



衛生防護中心
Centre for Health Protection

Scientific Committee on Enteric Infections and Foodborne Diseases

Foodborne illness – Intersection between Clinical and Public Health Approaches

Purpose

Foodborne infectious diseases are commonly encountered in primary care setting and emergency departments of hospitals. Occasionally, patients may need to be admitted to hospitals because of severe symptoms. With epidemiological investigations, some patients may be traced to the sources of infection which may need immediate public health control. Infectious sources which are persistent, of wide community implications or related to new or rare pathogens are especially of major public health concern.

2. This paper aims at updating the local epidemiology of foodborne diseases with highlights on the relevant management aspects, with a view to enhance collaboration between clinicians and public health practitioners, to achieve coordinated and effective management of not only the patients themselves, but also to minimize any potential public health impacts.

Scope

3. Foodborne diseases can be defined as diseases commonly transmitted through food (1). Foodborne diseases comprise a broad group of illnesses caused by microbial pathogens, parasites, chemical contaminants and biotoxins. At present, seven diseases have been included in the statutory notifiable diseases namely food poisoning, cholera, viral hepatitis (A and E), bacillary dysentery, typhoid fever,



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paratyphoid fever, and amoebic dysentery. Another three diseases including listeriosis, botulism and E. Coli O157:H7 infection have also put under the case-based surveillance of the Centre for Health Protection.

4. In clinical setting, these diseases may be presented in four major categories of clinical presentation. (Table 1) Acute gastroenteritis, presenting with any combinations of the key symptoms of abdominal pain, vomiting and diarrhea in at least two patients who shared a common meal, can be readily related to the suspicion of a food poisoning episode. Many bacterial agents as well as norovirus can cause acute gastroenteritis. Bloody diarrhoea may be the presentation of bacterial agents known to attack lower intestinal tracts through breaching of mucosal barriers. A number of bacterial and viral agents, such as *Salmonella* Typhi and *Listeria monocytogenes* are known to cause systemic illness and even fatal complications. Further cases can be prevented should the source of infection be identified and removed. Biochemical or chemical agents in food, such as ciguatoxin and pesticides, could cause neurological symptoms. Most patients may experience transient symptoms but fatal complications can occur.

Table 1. List of causative agents for foodborne diseases according to the clinical presentation.

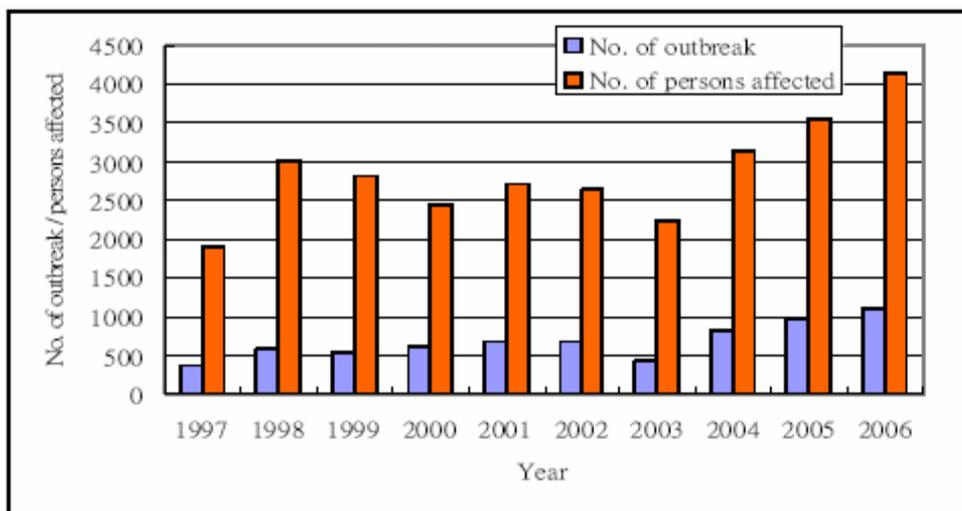
| Clinical presentation | Causative agents | |
|--------------------------|--|--|
| Acute gastroenteritis | <i>Vibrio parahaemolyticus</i> Non-typhoidal <i>Salmonella</i> spp. <i>Staphylococcus aureus</i> <i>Bacillus cereus</i> | <i>Vibrio cholerae</i> O1/ O139 <i>Clostridium perfringens</i> Norovirus |
| Bloody diarrhoea | <i>Shigella</i> spp. <i>Campylobacter</i> | <i>E. coli</i> O157: H7 <i>Entamoeba histolytica</i> |
| Systemic illness | <i>Salmonella</i> Typhi <i>Salmonella</i> Paratyphi <i>Listeria monocytogenes</i> | Hepatitis A virus Hepatitis E virus |
| Neurological involvement | <i>Clostridium botulinum</i> Ciguatoxin Shellfish poisoning Scombroid fish poisoning | Puffer fish poisoning Mushroom poisoning Clenbuterol Pesticides |

Local Epidemiology

5. Over the past decade, food poisoning has all along been the most commonly reported conditions among all foodborne illnesses under surveillance. Food poisoning is a heterogeneous entity that can be due to a wide range of different agents ranging from bacterial, viral or chemical causes. From 2004 to 2006, there were a total of 2888 outbreaks affecting 10818 persons

(Figure 1). The mean number of persons affected was 4 per outbreak (range 1 to 167, median 3). Among these 2888 outbreaks, bacteria account for the most common cause (76%), followed by viruses (12%), biotoxin (8%), chemicals (2%) and others (2%). Common bacteria include *Vibrio parahaemolyticus*, *Staphylococcus aureus* and *Salmonella non-typhi*.

Figure 1. Number of food poisoning reported to Department of Health, 1997-2006.



6. Among other statutorily notifiable diseases, hepatitis A and bacillary dysentery have demonstrated most markedly decrease in the previous decade. There were about 100 cases annually each (please refer to the left axis of Figure 2). Similarly, typhoid fever and cholera are on declining trends with 36 to 53 cases of typhoid fever and 1 to 7 cases of cholera each year in the past four years (please refer to the right axis of Figure 2). Similar to other developed world, hepatitis E and paratyphoid fever have become more important in recent years with about 40 cases per year in the last three years. (Figure 3) (2,3). The number of amoebic dysentery remains low except for a transient upsurge in 2004. There was no specific source identified.

Figure 2. Number of cases of bacillary dysentery, cholera, hepatitis A, and typhoid fever, reported to Department of Health, 1997-2006.

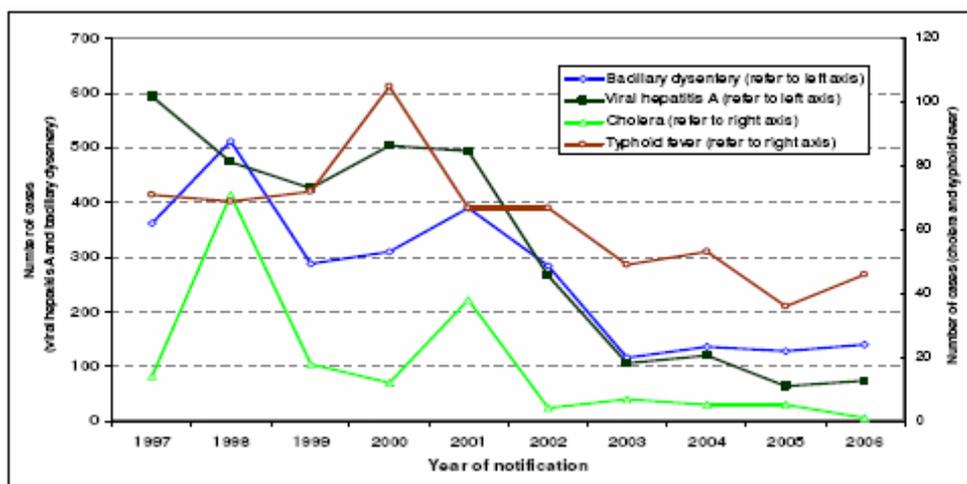
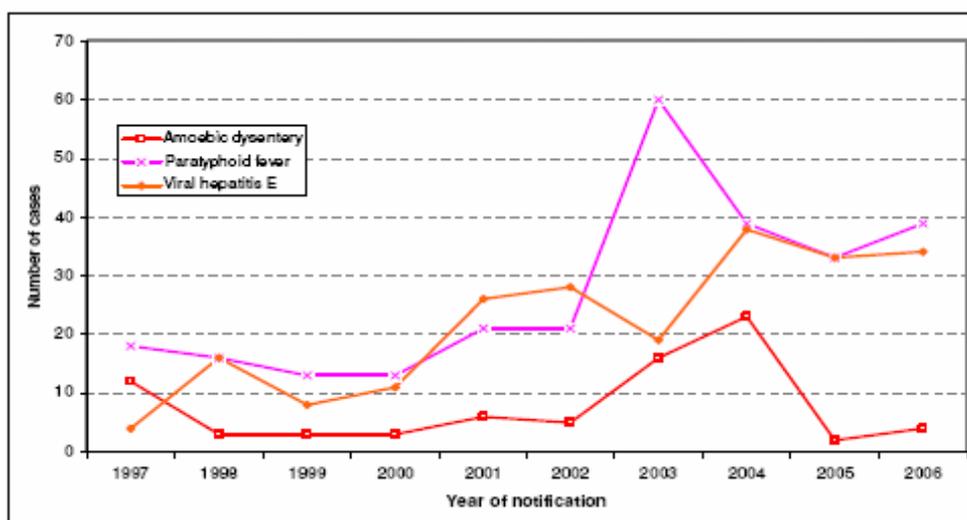
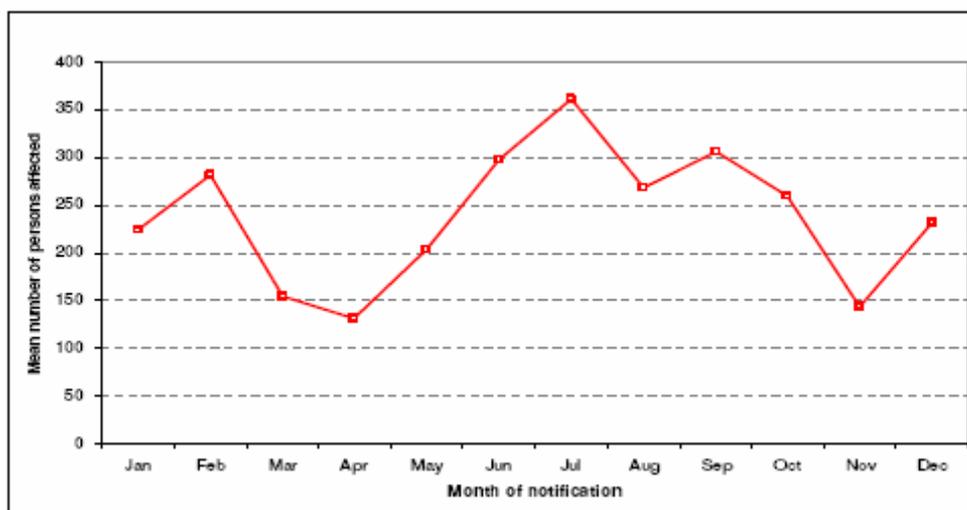


Figure 3. Number of cases of amoebic dysentery, paratyphoid fever, and viral hepatitis E, reported to Department of Health, 1997-2006.



7. Most foodborne illnesses demonstrate seasonal changes. Food poisoning outbreaks are more common in summer (June to September) and winter (December to February) months (Figure 4). The number of persons affected during the seasonal peaks is about double that reported during spring and autumn. Common bacteria causing food poisoning include *Salmonella spp*, *Vibrio parahaemolyticus* and *Staphylococcus aureus* grow more readily in summer months. Norovirus is another common causative agent which is more active in winter time.

Figure 4. Mean number of persons affected by month due to food poisoning, 1997-2006



8. For other diseases, bacillary dysentery, typhoid and paratyphoid fever are notably more common in summer months while more cases of hepatitis A and E are recorded during winter (Figure 5 and 6). There are few cases of cholera and amoebic dysentery reported each month (around one to two cases) and the seasonal patterns are less obvious with cases occurring all year round.

Figure 5. Summer peak of bacillary dysentery, paratyphoid fever, and typhoid fever demonstrated by mean number of cases by month, 1997-2006

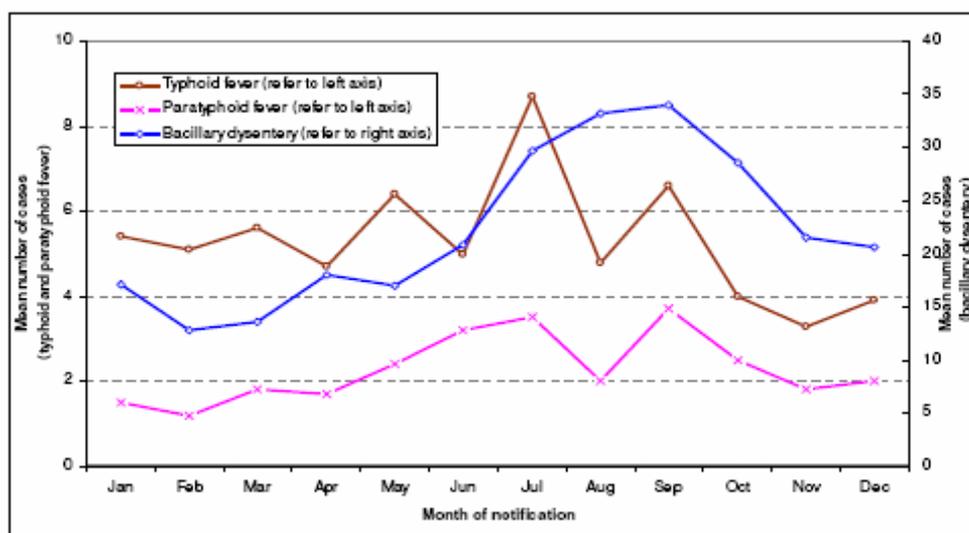
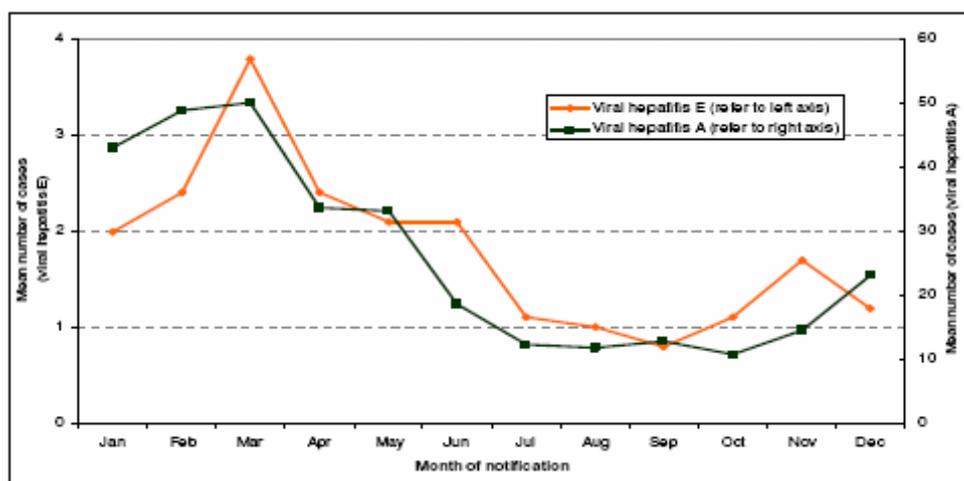


Figure 6. Winter peak of viral hepatitis A and E demonstrated by mean number of cases by month, 1997-2006



9. Most of the patients suffered from cholera, hepatitis A and E, typhoid fever, paratyphoid fever, and amoebic dysentery required admission to hospitals (Table 2). In contrast, only 5.7% (1622) patients affected by food poisoning required hospitalization.

Table 2. Proportion of patients suffering from notifiable foodborne diseases required hospitalization, 1997-2006

| Foodborne diseases | Number of patients required hospitalization (%) | Total number of patients |
|---------------------|---|--------------------------|
| Cholera | 160 (91.4%) | 175 |
| Typhoid fever | 572 (90.1%) | 175 |
| Paratyphoid fever | 233 (85.3%) | 273 |
| Hepatitis E | 168 (77.4%) | 217 |
| Amoebic dysentery | 49 (63.6%) | 77 |
| Hepatitis A | 1879 (60.1%) | 3127 |
| Bacillary dysentery | 1192 (44.7%) | 2669 |
| Food poisoning | 1622 (5.7%) | 28576 |

10. About 50% of all patients affected by food poisoning outbreaks are either with laboratory confirmation or are linked to a case with laboratory confirmation. Among these, the five most common agents encountered are *Vibrio parahaemolyticus*, non-typhoidal *Salmonella spp.*, norovirus, ciguatera fish poisoning, and *Staphylococcus aureus*. While *Vibrio parahaemolyticus* is the most common agent causing food poisoning outbreaks, patients affected by non-typhoidal *Salmonella* species, or biochemical food poisoning are more

likely to be admitted to hospitals. These may be related to more severe or alarming neurological symptoms associated with the latter.

Table 3. Causative agents accounting for food poisoning outbreaks in Hong Kong, 1997-2006

| Causative agents | Number of patients required hospitalization (%) | Total number of patients |
|--------------------------------------|---|--------------------------|
| <i>Vibrio parahaemolyticus</i> | 226 (4.5%) | 5074 |
| Non-typhoidal <i>Salmonella</i> spp. | 336 (10.4%) | 3218 |
| Norovirus | 34 (2.5%) | 1344 |
| Ciguatera fish poisoning | 84 (7.2%) | 1180 |
| <i>Staphylococcus aureus</i> | 58 (5.8%) | 1001 |
| Others | 81 (8.4%) | 962 |
| No laboratory confirmation | 803 (5.1%) | 15797 |
| Total | 1622 (5.7%) | 28576 |

11. In addition, the CHP has also recorded 20 sporadic cases of listeriosis and one case of *E. Coli* O157 from June 2004 to December 2006. Most of the patients with listeriosis were in the extremes of ages and 80% had chronic illnesses. Listeriosis is commonly associated with cheese and milk products although the source of incriminated food may be difficult to be traced in sporadic cases. In December 2006, a case *E. Coli* O157 was reported affecting a 16-month-child presented with persistent diarrhoea. Two household members of the child were also found to have asymptomatic infection. *E. Coli* O157 is commonly associated with undercooked beef and unpasteurized milk but the source of infection of the child could not be identified.

Diagnosing foodborne diseases

12. Diagnosis of foodborne diseases depends not only on the clinical presentations, but also a history of intake of relevant food, and the capability of the laboratory to detect the pathogen. Patients suffering from ciguatera fish poisoning usually have neurological involvement like limb numbness and may have characteristic sensation of heat/cold reversal. They may have consumed coral reef fish like grouper. Hepatitis A and E are typically presented with acute onset of fever, abdominal discomfort, tea-coloured urine and jaundice. Patients may acquire these infections through consumption of undercooked shellfish. Listeriosis is associated with cheese and unpasteurized milk while *E. Coli* O157 infection is related to undercooked beef. History of consumption of tuna fish may raise the suspicion of scombroid poisoning; raw egg and under-cooked chicken for non-typhoidal salmonellosis; and raw oyster for norovirus; fried

rice for *Bacillus cereus*.

13. While clinical management and public health control measures may be initiated before a definitive diagnosis is available, laboratory testing provides important clues in understanding epidemiology, identifying uncommon and rare pathogens or presentations, and assists in redefining and evaluating the control strategies. However, sometimes laboratory diagnosis may not be available if the appropriate specimens are not taken timely or when the appropriate testing medium has not been used. Moreover, laboratory diagnosis is of much importance should legal proceedings be intended. For example, *Vibro parahaemolyticus* may be cultured from patient's stool and the remnant of the incriminated food (e.g. shrimp) that the patient had taken from the food premises. This, together with other epidemiological investigation results, may serve as legal evidence to charge the food premises for providing food unfit for human consumption.

14. Most bacterial pathogens causing foodborne diseases can be diagnosed by stool culture. Some bacteria such as *E. Coli* O157 need special culture media. Typhoid fever may cause systematic illnesses and testing for the antibodies in serum may assist diagnosis. If parasite infection is suspected, fresh stool may be sent for examining ova and cyst for *Entamoeba histolytica*. Serology remains the standard test for hepatitis A and E while norovirus can be confirmed by polymerase chain reaction (PCR) for norovirus in stool or vomitus. Special test such as serum pseudocholinesterase level may be warranted if organophosphate poisoning is suspected. More details on the clinical, epidemiological and laboratory aspects of foodborne diseases have been summarized in the Annex.

The Public Health Aspect of Foodborne Diseases Management

15. While clinical care is of key importance to minimize morbidity and complication of the affected patients, there are some areas of patient management that are of concern from the public health perspective. We highlight the use of antibiotics, infection control as well as the epidemiological and laboratory investigations in the following paragraphs.

Use of antibiotics

16. Most episodes of gastroenteritis are self-limiting (4). Symptoms are usually mild and last for a few days only. Fluid and electrolyte replacement and other symptomatic treatment are sufficient for most cases. The choice of antibiotics, if indicated, should be based on clinical presentation, organism detected in clinical specimens and susceptibility test results. Judicious use of antibiotics is crucial in order not to further worsen the antibiotic resistance of enteric pathogens (4).

Infection control in clinical setting

17. Bacteria, virus and parasite may be excreted from infected patients who should be reminded to observe good personal hygiene. Adequate infection control measure should be implemented on all gastroenteritis cases (5). Patient should be nursed with standard precautions: handwashing; wearing gloves for contact with blood, excretions, secretions and contaminated items; eye protection and gown for splashes of blood, secretions and excretions (6). For patients with diarrhea, contact precautions may be considered (wearing gowns and gloves for contacting patients, preferably isolation in single room) (6).

18. Additional preventive measure against secondary person-to-person spread is necessary for patients suspected to be affected by norovirus. The virus is highly contagious and infection can be caused by as few as 100 particles (7). Apart from faecal-oral route, norovirus can also be transmitted through droplet from patients's vomitus. Delay or inappropriate management of patients may result in secondary spread in hospital setting or in the community. Prompt disinfection of the environment (including toilet used by patients or the environment soiled with patients' vomitus) using 1,000 ppm hypochlorite solution (one part of 5.25% hypochlorite solution added to 49 parts of water) is important to prevent nosocomial outbreak of norovirus (8). Patients suspected with norovirus should be isolated or cohorted until symptoms subsided for 48 hours (8).

19. Epidemiological and laboratory investigations of foodborne diseases 19. Ongoing surveillance on foodborne diseases can track the trend and epidemiology of enteric diseases. More importantly, early reporting of notifiable diseases assists rapid detection and control of outbreaks. Several diseases are statutory notifiable in Hong Kong including cholera, amoebic dysentery, bacillary dysentery, food poisoning, typhoid fever, paratyphoid fever, hepatitis A and E (reported as viral hepatitis). Some of these diseases often require laboratory investigation results to make the diagnoses. Nevertheless, if there is strong clinical suspicion, any rare or clusters of foodborne illnesses, could be reported to the Centre for Health Protection (CHP) for tracing the source of infection and prevention of further incidence.

20. Food poisoning generally refers to an incident in which two or more persons experience a similar illness after ingestion of a common food, and epidemiological analysis implicates the food as the source of the illness. Exceptions are one case of botulism, chemical poisoning or biochemical poisoning because of potential public health impact. It is advisable to ask the patients whether other family members, friends, schoolmates, colleagues or residents of elderly homes, have similar symptoms. Early notification of such incident, without waiting for the laboratory result, will facilitate public health actions to prevent further spread of the outbreak.

21. Upon notification, CHP will confirm the occurrence of the outbreak, determine the causative agent based on information including clinical symptoms, incubation period and type of food consumed; collect clinical specimens for laboratory investigation; and implementation of control measures. For instance, outbreaks suspected to be associated with food premises may be referred to the Centre for Food Safety (CFS) of Food and Environmental Department for further investigation and appropriate follow up action.

Conclusion

22. Foodborne diseases are common conditions encountered in both in-patient and out-patient setting. It is important to maintain high index of suspicion if patients presented compatible symptoms with history of relevant food intake. Early notification can assist tracing of incriminated food and prevent further spreading the diseases in the community.

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Reference

1. World Health Organization. WHO Consultation to Develop a Strategy to Estimate the Global Burden of Foodborne Diseases, 25-27 September 2006. Available at: http://www.who.int/foodsafety/publications/foodborne_disease/fbd_2006.pdf accessed on October 1, 2007.
2. Lewis H, Lawrence J. Recent increase in *S. Paratyphi A* phage type 1 and *S. Typhi Vi*-phage type E1 in England and Wales, associated with travel to the Indian subcontinent. *Euro Surveill* 2006;11(3):E060309.4. Available at: <http://www.eurosurveillance.org/ew/2006/060309.asp#4>
3. Hannah Lewis, Dilys Morgan, Elizabeth Boxall. Indigenous hepatitis E virus infection in England and Wales. *BMJ* 2006;332:1509-1510 Available at <http://www.bmj.com/cgi/content/full/332/7556/1509-b>
4. Centers for Diseases Control and Prevention, United States. Diagnosis and Management of Foodborne illnesses: A Primer for Physicians. *MMWR*, January 26, 2001 / Vol. 50 / No. RR-2
5. Hospital Authority Central Committee on Infectious Diseases, Hospital Authority, HKSAR. Fact Sheet on Cholera, revised on Oct 2005.
6. Hospital Authority Central Committee on Infectious Diseases, Hospital Authority, HKSAR. Fact Sheet on Typhoid and Paratyphoid Fever, Nov 2003.
7. Centers for Diseases Control and Prevention, United States. Norovirus in Healthcare Facilities Fact Sheet, December 2006.
8. Hospital Authority Central Committee on Infectious Diseases, Hospital Authority, HKSAR. Fact Sheet on Noroviurs associated Diarrhoea, revised on Jun 2005.
9. David L. Heymann. *Control of Communicable Diseases Manual*.
10. Working Group of the former PHLS Advisory Committee on Gastrointestinal Infections, Health Protection Agency, UK. Preventing person-to-person spread following gastrointestinal infections: guidelines for public health physicians and environmental health officers. *Communicable Disease and Public Health* 2004; 7(4): 362-384.
11. Food and Environmental Hygiene Department, HKSAR. Food Hygiene Code, Appendix V: Common and Important Types of Food Poisoning in Hong Kong. Available at:

http://www.fehd.gov.hk/publications/code/allc_ap5.htm#top accessed on October 9, 2007

Annex : Foodborne diseases due to different causative agents (4,9-11)

| Causative agent | Clinical presentation | Incubation period / latency of onset | Laboratory test | Associated foods |
|--|---|---|--|--|
| Bacteria | | | | |
| <i>Bacillus cereus</i> | Abdominal cramps, watery diarrhoea, | 10-16 hours | Stool culture | Meats, stews, gravies, vanilla sauce. |
| <i>Bacillus cereus</i> (performed toxin) | Sudden onset of severe nausea and vomiting. Diarrhoea may be present. | 1-6 hours | Stool culture | Improperly refrigerated cooked and fired rice, meat |
| <i>Campylobacter jejuni</i> | Diarrhoea which may be blood stained, abdominal pain, fever and vomiting. | 2-5 days | Stool culture; <i>Campylobacter</i> requires special media and atmosphere to grow. | Raw and undercooked poultry, unpasteurized milk, contaminated water |
| <i>Clostridium botulinum</i> | Diplopia, blurred vision, and bulbar weakness. Symmetric paralysis may progress rapidly. | Usually 12-36 hours | Stool culture Detection of botulinal toxin in serum or stool. | Home-canned vegetables and fruits foods; fermented, salted or smoked fish and meat products |
| <i>Clostridium perfringens</i> | Sudden onset of colic followed by diarrhoea; nausea is common, vomiting and fever are usually absent. | Usually 10-12 hours, range from 6 to 24 hours | Stool culture and detection of enterotoxin | Inadequately cooked or reheated meat and meat products (e.g. stew, meat pie, gravies made of beef or chicken). |
| E. Coli O157 | Acute bloody diarrhoea and abdominal cramps with little or no fever. May be complicated with haemolytic uraemic syndrome. | Range from 2-10 days, with a median of 3-4 days | Stool culture, requires special media to grow. Detection of Shiga toxin produced from culture isolates. | Undercooked beef, and unpasteurized milk. Others include raw fruits and vegetables, contaminated water |

| Causative agent | Clinical presentation | Incubation period / latency of onset | Laboratory test | Associated foods |
|--|--|--|---|---|
| <i>Listeria monocytogenes</i> | Fever, muscle aches, and sometimes nausea or diarrhoea. An invasive disease manifests most commonly as meningitis or septicaemia. Infection during pregnancy may result in abortion, premature delivery, stillbirth, or neonatal meningitis. | 9-48 hours for gastrointestinal symptoms; 2-6 weeks for invasive disease | Blood or cerebrospinal fluid culture. Stool culture may not be helpful in sporadic case because asymptomatic fecal carriage occurs. | Fresh soft cheeses, unpasteurized milk, inadequately pasteurized milk |
| Non-typhoidal <i>Salmonella spp.</i> | Diarrhoea, abdominal pain, vomiting and fever. | Usually about 12-36 hours, can range from 6-72 hours | Stool culture | Inadequately cooked meat and poultry. Contaminated raw egg and egg products, milk and milk products, foods contaminated by food handlers. |
| <i>Salmonella Typhi</i> (Typhoid fever) | Fever, headache, malaise, anorexia, relative bradycardia and splenomegaly, non-productive cough and rose spots on the trunk. Diarrhoea is uncommon and vomiting is not severe. | Usually 8-14 days, range from 3 to over 60 days | Blood, stool, or urine culture. Widal test may be helpful | Contaminated water and food, in particular contaminated shellfish, raw fruits and vegetables, and raw milk. |
| <i>Salmonella Paratyphi</i> (Paratyphoid fever) | Similar to typhoid fever but tends to be milder. | 1-10 days | Blood, stool, or urine culture. Widal test may be helpful | Contaminated water and food, in particular contaminated shellfish, raw fruits and vegetables, and raw milk. |

| Causative agent | Clinical presentation | Incubation period / latency of onset | Laboratory test | Associated foods |
|-----------------------------------|---|--|--|---|
| <i>Shigella spp.</i> | Abdominal cramps, fever and diarrhoea and stool typically contain blood and mucus. | Usually 1-3 days, range from 12 to 96 hours, up to 1 week for <i>S. dysenteriae</i> 1. | Stool culture | Usually person-to-person spread through food or water contaminated with human fecal material |
| <i>Staphylococcus aureus</i> | Sudden onset of severe nausea and vomiting, abdominal pain. Diarrhoea and fever may be present. | 1-6 hours | Stool culture Detection of staphylococcal enterotoxins in food remnants | Food contaminated by food handlers with skin infection or nasal carriers, especially those food involving manual handling and no reheating afterwards (e.g. sandwiches, cakes and “siu mei”, “lo mei”). |
| <i>Vibrio cholerae</i> O1 or O139 | Acute painless watery diarrhoea with or without vomiting. | Usually 2-3 days, range from a few hours to 5 days | Stool culture; <i>Vibrio cholerae</i> requires special media to grow and confirmation by Public Health Laboratory Services Branch of CHP | Contaminated water and food such as raw or undercooked seafood |
| <i>Vibrio parahaemolyticus</i> | Watery diarrhoea and abdominal pain. May also have nausea, vomiting and fever. | Usually 12-24 hours, can range from 4-30 hours | Stool culture | Inadequately cooked seafood (e.g. crab, shrimp, clams) or other food cross-contaminated by seafood. |

| Causative agent | Clinical presentation | Incubation period / latency of onset | Laboratory test | Associated foods |
|------------------------------|---|---|--|--|
| Virus | | | | |
| Hepatitis A | Fever, malaise, anorexia, nausea, abdominal discomfort, tea-colored urine and jaundice. | Usually 28-30 days but may range from 15-50 days. | Serum for IgM anti-HAV | Food and water contaminated by food handler, such as shellfish |
| Hepatitis E | Fever, malaise, anorexia, nausea, abdominal discomfort, tea-colored urine and jaundice. | Usually 26-42 days, range from 15-64 days | Serum for IgM anti-HEV or HEV by polymerase chain reaction (PCR) | Contaminated water and food |
| Norovirus | Usually symptoms of nausea, vomiting, diarrhoea, abdominal pain, low-grade fever and malaise. | Usually 24-48 hours, range from 10-50 hours | Stool for norovirus by RT-PCR | Shellfish such as oyster, faecally contaminated foods. |
| Parasite | | | | |
| <i>Entamoeba histolytica</i> | Diarrhoea (may be bloody), frequent bowel movement, lower abdominal pain | Usually 2-4 weeks, can be as short as 2-3 days | Examination of stool for cysts and trophozoites. Demonstration of trophozoites in tissue biopsy or ulcer scrapings by culture or histopathology. | Contaminated food and drinking water |

| Causative agent | Clinical presentation | Incubation period / latency of onset | Laboratory test | Associated foods |
|--------------------------------------|---|--|--|---|
| Biochemical | | | | |
| Ciguatera toxin | Numbness in limbs, face, tongue or perioral area, heat /cold reversal. May have gastrointestinal symptoms include diarrhoea, abdominal pain, nausea and vomiting. | Usually 1-6 hours, may be up to 36 hours | Detection of toxin in fish | Large coral reef fish such as a variety of grouper, hump head wrasse and black fin red snapper. |
| Mushroom poisoning | Vomiting, diarrhoea, confusion, visual disturbance, salivation, diaphoresis, hallucinations, disulfiram-like reaction, confusion, visual disturbance | <2 hours | Mushroom identification and/ or demonstration of the toxin | Wild mushrooms |
| Puffer fish (Tetrodotoxin) poisoning | Paraesthesias, vomiting, diarrhoea, abdominal pain, ascending paralysis, respiratory failure | <30 minutes | Detection of tetrodotoxin in fish | Puffer fish |
| Scombroid (histamine) poisoning | Flushing, rash, burning sensation of skin mouth throat, dizziness, urticaria, paraesthesias | 1 min -3 hours | Detection of histamine level in food | Fish include Tuna, mackerel, marlin, escolar and mahi mahi |

| Causative agent | Clinical presentation | Incubation period / latency of onset | Laboratory test | Associated foods |
|---|---|--|---|--|
| Shellfish toxins (Paralytic shellfish poisoning) | Diarrhoea, nausea, vomiting; paresthesias of mouth, lips, weakness, dysphasia, dysphonia, respiratory paralysis | 30 minutes -3 hours | Detection of toxin in shellfish/food | A variety of bivalve shellfish, primarily scallops, mussels and oysters. |
| Shellfish poisoning (Diarrhoeic shellfish poisoning) | Nausea, vomiting, diarrhoea, and abdominal pain accompanied by chills, headache and fever. | 30 minutes to 2 hours | | |
| Shellfish poisoning (Neurotoxic shellfish poisoning) | Tingling and numbness of lips, tongue, and throat, muscular aches, dizziness, hot/cold reversal, diarrhoea and vomiting. | Few minutes to hours | | |
| Shellfish poisoning (amnesic shellfish poisoning) | Vomiting, diarrhoea, abdominal pain and neurological problems such as confusion, memory loss, disorientation, seizure and coma. | 24-48 hours | | |
| Chemical | | | | |
| Clenbuterol poisoning | Tremor, palpitation, headache, nausea, weakness, dizziness and nervousness | Usually about 1 hour, range from 10 minutes to 8 hours | Detection of clenbuterol in urine or food remnant | Clenbuterol contained pig's offal or pork |

| Causative agent | Clinical presentation | Incubation period / latency of onset | Laboratory test | Associated foods |
|---|--|---|--|---|
| Pesticides (organophosphates) poisoning | Numbness, weakness, dizziness, abdominal pain, nausea and vomiting | 4-12 hours, can happen within few minutes | Serum pseudocholinesterase level may be lowered. Detection of organophosphates in food remnant | Inadequately soaked or rinsed contaminated leafy vegetables |