. Most cooking processes destroy heat-sensitive type E spores and toxins. C. botulinum type F and proteolytic strains of types B and F produce highly heat-resistant spores, although they have been rarely associated with fish.

### Table 1 Outbreaks associated with bacteria

<table>
<thead>
<tr>
<th>Bacterial species</th>
<th>Number of outbreaks</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Naturally occurring in water</strong></td>
<td>USA 1973-87</td>
</tr>
<tr>
<td>Vibrio cholerae O1</td>
<td>5</td>
</tr>
<tr>
<td>V. cholerae non O1</td>
<td>2</td>
</tr>
<tr>
<td>V. furnissi</td>
<td>-</td>
</tr>
<tr>
<td>V. hollisae</td>
<td>-</td>
</tr>
<tr>
<td>V. mimicus</td>
<td>-</td>
</tr>
<tr>
<td>V. parahaemolyticus</td>
<td>19</td>
</tr>
<tr>
<td>V. vulnificus</td>
<td>Unknown</td>
</tr>
<tr>
<td>Aeromonas hydrophila</td>
<td>2</td>
</tr>
<tr>
<td>Plesiomonas shigelloides</td>
<td>-</td>
</tr>
<tr>
<td>Clostridium botulinum</td>
<td>35</td>
</tr>
<tr>
<td><strong>2. Occurring in polluted waters:</strong></td>
<td>USA 1978-83</td>
</tr>
<tr>
<td>Salmonella sp.</td>
<td>8</td>
</tr>
<tr>
<td>Shigella sp.</td>
<td>7</td>
</tr>
<tr>
<td><strong>3. Post-catching contamination:</strong></td>
<td>USA 1978-83</td>
</tr>
<tr>
<td>Bacillus cereus</td>
<td>3</td>
</tr>
<tr>
<td>Campylobacter</td>
<td>2</td>
</tr>
<tr>
<td>Clostridium perfringens</td>
<td>5</td>
</tr>
<tr>
<td>Escherichia coli</td>
<td>3</td>
</tr>
<tr>
<td>Staphylococcus aureus</td>
<td>5</td>
</tr>
<tr>
<td>Streptococcus pyogenes</td>
<td>1</td>
</tr>
</tbody>
</table>

Salmonella sp. have also been implicated in post-catching contamination.

**Communicable Disease Report**

**11 Oct 1991**
Listeria species has been demonstrated in frozen seafood products such as shrimp and crab as well as in fresh seafood and coastal waters although no outbreaks of listeriosis have been directly linked to seafood so far. It is important, therefore, to consider Listeria in the microbiological assessment of seafood for human consumption.

Viral diseases
Viruses enter the marine environment by the direct discharge of domestic sewage and the ocean dumping of sewage sludge. Enteric viruses show greater resistance to sewage treatment processes than enteric bacteria and remain viable in seawater for many weeks or months (hepatitis A can survive for over a year). Small round structured viruses (SRSV) of the Norwalk group are the commonest cause of viral gastroenteritis associated with shellfish. The main symptoms are nausea, vomiting, diarrhea and abdominal cramps, which develop after 24-48 hours and last for 1-2 days. Other viruses, such as astrovirus and calicivirus, have been implicated in seafood associated outbreaks.

The first report linking an outbreak of hepatitis A with the consumption of clams occurred in 1955 and shellfish transmission of this virus remains a major public health concern. A UK study attributed 25% of hepatitis A outbreaks to shellfish consumption up to 1983. A threat is also posed by enterically transmitted non-A non-B hepatitis. Cases of waterborne hepatitis have been reported recently, mostly in developing countries and, despite the use of chlorination and other water treatment processes, outbreaks of A non-B hepatitis have occurred. Viruses enter coastal water although no outbreaks of listeriosis have been linked with viruses as well as bacteria but the removal of viruses is much slower and may be incomplete. Enteric viruses generally require wild or marine fish as a host, with the exception of calicivirus, which is associated with food and human consumption. Waterborne viral disease can be an important vehicle of transmission. The non-multiplication of viral particles in food and the low infective dose (about 10⁴-10⁵ particles per ml) for some viruses make the laboratory investigation of viral food poisoning difficult. This is exacerbated by the limitations of the available detection methods: electron microscopy (detection limit 10⁴-10⁵ particles per ml) and radioimmunoassay (10⁴-10⁵ particles per ml). However, the situation should improve with the adoption of gene probes and polymerase chain reaction techniques for sample analysis.

Depuration and relaying (see above) reduce contamination with viruses as well as bacteria but the removal of viruses is much slower and may be incomplete. Heat treatment is the only sure way to eliminate viruses. Recommendations from the Ministry of Agriculture, Fisheries and Food (MAFF), introduced in 1988, stated that the internal temperature of molluscs must be maintained at 90°C for 1.5 minutes before consumption. This has had a significant effect on the number of cases of viral gastroenteritis transmitted by molluscs.

Parasitic diseases
Parasitic infections following fish consumption are rare in the UK. Trematode and cestode (fluke and tapeworm) infections are common only where it is customary to consume raw or undercooked fish, eg, Japan, Saudi Arabia and South and East Asia. The larvae are commonly found in fish flesh but are destroyed by cooking, smoking and freezing. Fish destined for raw fish dishes should be kept at -20°C for 24 hours.

The main source of parasitic infection from the marine environment is the nematode Anisakis. Anisakiasis is commonly associated with marine salmon and herring and results from the burrowing of larvae into the gastrointestinal tract of the host. The disease can remain asymptomatic until the worm is vomited by the patient. Giardia lamblia and helmints are mainly associated with freshwater fish.

Intoxications
Toxins ingested with seafood produce many diverse symptoms (Table 2). Four groups of toxin exist: those of algal origin which go through a series of concentration steps up the food chain (shellfish toxins, ciguatoxins); toxins resulting from bacterial spoilage (scombrotxin and possibly tetrodotoxin); naturally occurring toxins (tetramine), and chemical contaminants present in polluted marine environments.

Paralytic shellfish poisoning (PSP)
PSP is caused by the consumption of shellfish contaminated with marine dinoflagellates occurring in latitudes greater than 30° North or South, where ambient temperatures are about 15-17°C. The algae exist as cysts on the seabed. When the temperature, pH and available nutrients are suitable, they rise to the surface and proliferate creating visible "red tides". These algal blooms last for 2-3 weeks during the summer months before they die away and are replaced by other non-toxic algae. Three algal species are linked to PSP: Gonyaulax catenella is found along the Pacific coastline of North America and in Japan; G. tamarensis has been associated with outbreaks occurring on the East coast of America and the coast of mainland Europe and, since the early 1970s, Pyrodinium bahamenese has been identified with PSP outbreaks in South America and South East Asia. One hundred and eighty-seven people, of whom 26 died, were poisoned with PSP in Guatemala in 1987 after eating local clams contaminated with this organism.

All UK incidents have involved mussels and 10 outbreaks of PSP were identified between 1927 and 1975, with 116 patients affected and 14 deaths. No further cases of PSP have been recorded in the UK since the epidemic of 1968 involving 78 people who ate mussels fished from the Northumbrian coast contaminated with G. tamarensis. An extensive monitoring programme has since been carried out during high risk periods by MAFF. This analyses coastal waters, shellfish and some crustacea for PSP toxin levels. In 1990, crabs from North Eastern waters were found to contain toxic levels greater than 50 times the acceptable level.

PSP is characterised by numbness in the mouth and fingertips followed by impaired muscle co-ordination. Respiratory distress and paralysis can occur in severe cases, occasionally with fatal results. The major toxin is saxitoxin – a substituted dibasic tetrahydropurine. It induces paralysis by blocking sodium channels in cell membranes. At least 18 other toxic derivatives have been identified, either natural algal toxins – such as neosaxitoxin produced by G. tamarensis var exuvata – or metabolised derivatives of algal products found in shellfish. All are heat stable and will survive normal cooking processes.

Neurotoxic shellfish poisoning (NSP)
NSP toxins are produced by the dinoflagellate Pychodiscus brevis. Outbreaks of NSP have been associated with the consumption of oysters, clams and other bivalve molluscs mainly in North America. P. brevis is the organism responsible for Florida's famous red tides which cause eye irritation and coughs in swimmers exposed to toxins released into the surf by lysed algae. The neurotoxins produced by this organism are brevetoxins and possibly tetrodotoxin; naturally occurring toxins (tetramine), and chemical contaminants present in polluted marine environments.

Paralytic shellfish poisoning (PSP)
PSP is caused by the consumption of shellfish contaminated with marine dinoflagellates occurring in latitudes greater than 30° North or South, where ambient temperatures are about 15-17°C. The algae exist as cysts on the seabed. When the temperature, pH and available nutrients are suitable, they rise to the surface and proliferate creating visible "red tides". These algal blooms last for 2-3 weeks during the summer months before they die away and are replaced by other non-toxic algae. Three algal species are linked to PSP: Gonyaulax catenella is found along the Pacific coastline of North America and in Japan; G. tamarensis has been associated with outbreaks occurring on the East coast of America and the coast of mainland Europe and, since the early 1970s, Pyrodinium bahamenese has been identified with PSP outbreaks in South America and South East Asia. One hundred and eighty-seven people, of whom 26 died, were poisoned with PSP in Guatemala in 1987 after eating local clams contaminated with this organism.

All UK incidents have involved mussels and 10 outbreaks of PSP were identified between 1927 and 1975, with 116 patients affected and 14 deaths. No further cases of PSP have been recorded in the UK since the epidemic of 1968 involving 78 people who ate mussels fished from the Northumbrian coast contaminated with G. tamarensis. An extensive monitoring programme has since been carried out during high risk periods by MAFF. This analyses coastal waters, shellfish and some crustacea for PSP toxin levels. In 1990, crabs from North Eastern waters were found to contain toxic levels greater than 50 times the acceptable level.

PSP is characterised by numbness in the mouth and fingertips followed by impaired muscle co-ordination. Respiratory distress and paralysis can occur in severe cases, occasionally with fatal results. The major toxin is saxitoxin – a substituted dibasic tetrahydropurine. It induces paralysis by blocking sodium channels in cell membranes. At least 18 other toxic derivatives have been identified, either natural algal toxins – such as neosaxitoxin produced by G. tamarensis var exuvata – or metabolised derivatives of algal products found in shellfish. All are heat stable and will survive normal cooking processes.

Neurotoxic shellfish poisoning (NSP)
NSP toxins are produced by the dinoflagellate Pychodiscus brevis. Outbreaks of NSP have been associated with the consumption of oysters, clams and other bivalve molluscs mainly in North America. P. brevis is the organism responsible for Florida's famous red tides which cause eye irritation and coughs in swimmers exposed to toxins released into the surf by lysed algae. The neurotoxins produced by this organism are brevetoxins B and C. The precise mode of action of these polyethers is unknown although they bind to nerve cells and cause gastrointestinal symptoms, numbness of the mouth, muscular aches and dizziness.
Amnesic shellfish poisoning (ASP)  
ASP was first described in Canada, when 107 patients were ill and three died after eating cultivated blue mussels in 1987. The symptoms include vomiting, diarrhoea, abdominal cramps, headache and loss of short term memory; the latter may be permanent in some cases. The causative agent was identified as domoic acid originating from the marine diatom *Nitzschia pungens*, which is widely distributed in coastal waters of the Atlantic, Pacific and Indian oceans. Domoic acid is acutely neurotoxic for the mammalian central nervous system.

**Diarrhetic shellfish poisoning (DSP)**

DSP has been a severe public health problem in Japan for many years (1300 cases were recorded between 1976 and 1982) and now has a worldwide distribution due to the shifting population patterns of the algae *Dinophysis* and *Prorocentrum*²⁹. Outbreaks have also occurred in the Netherlands, France and Italy; 150 people being poisoned by contaminated mussels in the last example.³⁰ No cases of DSP have been reported in the UK, although DSP toxins have been detected in cockles grown in estuarine waters.

The major toxic components are okadaic acid and dinophysistoxins 1 to 3, originally characterised from material obtained from the blue mussel. Other DSP toxins include yessotoxin and the pectenotoxins derived from scallops (*Patinopex yessoensis*). As toxin denaturation only occurs after very rigorous boiling (163 minutes at 100°C), the monitoring of toxic levels in shellfish beds and subsequent closure when high levels are detected remain the only safeguards against DSP.³¹ Shellfish acting as vehicles for DSP include mussels, scallops and clams. The most common symptoms are diarrhoea, nausea, vomiting and abdominal pain which can persist for up to 3 days.

Ciguatera  
Ciguatera was first documented in the West Indies in 1555, and in the Pacific in 1606 when a Spanish crew sailing with the explorer de Quins suffered typical gastrointestinal and neurological symptoms, although it has been suggested that this incident was, in fact, one of diarrhetic shellfish poisoning. The name ciguatera is derived from the marine snail *Turbo pica*, called Ciga by Spanish migrants to the Caribbean³².

Ciguatera is the largest global public health problem associated with seafood. The worldwide incidence has been estimated at 50,000 cases per year³³. Under-reporting accounts for most of the disparity between the estimate of 2000 cases per year in the USA and the 20-30 cases per year recorded by the Centers for Disease Control in Atlanta³⁴. So far, 3 incidents of ciguatera poisoning have been recorded in the UK. Those in 1979 and in 1980 resulted from the consumption of an eel cured in Antigua and imported privately into the UK. The second involved poached barracuda eaten immediately prior to a return flight to the UK. In 1990, three people suffered ciguatera poisoning after eating a home cooked red snapper imported from Oman³⁵. The Pacific, Caribbean and Indian oceans are the main danger areas for ciguatera. The principal fish involved are groupers, snappers, barracudas, jacks, surgeon fishes and sea basses, all of which are now regularly imported into the UK.

The symptoms of ciguatera vary widely as several toxins are involved and the response is dose-dependent. The clinical features

---

**Table 2 Toxic diseases associated with seafood**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Associated micro-organisms</th>
<th>Toxins</th>
<th>Clinical features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paralytic shellfish poisoning</td>
<td><em>Gonyaulax tamarenisis</em></td>
<td>Saxitoxins</td>
<td>Tingling in the mouth and extremities, dizziness, floating sensation, paralysis, respiratory distress, death</td>
</tr>
<tr>
<td>(PSP)</td>
<td><em>Gonyaulax catenellla</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td><em>Pyrodinium bahamense</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurotoxic shellfish poisoning</td>
<td><em>Ptychodiscus brevis</em></td>
<td>Brevetoxins</td>
<td>Paraesthesia, abdominal pain, nausea, diarrhoea, reversal of temperature sensation, temporary blindness, paralysis, death</td>
</tr>
<tr>
<td>(NSP)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amnesic shellfish poisoning</td>
<td><em>Nitzschia pungens</em></td>
<td>Domoic acid</td>
<td>Nausea, vomiting, disorientation, memory loss, organ failure (kidney, lung), death</td>
</tr>
<tr>
<td>(ASP)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diarrhetic shellfish poisoning</td>
<td><em>Prorocentrum lima</em></td>
<td>Okadaic acid</td>
<td>Nausea, vomiting, diarrhoea, abdominal pain</td>
</tr>
<tr>
<td>(DSP)</td>
<td><em>Dinophysis fortii</em></td>
<td>Dinophysis toxins</td>
<td></td>
</tr>
<tr>
<td></td>
<td><em>Dinophysis acuminata</em></td>
<td>Yessotoxin</td>
<td></td>
</tr>
<tr>
<td></td>
<td><em>Dinophysis norvegica</em></td>
<td>Pectenotoxins</td>
<td></td>
</tr>
<tr>
<td>Ciguatera</td>
<td><em>Gambierdiscus toxicus</em></td>
<td>Ciguatoxins</td>
<td>Nausea, vomiting, diarrhoea, abdominal pain, dizziness, blurred vision, reversal of temperature sensation, temporary blindness, paralysis, death</td>
</tr>
<tr>
<td></td>
<td><em>Prorocentrum concavum</em></td>
<td>Maitotoxin</td>
<td></td>
</tr>
<tr>
<td></td>
<td><em>Prorocentrum mexicana</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scombroide fish poisoning</td>
<td><em>Morganeila morganii</em></td>
<td>Histamine</td>
<td>Nausea, vomiting, diarrhoea, headache, flushing, burning in the mouth, hypotension, rash, urticaria, oedema, localised inflammation (chest and neck)</td>
</tr>
<tr>
<td></td>
<td><em>Hafnia alvei</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td><em>Klebsiella pneumoniae</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td><em>Proteus sp.</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td><em>Vibrio sp.</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td><em>Pseudomonas sp.</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td><em>Alteromonas sp.</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td><em>Shewanella sp.</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Puffer fish poisoning</td>
<td></td>
<td>Tetrotoxin</td>
<td>Paraesthesia, floating sensation, dysphagia, hypotension, bradycardia, respiratory distress, ascending paralysis, death</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Red whelk poisoning</td>
<td></td>
<td>Tetramine</td>
<td>Blurred vision, muscular twitching, weakness, paralysis, collapse</td>
</tr>
</tbody>
</table>
include nausea, vomiting, abdominal pain, dizziness, blurred vision, reversal of the sensations of heat and cold, blindness which may be temporary, paralysis and death. It has a mortality rate of 7-20%. Onset is usually within a few hours of consumption and the neurological effects can persist for several months. As yet, there is no antidote to ciguatoxin.

The origins of ciguatera poisoning remained unknown until 1977 when the marine dinoflagellate Gambierdiscus toxicus was implicated as the causative alga. Subsequently two other algae, Proorocentrum concavum and P. mexicana, were also shown to produce similar neurotoxins. Ciguatoxins are low molecular weight polyethers which act by causing increased depolarisation of nerve endings and release of neurotransmitters. Maitotoxin has also been purified from G. toxicus and from ciguatoxic fish but little is known, as yet, about its structure or mode of action.

These heat stable toxins are unaffected by cooking and processing methods and ciguatoxic fish are normal in appearance. The only advice available to consumers is to avoid large fish of the hazardous species, in which the effects of toxin concentration up the food chain are greatest. Toxin detection is difficult only advice available to consumers is to avoid the spoilt fish after ingestion of histamine by the gastrointestinal tract. Other fish spoilage products, such as cadaverine and putrescine, may act as potentiators for histamine activity. Histamine is heat stable and will withstand canning procedures, so the main control measures for scombroid poisoning are low temperature storage of fish immediately after catching and during processing and the use of hygienic food handling practices throughout preparation.

Puffer fish poisoning

Incidents of puffer fish poisoning are rare outside Japan, where over 6000 cases have been recorded in the last 78 years. The causative toxin is tetrodoxin (and its derivative anhydrotetrodotoxin), a heat stable, non-protein neurotoxin concentrated in the skin and viscera of puffer fish, porcupine fish, ocean sunfish and species of newts and salamanders. No cases have, as yet, been recorded in the UK.

The toxin blocks the sodium fast channel during depolarisation, in a similar way to saxitoxin, and causes numbness in the face and extremities, a floating sensation, weakness, ascending paralysis and respiratory failure. The disease has a mortality rate approaching 60%. Onset is usually within 3 hours and symptoms may last for 3 days, if the patient survives the first 24 hours. There have been recent suggestions that tetrodoxin may have a bacterial origin. Bacteria belonging to Vibrio sp, Sheananella sp, Pseudomonas sp and Alteromonas sp have been shown to produce tetrodotoxin.

Red whelk poisoning

Six incidents of red whelk poisoning have been recorded in the UK since 1970 - four in Scotland (1970, two in 1986, and 1987) and two in the North of England, in 1979 and 1991. Poisoning results from the consumption of the red whelk (Neptunea antiqua) which is occasionally caught by fishermen trawling for the edible whelk (Buccinum undatum) off the North East coast of Britain. Both whelks appear similar although the red whelk is larger, smoother and has a yellow/orange coloration. The red whelk contains the metabolite tetramine in its salivary gland. This curare-like compound produces blurred vision, muscular twitching, weakness, paralysis and collapse. The symptoms usually resolve within 24 hours.

<table>
<thead>
<tr>
<th>Year</th>
<th>Number of suspected incidents</th>
<th>Number of cases</th>
<th>Number of incidents &gt;5mg% histamine</th>
</tr>
</thead>
<tbody>
<tr>
<td>1976-78</td>
<td>7</td>
<td>19</td>
<td>3</td>
</tr>
<tr>
<td>1979</td>
<td>44</td>
<td>178</td>
<td>29</td>
</tr>
<tr>
<td>1980-82</td>
<td>85</td>
<td>240</td>
<td>44</td>
</tr>
<tr>
<td>1983-85</td>
<td>95</td>
<td>151</td>
<td>21</td>
</tr>
<tr>
<td>1986-88</td>
<td>117</td>
<td>197</td>
<td>34</td>
</tr>
<tr>
<td>1989-90</td>
<td>93</td>
<td>177</td>
<td>36</td>
</tr>
<tr>
<td>Total</td>
<td>441</td>
<td>962</td>
<td>167</td>
</tr>
</tbody>
</table>

* Food Hygiene Laboratory data

The proof that histamine is the causative agent of scombroid poisoning lies in three areas; the detection of high histamine levels in implicated fish samples, the resemblance of the symptoms to known histamine responses and the successful use of antihistamines in the treatment of the disease. A recent study measured the urinary excretion of histamine and its metabolite, N-methylhistamine, in scombroid patients and concluded that the high levels detected were derived from the spoiled fish after ingestion of histamine by the gastrointestinal tract. Other fish spoilage products, such as cadaverine and putrescine, may act as potentiators for histamine activity. Histamine is heat stable and will withstand canning procedures, so the main control measures for scombroid poisoning are low temperature storage of fish immediately after catching and during processing and the use of hygienic food handling practices throughout preparation.

Table 3: Incidents of scombrotoxic fish poisoning

<table>
<thead>
<tr>
<th>Year</th>
<th>Number of suspected incidents</th>
<th>Number of cases</th>
<th>Number of incidents &gt;5mg% histamine</th>
</tr>
</thead>
<tbody>
<tr>
<td>1976-78</td>
<td>7</td>
<td>19</td>
<td>3</td>
</tr>
<tr>
<td>1979</td>
<td>44</td>
<td>178</td>
<td>29</td>
</tr>
<tr>
<td>1980-82</td>
<td>85</td>
<td>240</td>
<td>44</td>
</tr>
<tr>
<td>1983-85</td>
<td>95</td>
<td>151</td>
<td>21</td>
</tr>
<tr>
<td>1986-88</td>
<td>117</td>
<td>197</td>
<td>34</td>
</tr>
<tr>
<td>1989-90</td>
<td>93</td>
<td>177</td>
<td>36</td>
</tr>
<tr>
<td>Total</td>
<td>441</td>
<td>962</td>
<td>167</td>
</tr>
</tbody>
</table>

* Food Hygiene Laboratory data

The proof that histamine is the causative agent of scombroid poisoning lies in three areas; the detection of high histamine levels in implicated fish samples, the resemblance of the symptoms to known histamine responses and the successful use of antihistamines in the treatment of the disease. A recent study measured the urinary excretion of histamine and its metabolite, N-methylhistamine, in scombroid patients and concluded that the high levels detected were derived from the spoiled fish after ingestion of histamine by the gastrointestinal tract. Other fish spoilage products, such as cadaverine and putrescine, may act as potentiators for histamine activity. Histamine is heat stable and will withstand canning procedures, so the main control measures for scombroid poisoning are low temperature storage of fish immediately after catching and during processing and the use of hygienic food handling practices throughout preparation.
References


AC Scoging BSc
Food Hygiene Laboratory
Central Public Health Laboratory
OUTBREAK FORUM - II

This feature aims to provide an opportunity for the publication of short reports (400 – 1000 words plus one figure or table) that highlight an important message culled from an outbreak investigation. These items are peer reviewed and, if accepted, will be published in conjunction with an expert comment. Individual contributions should be cited in the usual way, eg, Evans MR, Riley CL, Ribeiro CD. Fried rice from the take-away. Communicable Disease Report 1991; 1: R100. The Editor of the CDR also welcomes the submission of fuller reports to be considered for publication as articles elsewhere in the CDR review.

Sequential outbreaks in a hotel

One hundred and thirty-three people attended a buffet for school leavers held in a hotel in the North East. Two days later, several were reported to have developed some or all of the following: nausea, vomiting, diarrhoea, abdominal pain, headache and fever. While investigation of this outbreak was in progress, information was received that some of a party of 95 who attended a wedding party at the same hotel four days later had developed similar symptoms.

Investigation

The hotel premises were inspected and food storage, handling and preparation practices observed. A list of guests was obtained for both functions and questionnaires seeking information about illness and the consumption of food items were sent to the exposed populations. For outbreak I, they were administered through the school or by interview at home for those too ill to attend school. For outbreak II, they were delivered by environmental health officers in the district of residence of the guests. Faecal samples were taken, where possible, from those guests who had developed diarrhoea after either function. A case was defined as anyone who developed vomiting, diarrhoea or abdominal pain, within 72 hours of attending either function. The epidemic curve is shown in figure 1.

Figure 1 Onset of illness for both outbreaks

<table>
<thead>
<tr>
<th>Days</th>
<th>Number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>20</td>
</tr>
<tr>
<td>2</td>
<td>15</td>
</tr>
<tr>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>6</td>
<td>1</td>
</tr>
</tbody>
</table>

Outbreak I

One hundred and twenty-seven questionnaires were returned (response rate 91%) and 66 cases were identified. The mean time between the buffet and onset of symptoms was 31 hours (range 6-64 hours). Chi-square analysis of the data showed a significant association between illness and the consumption of tuna (p <0.0001), prawns (p <0.01) and chicken (p <0.05). Further analysis using logistic regression1 to identify the separate and combined effects of the three foods showed that chicken had little effect, prawns with or without tuna were strongly associated with illness (p <0.01; odds ratio = 5.2) while tuna alone was very strongly associated with illness (p <0.00001; odds ratio = 36.0). Leftover food from the buffet (including the tuna) and 26 faecal specimens obtained from cases were examined for bacteria and viruses with negative results.

Outbreak II

Eighty-five questionnaires were returned (response rate 89%) and 19 cases identified. The mean time between the party and onset of symptoms was 28 hours (range 6-48 hours). Chi-square analysis showed a significant association between illness and the consumption of tuna (p <0.0001), roast ham (p <0.02) and seafood cocktail (p <0.03). Analysis to assess the separate and combined effects of these three foods showed that ham had no independent effect, seafood cocktail showed slight evidence of an effect (p <0.05; odds ratio = 4.4) and tuna showed a strong association with illness (p <0.0005; odds ratio = 17.0). No food samples were obtained and microbiological examination of 12 faecal specimens was negative.

Interviews with the hotel staff revealed that no exclusion policy was in operation for sick food handlers and hotel staff sometimes prepared their own meals in the kitchen. One staff member had symptoms of gastroenteritis in the week before the first outbreak. Another had diarrhoea during the night preceding the buffet and made tuna sandwiches for herself the next day from the tuna subsequently used at the buffet. Another ate the tuna on the day of the buffet and was ill two days later. Separate tuna was prepared for the two functions. Faecal specimens obtained from all affected staff members were negative on microbiological examination.

Discussion

The clinical presentation in both outbreaks was typical of viral gastroenteritis. The most likely cause of the first was contamination of tuna by an infected food handler. The second outbreak may have arisen for the same reason or following contact with contaminated surfaces. The occurrence of illness in several (c.6) family contacts of cases supports a viral cause although secondary cases were not sought specifically. Laboratory confirmation of a viral cause was not, however, forthcoming. The samples may have been sent too late in the illness for virus particles to be detected or may have been too small for complete examination for viruses and bacteria.

Sequential outbreaks occurring at short intervals emphasise the need for prompt investigation and analysis to enable appropriate action to be taken. The gap of only 48 hours between the two makes it unlikely that any intervention...
would have prevented the second outbreak, but this sequence underlines the need for catering management to comply with food hygiene regulations \(^2\) and operate an effective exclusion policy for food handlers with gastroenteritis.

*I Holtby, MFPHM, Consultant in communicable disease control, South Tees Health Authority; I Gillis, MB ChB, Senior registrar in public health medicine, Northern Regional Health Authority and R Madhok, MSc MFPHM, Consultant in public health medicine, South Tees Health Authority. The authors thank the PHLS Statistics Unit at CDSC for helpful advice.

**Comment**

Consecutive outbreaks of foodborne disease of presumed viral aetiology are not unusual. It is uncommon, however, for a single food vehicle to be identified epidemiologically in viral gastroenteritis, possibly because the small inoculum required leads to multiple vehicles being implicated. When foods are identified they are usually dishes which have been handled and not subsequently heat treated. Unlike outbreaks of salmonellosis, symptomatic or convalescent food handlers are likely sources of infection. PHLS guidance \(^2\) advises that food handlers be excluded from work for 48 hours after the resolution of symptoms of viral gastroenteritis, although scrupulous personal hygiene is still necessary when they return. Food handlers were suspected as the source in five recent outbreaks reported to CDSC in three of these recovery was said to have taken place more than 48 hours before preparing the food.

**Gastroenteritis and poached salmon**

About fifty guests developed symptoms of acute gastroenteritis shortly after attending a twenty-first birthday reception at a hotel in the North West.

**Investigation**

Seventy-two of the 78 guests returned questionnaires, 50 of whom were ill. The mean incubation period was 40 hours (range 17-85 hours) with a duration of illness of 1-2 days (Figure 2). Cases were defined as those developing two or more of the following symptoms within four days of the party: nausea, vomiting, diarrhoea or abdominal pain – 47 fitted this definition. Sixty-nine food histories were analysed. Of the 16 foods eaten, only poached salmon showed a significant association with illness (\(p = 0.01;\) Fisher’s exact test). Seventy-nine percent of those who became ill ate salmon compared with 40% who did not (Table). The guests had come from many parts of the country and faecal samples were only obtained from four cases. Bacteriological examination proved negative. No remaining foods were available for sampling.

All the fish dishes were prepared by the same person. The salmon, which was fresh, was gutted and cleaned prior to poaching for 20 minutes. It was kept refrigerated before laying out. Two hours before serving it was cut into portions, laid on plates and garnished with cucumber and lettuce. The garnish was prepared by another food handler and was used for many of the other dishes. None of the food handlers or their close contacts was ill prior to or on the day of the event.

**Discussion**

The response rate for the questionnaires was high (92%). The analysis yielded a significant association with poached salmon. The incubation period, symptomatology and negative bacteriology suggest a viral gastroenteritis although the incriminated food appeared well-cooked and none of the food handlers admitted to illness.

R Cruickshank, MB ChB, Registrar in public health medicine and C Quigley, MSc MFPHM, Consultant in communicable disease control, Trafford Health Authority.

**Table**

<table>
<thead>
<tr>
<th></th>
<th>Ill</th>
<th>Not ill</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salmon eaten</td>
<td>38</td>
<td>10</td>
<td>48</td>
</tr>
<tr>
<td>Salmon not eaten</td>
<td>6</td>
<td>9</td>
<td>15</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>44</td>
<td>19</td>
<td>63</td>
</tr>
</tbody>
</table>

**Comment**

Several recent outbreaks of bacterial food poisoning (eg, *Clostridium perfringens* \(^4\) and *Salmonella* \(^5\)) have been associated with poached salmon but the incubation periods were shorter, incriminated organisms were isolated from patients and food, and there was evidence of failure of kitchen hygiene. In this outbreak, it is possible that viruses present in the raw fish were not inactivated by the cooking procedure. The diagnosis of viral gastroenteritis is usually made by electron microscopy of food or faecal samples. Large volumes are not required and virus structures may still be detected in samples after several days. However, faecal samples need to be taken within 24-48 hours of onset of illness as the amount of virus present declines rapidly in some forms of virus gastroenteritis (eg, SRSV). Serological assays are under evaluation and offer the prospect of diagnosing a viral cause in a much larger proportion of cases \(^6\).
Red spells danger for whelk eaters

In July 1991, an outbreak of red whelk poisoning occurred in North East England. A man (A) from Newcastle-upon-Tyne purchased a quantity of fresh whelks in their shells from a quayside vendor in a nearby coastal town. He gave half the whelks to his sister (B) who lived within two miles of him. Both boiled their whelks in their shells for twenty minutes (A boiled his in a pressure cooker at full pressure, B boiled hers over an open fire), then shelled and seasoned them before serving them to other members of their respective families. The whelks were eaten by six people in all; mostly that evening but some the following day. Members of both families developed symptoms within 30-120 minutes which included dizziness, ataxia, diplopia, impaired accommodation, muscle cramps and weakness. Two were driving when their visual symptoms occurred and one narrowly avoided an accident. Symptoms resolved within a few hours.

The man (A) complained to the vendor to no effect and then contacted the local environmental health department. After obtaining details of the cases and contacting CDSC, the investigating officer collected some of the discarded shells. CDSC alerted the regional CCDC.

Investigation

A case of red whelk poisoning was defined as (i) eating red whelks and (ii) developing neuromuscular symptoms including at least one of the following: diplopia, difficulty in focusing, dizziness and muscle cramps. Five cases fell within this definition. No regular commercial whelk fishery or retail operation exists in the North East and enquiries yielded no further cases. No further epidemiological study or laboratory investigations were carried out.

Discussion

Both families eat shellfish regularly, buying them fresh or prepared or, at certain times of the year, gathering them from the shore at low tide. The purchaser knew that his batch of whelks were not dog whelks (*Nucella lapillus*) which he thought unfit for consumption. He regarded the batch as good sized and free from slime. Although he and his sister noticed an orange colour on the inner aspect of some of the shells, they did not realise that this was distinctive of the red whelk (*Neptunea antiqua*) (Figure 3). The edible whelk (*Buccinum undatum*) lacks this coloration. It was not possible to identify how many of the whelks eaten were, in fact, red whelks.

The only known vendor of locally caught whelks voluntarily agreed to stop selling, and the environmental health officer (EHO) is maintaining close vigilance for whelks being sold elsewhere at the port. EHOs have been asked to advise on whelk selling from Berwick-on-Tweed to Grimsby. Many fishermen and vendors are already aware of the problem, discarding red whelks without landing them, and they are probably only sold inadvertently mixed with common whelks.

Guidance on identifying the different species of whelk has been issued to fishermen and the retail trade in previous years, and will be repeated at the beginning of the next season.

N M I Black, MFPHM MRCGP, Regional consultant in communicable disease control and environmental health, Northern Regional Health Authority; S J O’Brien, MB BS, Senior registrar in public health medicine, Northern Regional Health Authority and P Blain, MSc MB ChB, Senior registrar in public health medicine.

References

Influenza surveillance in England and Wales: November 1990 - June 1991

K J Fern, C A Joseph, J M Watson, P Chakraverty

This report summarises the available information on influenza activity in England and Wales from November 1990 - June 1991 (weeks 90/44 to 91/25). It also includes information on influenza activity in Scotland which has been made available by the Communicable Disease (Scotland) Unit. A description of the format and indices used was given in an earlier report1.

Laboratory reports

Influenza B

CDSC received 1941 reports of infections due to influenza B between weeks 90/44 and 91/25. Of these, 280 were diagnosed by isolation, 406 by a four-fold rise in antibody titre and 1255 by a single titre. The first isolate of influenza B virus was received in week 90/42 and reports of influenza B infections rose during the middle of December (week 90/50) to reach a peak during February (week 91/07) (Figure 2A). Increased activity continued, although at a declining rate, until mid-May (week 91/19).

During February a large outbreak occurred in a school in the English Midlands, when approximately 100 out of nearly 500 boarding pupils developed an influenza-like illness. The outbreak began in mid-January and influenza B virus (similar to the B/Yamagata/16/88 strain) was isolated from five of the affected pupils. A large proportion of the pupils had been immunised in 1989 and/or 1990 with a vaccine containing the B/Yamagata/16/88 component, and an unpublished study of vaccine efficacy indicated a protective effect of about 40%. Other outbreaks were reported in nursing homes (2), hospital wards (4), schools (8), a psychiatric unit, a camp for 16-19 year olds, and on a cruise-ship.

The age distribution of reported influenza B infections was similar to that seen in 1987/88, the last year of appreciable influenza B activity (Figure 2C). Rather more infections were reported in the age groups under 40 years. Figure 1 shows the geographical distribution of reported influenza B infections per 100,000 population. These figures should be interpreted with caution as diagnosis, testing and reporting rates vary between regions.

Influenza A

CDSC received 128 reports of influenza A infection between weeks 90/44 and 91/25. Two were diagnosed by isolation, three by a four-fold rise in antibody titre and 123 by a single titre. There was no identified peak of infection during this period (Figure 2B).

Virus Reference Laboratory

The PHLS Virus Reference Laboratory received and typed 263 influenza virus isolates between weeks 90/44 and 91/25 of which 260 were influenza B and three were influenza A. Two hundred and twenty-eight of the isolates were from England and Wales and the remaining 35 were from Scotland.

The isolates received from Scotland were all influenza type B. Of the 260 influenza B isolates, 59% of the strains were similar to B/Victoria/2/87 and 40% were similar to B/Yamagata/16/88. The strain B/Yamagata/16/88 was present in the 1990/91 vaccine. Two isolates of influenza A subtype H1N1, one of strain similar to A/England/427/88 and one similar to A/Beijing/353/89, were received. One isolate of influenza A subtype H3N2, similar to A/Victoria/36-43/88, was also received.

Spotter practice data

The Royal College of General Practitioners (RCGP) data2 on the new episode rate for 'epidemic influenza' showed a small rise between the end of December (week 90/51) and the end of February (week 91/09) (Figure 2D). The new episode rate rose from 7.8 in week 90/50 to 15.2 in week 90/51. It reached 36.9 in week 91/05 and had declined to 7.7 by week 91/11. This compares with the peak of 272.1 during the epidemic of the previous year. Since week 91/11 'epidemic influenza' has remained at low levels. Figure 2E shows a combined index for 'epidemic influenza' and 'influenza-like illness' and compares that with indices of influenza activity from spotter practice schemes organised by the CDSC Welsh Unit and the Communicable Disease (Scotland) Unit. The combined RCGP index suggests that influenza activity began around week 90/50, reached a peak in week 91/05 and then began to decline although increased activity was seen until around week 91/12.

The Welsh spotter practice data3 indicate that the epidemic in Wales occurred over a slightly shorter period of time but reached a sharper peak. Reports began to rise during mid-January (week 91/03) when the new episode rate per 100,000 population rose to 24.7 from 11.9 in the previous week. The peak occurred at the beginning of February (week 91/06) when the new episode rate reached 159, and then declined rapidly. The Scottish data1 show a more prolonged period of
activity which had already begun around the beginning of December, reached a peak at the end of February (week 91/09) when the rate reached 163 consultations per 100,000 population, and then declined rapidly (Figure 2E).

Respiratory syncytial virus (RSV) and Mycoplasma pneumoniae may also have contributed to the observed influenza-like illness during the winter. The annual epidemic of RSV reached a peak at the beginning of January (week 91/01). Reports of M. pneumoniae increased at the end of 1990 consistent with the four yearly epidemic, and laboratory reports continue to be received in high numbers.

OPCS weekly mortality data
The Office of Population Censuses and Surveys (OPCS) data for England and Wales show that total respiratory deaths (the combined total of deaths attributed to influenza, bronchitis and pneumonia) rose in mid-December (week 90/50) consistent with the usual annual rise in these deaths during the winter period (Figure 2F). Between weeks 91/06 and 91/11, however, the weekly figure exceeded the number seen in previous winters which lacked appreciable influenza activity. This coincided with the peak of influenza B infection reports. The changes observed are small by comparison with
those seen in years of significant influenza A activity. 1986/87 data are provided for comparison in figure 2F as this was a year of very limited influenza activity when there were estimated to be no excess respiratory deaths. CDSC have estimated the number of excess respiratory deaths for 1990/91 to be 3000. The weekly total of deaths from all causes exceeded the OPSC's 'expected' number between weeks 91/06 and 91/09 with a peak occurring at the same time as the peak of laboratory reports to CDSC of influenza B infections.

Other indices
The Medical Officers of Schools Association (MOSA) reported a small increase in the rate of influenza and influenza-like illness in pupils in the collaborating schools during late January and February (weeks 91/04 - 91/08).

Applications to the Emergency Bed Service (London) rose during December from a baseline of 400-500 and reached a peak of 769 in the first week of 1991. This compares with a peak of 942 occurring during the epidemic of the previous year. The number of applications remained above 500 until the end of March (week 91/12) when they began to drop back towards the baseline.

Influenza in other parts of the world

Northern hemisphere
Influenza activity throughout Europe and North America has been generally low during the 1990/91 season. Influenza B viruses were isolated most frequently. A few cases of influenza A were reported during the season, mainly of the H3N2 subtype.

Increases in influenza-like illness were reported from sentinel practice networks in the Eurosentinel scheme from France, Holland, Ireland and Spain: the peaks of reported activity, which were associated with influenza B infections, also occurred in February and early March. An epidemic occurred in Japan from January to March during which the influenza A subtype H3N2 was isolated most frequently.

Southern hemisphere
Influenza activity has also been quiet during the winter of 1991 (May to August) in the Southern hemisphere. Sporadic laboratory confirmed cases of influenza B have occurred in Australia. Outbreaks have been reported in the North and South islands of New Zealand. They have mostly been associated with influenza B, but influenza A subtype H3N2 has also been isolated. Two outbreaks of influenza A subtype H3N1 were reported in South Africa during July and August.

Vaccine recommendations
The World Health Organisation recommendations for the antigenic composition of influenza vaccine for the 1991/92 season, which have been adopted by the Department of Health, are that it should contain an A/Beijing/353/89 (H3N2)-like strain, an A/Singapore/6/86 (H1N1)-like strain, and either a B/Yamagata/16/88 or a B/Panama/45/90-like strain.

Comment
By comparison with the epidemic due to influenza A infection in 1989/90, the influenza season of 1990/91 was relatively quiet. Nevertheless an epidemic due to influenza B occurred with infections reported from all regions of the country: it began at the turn of the year, peaked in February and declined over the next two months. This epidemic was associated with outbreaks in a number of institutions and the associated illness was reported to be milder to moderate in severity. Laboratory reports may underestimate the size of the epidemic, in comparison to an influenza A epidemic, as fewer infected people may have consulted their doctors and fewer specimens may have been taken as a result of the generally milder illness. The peak of the epidemic was reflected in increases in reported rates of clinical influenza activity from the CDSC Welsh scheme and in the RCGP 'influenza-like illness' index. Similar increases were also seen in clinical reporting schemes in several European countries. However, very little change was seen in the RCGP 'epidemic influenza' index which is usually the most reliable of indicators of 'true' influenza activity in the community. This diagnostic label is generally reserved for the more severe forms of the illness and may not often have been considered appropriate for the illness associated with influenza B infection. The epidemics of RSV and M. pneumoniae infections may also have contributed to the increases observed in the other clinical indices. The observed excess of respiratory deaths was higher than would have been expected from calculations based on the 'epidemic influenza' index alone and was probably due to a combination of the epidemics of influenza B virus and M. pneumoniae.

Acknowledgements
We would like to thank the hospital and PHLS laboratories for their reports of infections, and the Birmingham Research Unit of the RCGP and the Communicable Diseases (Scotland) Unit for sharing their data.

References
3. Palmer SR, Smith RMM. GP surveillance of infections in Birmingham Research Unit of the RCGP and the Communicable Diseases (Scotland) Unit of the RCGP and the Communicable Diseases Surveillance Centre.

K J Fern BSc
CA Joseph MSc
J M Watson MRCP MFPHM
Respiratory Diseases Section
Communicable Disease Surveillance Centre
P Chakraverty PhD
Virus Reference Laboratory
Central Public Health Laboratory