# 1 Globalization and epidemiology of foodborne disease

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## 1.1 Introduction

Infectious and toxigenic pathogens transmitted through food have been recognized for over 100 years. By the 1950s, the main pathogens of concern in the UK and the US were *Salmonella*, *Staphylococcus aureus* and *Clostridium perfringens*. Botulism had also been understood as a dangerous disease related only rarely to commercially canned food, home canning of vegetables, or associated with traditional marine mammal products in the Arctic. Therefore, for most public health officials, foodborne disease, or food poisoning as it was called then, was generally considered to be an inconvenience for a day or two, and more of a nuisance than a threat to life. Not much was known in other countries because of a lack of any systematic reporting program. In fact, there was little interest or research being carried out on acute foodborne disease agents. We knew from outbreaks that most of the situations could have been avoided if there was proper time and temperature handling and storage of food, especially meat and poultry. It seemed that once staff in foodservice establishments became better educated, these problems with *Clostridium, Salmonella*, and *Staphylococcus* would resolve themselves. However, by the time that the 1980s came in, we were beginning to be a little more concerned with agents like *Campylobacter*, *E. coli* O157:H7, *Listeria monocytogenes* and *Yersinia enterocolitica*. However, it took several years for health authorities to recognize that these had the potential to cause serious complications or death, could be transmitted by a variety of products, and that there were limited control mechanisms in place to reduce such foodborne disease. Large outbreaks in the US arising from *Listeria monocytogenes* in 1985 and *E. coli* O157 in 1993 resulted in changes to food safety policy in the US and other countries, specifically aimed at these organisms. Now, it is recognized that a variety of pathogens in many different types of foods can cause illnesses that may be life-threatening. These include *Cyclospora*, *Cryptosporidium, E. coli* O157 and other shigatoxin/verotoxin-producing *E. coli*, multidrug resistant *Salmonella*, *Shigella* and small round structured viruses (SRSV), mainly norovirus, in produce, dairy products, eggs, ice

cream, and shellfish. Current surveillance systems are only capable of detecting a few of these pathogens. However, with DNA typing systems, like PulseNet, more interstate and international outbreaks are being detected.

## 1.2   Globalization of foodborne disease

There appears to be a general increase – or at least a plateauing – for foodborne disease cases throughout the world, even though new regulations and educational strategies are being adopted nationally and internationally. One of the reasons for this is that surveillance of foodborne and waterborne disease has been very limited in its ability to detect cases other than small clusters of ill persons in the same general geographic area. The traditional passive system of letting outbreak reports be sent to a central source in a very few countries has been the source of our knowledge for decades, but it is far from adequate. Outbreaks tend to be only investigated and written up if they are large enough. Many household illnesses are never documented. Therefore, we are more familiar with mass catering or restaurant outbreaks or those involving a well-publicized processed product. Even with these limitations, we have learned much about the types of implicated foods for which we can anticipate problems even if we are not ready to initiate targeted control programs.

The globalization of the food supply is another issue that can increase the risk of foodborne disease. Changes in farming practices, with larger operations and faster throughput, the drive to increase profit by recycling all animal materials, and the difficulty in disposing of manure, all lead to the increased likelihood of contamination of raw animal products. There has been much more intensive rearing of animals, which allows transmission of pathogens even if the animals themselves are not affected. Treatment of flocks and herds with antibiotics is primarily for growth enhancement but extensive applications have led to increased antimicrobial resistance in the gut flora of animals, and combined with inappropriate use for self-treatment of human infections is increasingly a problem for the population worldwide.

Environmental sources of contamination, such as rodents in barns and gulls in fields, are being recognized as important links in the transmission chain in zoonotic diseases. New varieties of strains appear in human cases, but may originate in environmental or animal-raising conditions where genes can be transferred from one organism to another. Large-scale aquaculture is another rapidly-expanding industry in many parts of the world where fish and shellfish are raised in close proximity to contamination sources and are prone to carry enteric viruses or bacterial pathogens, as well as seafood toxins if they are present in the aquaculture areas. We have also moved rapidly from local manufacturers producing our food to national industries and now international trade with wholesalers packaging products in ways appealing to local populations. The larger market size and wide geographic distribution of products means that, if problems occur, many people are at risk and extremely large outbreaks with thousands of cases have occurred. One example is the 2011 *Escherichia coli* O104:H4 outbreak with more than 4000 cases and 50 deaths; these were mainly in Germany but many people, including visitors to Germany, were affected in another 15 nations. The source was eventually tracked to fenugreek seeds from Egypt sprouted in Germany but not before the Spanish cucumber industry was largely destroyed over the outbreak period as it was initially fingered as the most likely vehicle for the pathogen's transmission. This illustrates how trace-back of a product to its source becomes difficult, especially if the originating company is in a foreign country.

Immigrants and travelers introduce people to new types of foods, and the public today wants a wide choice of products to eat. This leads to many companies trying out new

products or modifying traditional ones, with each step a potential for the introduction or growth of a pathogen. The consumer wants more varieties of food and in packages that require the minimum of preparation. This means that many types of ready-to-eat (RTE) packaged food may be stored in a refrigerator for long periods of time. This allows various organisms, such as *Listeria*, *Yersinia*, and *C. botulinum* type E, if present, to grow slowly to reach numbers that can cause illness. Therefore, it is perhaps not surprising that we see an increase in foodborne disease throughout the world, especially as we are living in an age with a rapidly growing and migrating society. The demographics of the population are also changing due to our ability to live longer and improved public health services and medical treatment. However, as a result of this there is an increase in the immunocompromised group, especially those who are aging. These people are at risk from infections by opportunistic pathogens, for which we have no or limited surveillance mechanisms and laboratory capabilities to detect.

## 1.3 Measuring the impact of the burden of foodborne disease

Ways to improve the surveillance system include linking newer epidemiological approaches with molecular fingerprinting of clinical and food isolates. In the US and many other countries around the world, the PulseNet laboratory typing of strains by molecular techniques is possible by comparing isolates from across the country to look for identical strains and possibly detect common sources. The FoodNet program in several sites allows detailed investigative studies in consumer food hygiene and physician diagnostic practices, food preferences, and case-control studies when illnesses have occurred. For each outbreak, there is much information that can be gathered in if the incriminated food is available. Counts of pathogens in food, for example, are useful for determining doses and quantitative risk assessments. Since we know that surveillance data are very much underestimated, more realistic figures can be made, even if they are not precise, such as the recently estimated 48 million foodborne cases in the US (range, 28.7–71.1 million). From these, the economic burden of disease can be estimated. Although these are crude figures, they put the costs of foodborne disease and potential control measures into perspective. For instance, in the US the cost of six foodborne bacterial diseases alone totalled US $2.9–$6.7 billion in the 1990s, excluding industry losses and legal suits; these values would much higher today. Surveillance must not only be national but also continental and eventually global. Enter-Net (formerly Salm-Net) allows tracking of *Salmonella* and other enteric pathogen isolates throughout Europe and a few other countries. There is logic for blocks of free-trade countries to compare systems and eventually adopt a common one, not to rapidly track international outbreaks and stop them, but to learn from the experience and understand the factors that led to them occurring.

The World Health Organization (WHO) has established a Foodborne Disease Burden Epidemiology Reference Group (FERG) which is charged to:

- assemble, appraise and report on the current, the projected as well as the averted burden of foodborne disease estimates, with the burden expressed in Disability-Adjusted Life Years (DALYs);
- conduct epidemiological reviews for mortality, morbidity and disability in each of the major foodborne diseases;

- provide models for the estimation of the burden of foodborne disease where data are lacking;
- develop cause attribution models to estimate the proportion of diseases that are foodborne; and, most importantly
- use the FERG models to develop user-friendly tools for burden of foodborne disease studies at country level.

Data generated from these will allow prioritization of food-pathogen safety issues for future research, prevention and control measures.

## 1.4 Investigation of foodborne disease outbreaks

To determine the agent and vehicle for a outbreak, an epidemiological investigation is carried out through epidemiological associations and laboratory analysis. Awareness of a foodborne disease outbreak by public health authorities is triggered by: consumer complaints; government laboratory-based surveillance for reportable diseases, food monitoring programs; and industry alerts and recalls. However, by the time an investigation is under way, many people may be exposed to the contaminated product and, in some cases, the outbreak may be over. Since most alerts start at the local level with one or two complaints, the appropriate agency has to make a decision when to assemble a team of experts to follow up with these. More often, one food or environmental health inspector will conduct telephone interviews with the complainant(s) and may visit any establishment(s) suspected to be the causative location by those who have fallen ill. In many cases, the last foodservice facility visited will be the prime suspect in the complainant's mind but, through judicious questioning, the inspector may identify other scenarios or consider there is no link between the illness and food. If further investigation is warranted, the inspector may request assistance to conduct more interviews and analyze food and environmental samples. For multiple reports of illness seemingly associated with one food, a case definition is desirable. Case definitions are defined based on: the characteristics of the illness, the pathogen, toxin or other agent, if known; symptoms typical for that agent; time range for when the illnesses occurred; the regional distribution of the cases; and any other pertinent laboratory or epidemiological data. It is important to locate the extent of the outbreak as soon as possible. This is done by: monitoring for more consumer complaints and checking emergency room records; reviewing surveillance and laboratory reports; and asking lab personnel and health officials in other jurisdictions to report any potential cases that fit the case definition even if they are not yet confirmed. At this stage, an epidemic curve will demonstrate the occurrence of a point-source outbreak (the food was all eaten in one time period) or one with a continuing source (exposure to a population over a longer period of time); plotting such a curve also indicates that cases have a short or long incubation periods.

By this time, it should be possible to generate a reasonable hypothesis about the causative agent and its source. Initially, any enteric infection can arise from exposure to food, water or a colonized/infected person or animal, or even touching surfaces that have been contaminated by the above. Before any food is suspected, drinking and recreational water uses and other activities such as visiting a petting zoo or playing with pets need to be ruled out. Initially, it may be difficult to formulate an hypothesis because of limited data. People do not remember all the details of their illnesses or what they ate and did days or weeks ago. It may be necessary to ask about food preferences, including what is typically or rarely eaten as

well as what they ate over seven days prior to their illnesses. Food ingredients, including spices, herbs and garnishes, are easily forgotten or never noticed at the time. It may be more time consuming to probe further, but checking foods in refrigerators, freezers and pantries, and reviewing shopper card purchases can yield useful supporting data.

Hypotheses can be tested by epidemiological means and laboratory analyses. All the obtained data must be reviewed to determine the probability that the illness came from one contaminated food source. The food consumed most frequently by the victims is a good place to start, although a contaminated ingredient may be in more than one food. Statistical approaches are used to link those ill with specific foods, e.g., food-specific attack rate tables, chi-square, odds ratio. A dose–response relationship may be worth following up if the quantities of food and beverages consumed are known (e.g., did those ill eat more of the suspect food?). Consider whether the distribution of potential food items matched the geographic spread of cases. Based on these data, ideas may be possible on the likely contamination points in the food chain, i.e., production, transportation, storage, preparation of food. Laboratory analyses of recovered food samples may be fruitful in isolating an agent. Ideally, the laboratories should be competent and certified, and use the appropriate culture methods to isolate the agents. Clinical and food isolates can be compared for similarity, with the likelihood enhanced if serotyping, phage typing or molecular typing methods are used. PulseNet comparisons are normally done at the state/provincial and national level. However, confirmation by laboratory methods is not always possible, because the sample may be wrongly selected or in inadequate amounts, contaminated by the producer or the consumer, or simply not available. Also, the laboratory staff may not have the required culture media or be sufficiently expert to isolate a pathogen or its toxin, even if it is present. Thus, it is not always possible to categorically state the cause of an outbreak even though all the epidemiological findings clearly point to foodborne transmission. In fact, investigators identify a specific food as the source of illness in only about half of the foodborne disease outbreaks in the US and other countries. If the outbreak has apparently ended (no more cases are reported), the source of the outbreak is declared as unknown. However, if indications of illnesses are still occurring, investigators must keep gathering information and studying results to find the food that is causing the illnesses and remove it from the market. This may require an environmental assessment to discover how the food was contaminated.

An environmental assessment could point to one food facility or multiple food facilities. In the former case, contamination was likely in that facility or during transportation to it, where employees, kitchens, and environmental sources would be investigated in more detail. The investigation of ill and well persons and the facility, e.g., restaurant or retail store, will narrow likely sources of contaminated food. In a commercial establishment, a history of regulatory compliance and violations may give clues to one or more contamination scenarios. Where more than one facility has been implicated, the contamination probably occurred earlier in the food production chain and this would require a trace-back approach, a more complex form of investigation. This requires checking on the food suppliers and the delivery systems. The further back in the food chain, the more likely other facilities will have received the contaminated food and a trace-forward is then required. Interstate/provincial sources and imported food may have to be investigated. If the contamination source is close to the beginning of the food chain, many agencies and even countries may be involved (multi-state/multi-province/international outbreak). The environmental assessment report will recommend control procedures to stop the outbreak, e.g., recalls and embargoes, and recommend prevention strategies to prevent future contamination. The source trace-back should suggest ways to control the outbreak and prevent similar outbreaks from happening in the

future. In some larger outbreaks, the authorities all the way up to the national level are familiar with details of the outbreak because they have been involved in the overall investigation. However, many outbreaks are small and local but, since every good investigation yields important information on causes and trends, they should be documented. Thus, even small outbreaks should be written up in a standardized format and copies sent to state/provincial or federal/national authorities for record keeping. Analyses of these over time will indicate how well existing prevention and control programs are working, and point to priorities for reducing food contamination and thus foodborne disease.

## 1.5 Vehicles frequently implicated in foodborne illness

In this section on foods associated with outbreaks, some recent but also some older ones are briefly described to show that many of these types of problems have occurred over decades. They also reflect a wide range of scenarios that may be encountered in any country. Zoonotic diseases are the most difficult to control because there are many entry points for the pathogens into the food chain. In contrast, there is a better chance of reducing foodborne diseases spread by human carriers such as *Staphylococcus aureus* where an organism must come from a food worker and grow to large numbers before the enterotoxin is produced. In fact, there has been a reduction in staphyloccocal food poisonings in the UK, the US and Canada. It is more difficult to prevent enteric virus transmission because the infectious dose is very low and no growth time is necessary, but better hygiene by food workers and restricting infected workers to non-food areas will help keep the contamination rate down. Food workers, knowingly or asymptomatically, have caused large outbreaks, particularly from norovirus and *Salmonella*. There are some more recent trends where the detection of multistate outbreaks involving a widespread distribution of a contaminated product is now feasible with molecular typing techniques even though the number of cases may be low in any one state or region.

### 1.5.1 Meat and poultry

It has been recognized for many decades that *Salmonella* has been transmitted by meat and poultry. Typically, limited food safety knowledge has led to undercooking, improper cooling, or cross-contamination. The following are some examples. In 1998, over 50% of guests at a church supper in Maryland were ill after eating ham (750 cases and one death). The hams had been packed too tightly into cooking pots, creating uneven cooking conditions. Then they were packed too tightly into the freezer, which prevented rapid cooling and gave the salmonellae present an opportunity to grow. One slicer was used to cut all the hams without any sanitation between the slicing process, thus allowing the transmission of salmonellae onto many of the ham pieces served. In Alaska in 1992, leftover improperly cooked roast pig was taken home by some picnic goers and only those who microwaved the meat to reheat it (10) versus those who used an oven or skillet for the same purpose (20) were infected. Microwaving was also an issue in 2007 when consumption of commercially sold pot pies caused 425 cases of salmonellosis in a multi-state outbreak because the labeling was insufficiently clear to indicate that some of the ingredients were uncooked, and the microwave reheating process used was insufficient to kill the *Salmonella*. In Texas, from May to October 1995, 59 persons were ill from *S*. Agona infection. All those who lived in

San Antonio and Houston had eaten food in a San Antonio Mexican restaurant. The same pulse field gel electrophoresis (PFGE) strain was isolated from machacado (air-dried raw beef), which had been shredded in a blender and cross-contaminated other foods such as salsa. Austin residents had eaten machacado at a similar Mexican restaurant. Both restaurants had purchased the beef from the same supplier in Corpus Christi. Without PFGE typing of isolates, these outbreaks would not have been detected. Poultry still causes many outbreaks including ones with widely distributed products. For instance, in 2011, there were two US multi-state outbreaks involving turkey (124 cases in 34 states of *Salmonella* Heidelberg from ground turkey, and 12 cases in six states of *S.* Hadar from turkey burgers).

Acute illnesses are not the only problems arising from salmonellosis and other enteric diseases. For example, in 1984, 423 provincial police officers were infected with *S.* Typhimurium after eating contaminated meat sandwiches provided to them as they lined a route in Ontario, Canada, for the Pope's visit; 6.4% had acute reactive arthritis (RA) with one third resolving within four months, and the majority continued to have mild symptoms in their joints for the following five years. Four had sufficient damage to force them to change jobs. The risk factors identified were severe diarrhoea at the time of the outbreak and presence of specific genes that coded for HLA-B27 and HLA-CREG antigens. In an Australian outbreak in March 1997, of those ill after eating pork rolls produced by a bakery, two died and 3% developed RA. One half of those with arthritis (1.5%) continued to have it 12 months later. In another outbreak in August from the same product, 5% developed RA. The difference in the percentage affected may be due to the ethnic background of those ill who may have a differing proportion of susceptible genes (Vietnamese in March and Cambodian in August). Sequellae such as RA are more frequent and are more long lasting than previously recognized.

Multidrug-resistant *Salmonella enterica* serotype Typhimurium definitive phage type 104 (DT 104) has emerged during the last decade as a global health problem because of its association with animal and human disease. Multidrug-resistant strains of this phage type were first identified from exotic birds in the UK in the early 1980s and in cattle and humans in the late 1980s but have since become common in other animal species such as poultry, pigs and sheep. Since 1991, this phage type has been second only to *S.* Enteritidis phage type 4 as the principal agent of human salmonellosis in the UK. The DT 104 epidemic has now spread globally, with several outbreaks since 1996 in the US and Canada. No effective treatment of infected animals has yet been found. In humans, there is a relatively high mortality rate of 3%, especially in the elderly. It is spread from farm to farm by water and is difficult to eradicate since it survives well in both dry and wet environments. It has been found in beef, pork, salami, chicken and cereals, and foodborne disease outbreaks have been associated with hamburgers, sausages and sausage rolls. This pathogen can be present in poultry flocks and other meat animals and extend down the food chain from farm to fork. Because of its ability to colonize many farm animals and be in the environment, its impact will likely be worse than most other serovars including *Salmonella* Enteritidis worldwide.

*Campylobacter* has been associated in outbreaks and epidemiological studies with undercooked chicken and meat, as well as from other sources. A case-control study in four cities in New Zealand bears this out. Risk factors were: raw or undercooked chicken; any chicken prepared in a sit-down restaurant; unpasteurized milk or cream; overseas travel; rain water; contact with puppies and calves. Barbecued or fried chicken was associated with illness whereas baked or roasted chicken was protective, although the reason for this was not determined, unless the former was more typically undercooked. Although cases of sporadic campylobacteriosis are frequent, large *Campylobacter* outbreaks from poultry or meat are

relatively rare. In the following example, chicken was the source of the pathogen, but it was other foods that caused the illness through cross-contamination. In 1996, at least 14 patrons of a restaurant in Oklahoma were ill with campylobacteriosis after eating lasagna and lettuce. Raw chicken had been cut up on a cutting board and the lettuce and lasagna contaminated by the cook's unwashed hands, utensils or counter top. Because the infectious dose of *Campylobacter* is low (500 cells), one drop of chicken exudate can be enough to infect a consumer. The workers had frequently used the same towel to dry their hands. The investigation emphasized the importance of clean up and sanitation of preparation areas and of handwashing facilities. In 2010 in Scotland, a chicken liver paté was improperly cooked and caused 24 cases of *Campylobacter* gastroenteritis at a wedding reception. The raw chicken livers were mixed with a red wine reduction and raw eggs, heated using a bain marie (water bath) to a core temperature of 65 °C and then immediately removed from the oven and cooled for 15 minutes. According to the UK Food Standards Agency advice, if liver is cooked at 65 °C, it should be held at this temperature for at least ten minutes to ensure adequate cooking.

   *E. coli* O157:H7 was first identified as a foodborne pathogen in 1982 from two outbreaks involving hamburgers served in fast food restaurants of the same chain in Michigan and Oregon. Reports of other outbreaks followed with undercooked ground meat as the most frequently-associated vehicle. By 1985, the most serious outbreak then reported occurred in a London, Ontario, home for the aged where 70 were ill and 17 died after eating ham sandwiches probably contaminated by an infected food worker. The most significant US outbreak occurred in 1992–1993 in four western US states with a total of over 700 cases and four deaths. Hamburgers from a fast food chain had been insufficiently cooked. Many children were affected which caused sufficient concern for the government to make the pathogen an adulterant in ground beef so that no level could be tolerated. This rule did not, unfortunately, stop the outbreaks which could involve very large amounts of ground beef. For instance in 2002, contaminated ground beef was linked to 28 *E coli* O157:H7 infections in Colorado and six other states. The manufacturer recalled 18.6 million pounds of ground beef products that were produced at the processing plant identified as the source.

   The most deadly outbreak of *E. coli* O157:H7 to date occurred in Scotland in November and December 1996, with nearly 500 cases and 20 deaths, mainly elderly people. Those ill had eaten cold cooked meats, meat sandwiches and cooked steak in gravy prepared by the same butcher, and distributed to many locations including nursing homes. As a result of this episode, numerous changes in food hygiene were initiated in the UK. These recommended changes were not sufficient, however, to prevent the death of a child in Wales in 2005, when 157 persons, mainly schoolchildren, suffered from *E. coli* O157:H7 infection because school meals supplied by a caterer butcher were cross-contaminated through dirty equipment and carelessness by employees and management. These two outbreaks remain those with the largest number of cases in UK history. In 2011, 14 persons in five US states were infected with *E. coli* O157:H7 in Lebanon bologna; this product is a fermented, semi-dry sausage, similar to salami. Salamis and other sausages have caused *E. coli* (O157 and non-O157) outbreaks in Australia, Canada, the US and Europe. There are also risks to hunters; 11 persons were ill after eating home-made jerky prepared from a deer that had been shot. The hunters were not hygienic in storing or dressing the carcass and preserving the meat. *E. coli* O157:H7 was found in the patients, jerky, uncooked deer meat, a saw used on the carcass, deer hide and deer fecal pellets. *Salmonella* has also been implicated in sausage outbreaks. Since this pathogen in food is primarily derived from the feces of domestic animals, it is not surprising that

petting zoos have been the source of infections. One example is from North Carolina, when in 2005 82 visitors to a petting zoo at the North Carolina state fair were infected.

In late 1998, at least 100 people contracted listeriosis after eating hot dogs and other processed meat cold cuts in several US states. There were at least 20 deaths and miscarriages. *L. monocytogenes* in the processing facility had contaminated the already cooked hot dogs before packaging. In 2007 in the EU, *L. monocytogenes* was found rarely in various meat products – pork, 2.2%; red, mixed or unspecified meat, 2.5%; 3.0% in RTE broiler meat – with less than 1% of samples exceeding the 100 cfu/g limit. However, some countries did have samples much more frequently contaminated: Germany, Greece, Italy, Poland and Slovenia reported presence of *L. monocytogenes* in samples of 25 g in 11.0%, 20.7%, 13.6%, 62.9% and 16.7%, respectively. A European Food Safety Authority (EFSA) study showed that *L. monocytogenes* was still a problem because of poor practices in the use of slicing machines for meat products, inadequate storage temperatures, the lack of an effective HACCP system, and lack of education and training of food workers. Storage temperatures in retail and domestic refrigerators were found to vary significantly, especially for the domestic refrigerators. Despite more awareness, the largest recent outbreak involving listeriosis in a meat product was the Canadian Maple Leaf outbreak in 2008 when over 50 persons were infected and at least 22 died. The use of large commercial slicing machines that were difficult to clean was a major contributing factor, leading to biofilms and contamination of the sliced deli products. Many deli meat products were contaminated, including salami brands. In 2009–2010, a smaller listeriosis outbreak occurred in Ontario from a fermented salami product. These two outbreaks have led manufacturers to consider ways of limiting or preventing growth of *L. monocytogenes* in these types of products. One approach is the development of specific starter cultures with multiple organisms to prevent the growth of *Listeria* and spoilage organisms under a wide range of temperature conditions while maintaining a reducing atmosphere to preserve the traditional colour and flavour of the salami-type products.

*Clostridium perfringens* toxico-infections are relatively frequent, and it is the pathogen third-most frequent cause of foodborne illnesses in the US. Most of the outbreaks involve slow cooling of large quantities of stews and soups where the spores have a chance to germinate and outgrow to numbers sufficient to cause abdominal cramps and diarrhea. In an institution in England, 12 joints of lamb were thawed and roasted for five hours and stacked on a single tray to cool before refrigeration. The next day, some of the meat was removed from the refrigerator and sliced, and left at room temperature for several hours before being served. This was repeated the next day with the rest of the lamb; 12 staff and residents were ill with cramps and diarrhea after an incubation period of 9–16 hours. *Clostridium perfringens* was found in the stools of the cases and in the leftover lamb ($>10^5$/g). Improper cooling of the joints and storage periods at ambient temperatures allowed growth of the pathogen. In London in 2009, 93 guests at two weddings suffered *C. perfringens* gastroenteritis after eating either a curried chicken or lamb dish that had not been cooled sufficiently quickly by the caterer, or reheated properly later. Also in this case the food was not refrigerated during transportation between the caterer and the wedding reception sites, and the food worker preparing the items had fecally-contaminated hands.

Foodborne outbreaks caused by norovirus (previously called small round structured virus – SRSV or Norwalk-like virus – NLV) typically involve an infected food worker preparing RTE foods which may include meat and poultry but more often multi-ingredient foods like hors d'oeuvres. One example is a caterer with a norovirus infection who transmitted it to 67 people attending an international AIDS conference in Wales. The food handler, who had boned the cooked chicken with bare hands, had been ill two days earlier.

## 1.5.2 Fish and shellfish

Cholera remains a threat in much of the world with limited sanitation and potable water. The seventh pandemic began when cholera was first documented in Peru in 1991 and rapidly spread to other Latin American countries with millions of cases. *V. cholerae* O1 was found in water, plankton, sewage and seafood. Street-vended foods, crabs, shrimp, ceviche and beverages containing ice were all vehicles of transmission. The economic loss from lost markets and tourism, absence from work, medical care and the value of lives lost was high. In Hong Kong in May 1998, 34 cholera cases occurred after they had been to restaurants where hotpot (raw meat and seafood dipped into a communal boiling broth and eaten with a raw egg and sauces) was served. Live seafood from polluted water was probably the source of cholera, as found in a 1994 outbreak when tank water for fish and lobsters contained *Vibrio cholerae*. Cholera can also affect trade, as in Mozambique in 1997. The country had a yearly catch of 20,000 tonnes, with more than 50% exported to the EU. When cholera caused over 30,000 cases and 780 deaths starting in August of that year, the EU banned import of Mozambican fish, costing the country US$240 million.

*Vibrio parahaemolyticus* is a pathogen that occurs naturally in warm waters, including North America. It is resident year round in the Gulf of Mexico and increases in number during the summer months. One example of an outbreak in that region occurred in July 1998, when over 400 persons in five states were ill from *V. parahaemolyticus* from contaminated oysters harvested in Galveston Bay, Texas. The pathogen was identical to ones found in SE Asia, and probably came from dumped ballast water. Texas had a heat wave with no water running into the Bay and any discharge would stay in same area allowing oysters to be contaminated. Sales were stopped if counts were >10,000/g, but this did not contain the outbreak which continued until December. The same strain caused illnesses in September in Oyster Bay, Long Island, New York, affecting an oyster festival. The warmer water and less oxygen from fertilizer run-off may have allowed the vibrios to multiply. *V. parahaemolyticus* has spread to more northern waters in North America because of increasing seawater temperatures, causing illnesses, as illustrated by two examples. In July–August 1997, 209 persons were infected with *V. parahaemolyticus* on the US west coast from California to British Columbia after eating oysters and other shellfish. The water temperature then was 1–5 °C above normal. This was the first *V. parahaemolyticus* outbreak in Canada linked to food. Another outbreak occurred in 2005 in Juneau, Alaska, when cruise ship passengers ate locally-harvested oysters growing in waters of >15 °C. This outbreak was 1000 km further north than any other previous US outbreak. Although outbreaks from *V. parahaemolyticus* are rarer in Europe, an emergent virulent serotype (O3:K6) is being more frequently found there. In July 2004, a *V. parahaemolyticus* outbreak with this serotype occurred among guests at several weddings in Spain after they ate boiled crabs at a restaurant. The crabs had been imported live from the UK, processed under unhygienic conditions, and stored at room temperature for several hours before being eaten.

*Vibrio vulnificus* is also a warm-water pathogen, more frequently found in tropical regions, and occurs more with wound infections and sporadic food-associated cases rather than large outbreaks. In 1994 in Sweden, the summer was unusually hot and water temperatures reached 17 °C. That year *V. vulnificus* was first observed after a bather was infected. Since then, some summers have been just as warm and other cases have been reported, and one individual died. *Vibrio vulnificus* septicemias occur each year as a result of consuming oysters harvested in the Gulf of Mexico during the summer months when the water is warm enough to allow rapid growth of the pathogen. Illnesses and deaths have also been documented

in Singapore, and probably all populations in tropical areas that eat undercooked or raw seafood are at risk of infections from this species and other vibrios.

*E. coli* infections are rarely associated with fish or shellfish but one unusual outbreak in Japan illustrates that this is possible. In 1998, 62 cases *E. coli* O157:H7 infection were reported in four separate locations after they ate salted salmon roe distributed to many sushi shops. The salmon roe had been soaked in a liquid seasoning consisting of soy sauce (79.0%), water (14.0%), chemical seasoning (6.5%), synthetic sake (0.3%), and a fermented seasoning (0.2%); its salt content was equal to a 13% NaCl concentration. This salted salmon roe is a popular component of Japanese sushi. Research subsequently showed that the strains isolated from the roe and patients entered the viable but nonculturable (VNC) state in the high salt concentration. Yet they were capable of causing illness at a concentration of 0.73–1.5 MPN/10 g of the processed salmon roe. Previously, this traditional way of preparation was thought to be safe from pathogen growth or survival. The normal isolation methodology would not have been successful in recovering these strains. Unfortunately, the company was liquidated, and the owner was arrested on criminal charges.

*Listeria monocytogenes* can be present in marine waters especially if there is agricultural runoff or sewage effluent, and seafood products found to contain the organism may be recalled. However, listeriosis is only occasionally linked to seafood, as in New Zealand in 1992 when two women contracted the disease after eating smoked mussels, which resulted in the death of newborn twins. The contamination was traced to the processor; the product had been widely distributed and other cases were probably ill in England. In Sweden, nine persons suffered from listeriosis from June 1994 to June 1995 after they had eaten fermented or smoked rainbow trout produced by one manufacturer. In Canada in 1996, six cases were linked to imitation crab meat.

Improperly canned food is always at risk for botulism, whether from underprocessing or from post-process contamination. These are rare in recent years but holes in some cans of Alaska canned salmon resulted in two small outbreaks in England and Belgium respectively, killing a total of three persons and affecting trade not only in Alaska but also Canada. Stored uneviscerated fish in various countries has also led to botulism outbreaks, with one of the biggest being in Egypt in 1991 which resulted in at least 20 deaths from ingestion of locally-made *faseikh*. Although outbreaks from commercial food are relatively few today, foodborne botulism remains a serious threat, as illustrated by recent botulism episodes in Finland from inappropriately processed or stored fish products including vacuum-packed smoked whitefish. Botulism is also well-documented arising from consumption of fermented marine mammal parts in Inuit communities in Alaska and northern Canada, and fermented salmon eggs in northwest coast First Nations/Native American communities. Parasites have been found in shellfish, such as *Cryptosporidium* in oysters in Chesapeake Bay near river tributaries where there may be sewage discharge or farm runoff. No cryptosporidiosis cases have been linked to the ingestion of raw shellfish, but the potential exists.

Because, for most countries, much of the seafood eaten is imported (for the US it is over 50%), there are risks of contracting illnesses from pathogens in the originating country, like *L. monocytogenes* in the New Zealand mussels episode, *Clostridium botulinum* in canned salmon or tuna, or from *Salmonella* derived from aquaculture practices in developing countries. Therefore, a vigorous inspection program by importing countries should be in place. Preferably, this should be based on HACCP rather than random samples and microbiological testing.

Enteric viruses have long been associated with shellfish. In early 1988, the residents in the Shanghai area were offered clams from a newly-discovered bed. Unfortunately, many of

the fishing boats did not have adequate toilet facilities on board and partially treated sewage from shore communities was also entering the sea, allowing the hepatitis A virus to enter the harvesting area and be taken up by the filter-feeding clams. Most consumers prepared the clams by only scalding them with boiling water, which was not sufficient to kill the virus. As a result, nearly 300,000 estimated cases of hepatitis A occurred. It was reported that industrial production in the city fell by about 17% in February, largely because of the large number of ill workers.

Scombroid or histamine poisoning arises in certain types of fish (such as tuna, mackerel, bonito, marlin, mahi-mahi, anchovies and sardines) that were inadequately refrigerated or preserved after being caught and prone to spoilage where histidine is converted to histamine by bacterial action. Scombroid poisoning can be confused with allergic reactions to food because some of the symptoms are similar (rapid onset, flushing, headache). A scombroid outbreak with 15 cases occurred in Spain from a fresh tuna sold at a hypermarket in June 1994. Typical symptoms of facial flushing, headache, diarrhea, nausea and abdominal pain occurred 45 minutes after the tuna was eaten. Histamine was found in leftover tuna samples and in urine specimens from the cases. Such illnesses are not uncommon every year, caused not only by fresh or frozen fish but by canned fish like tuna. In August 2003, an outbreak of scombroid fish poisoning occurred at a retreat centre in California with 42 persons ill after eating escolar fish. Individuals who ate at least two ounces of fish were 1.5 times more likely to develop symptoms and more of them than those who ate less. Samples of fish contained markedly elevated histamine levels (from 2000 to 3800 ppm). This was one of the largest reported outbreaks of scombroid fish poisoning in the US and was associated with a rare vehicle for scombroid fish poisoning, escolar.

Seafood toxins are found locally in molluscs and fish throughout the world, and illnesses frequently occur but are not always documented. Most of these originate from naturally toxic plankton that are ingested by shellfish, and include paralytic shellfish poison (saxitoxins and related toxins), diarrhoetic shellfish poison (okadaic acid and dinophysistoxins), and amnesic shellfish poison (domoic acid). The first two affect fisheries in the Americas, Europe, Asia and many tropical countries. Once illnesses occur or levels of toxins reach a certain limit, harvesting stops until the toxin level in the shellfish is below the limit or the toxic algae are no longer abundant – but not all countries have monitoring programs. PSP shellfish contamination regularly occurs every year on the northern west and east coasts of Canada and the US because of the presence of *Alexandrium* blooms, and illnesses are sometimes reported from recreational harvesters. In contrast, DSP poisoning has rarely occurred in North America, despite the presence of low levels of the causative *Dinophysis* dinoflagellate. Two small outbreaks affected those eating mussels in Nova Scotia and Newfoundland in the early 1990s, and there have been several reports of sporadic cases from the east coast. In 1997, the first incident of diarrhoetic shellfish poisoning (DSP) was recorded in the UK with 49 cases that may have been caused by water temperature changes. More significantly, in the summer of 2011 over 60 persons were ill after eating mussels harvested in British Columbia and shipped to four other provinces. It is not known what caused that *Dinophysis* bloom though water temperatures were high. The outbreak was quickly contained through a recall by the Canadian Food Inspection Agency, but this outbreak raises the question of the safety of local aquaculture industry, since this may be a recurring problem. Usually in DSP outbreaks, the amounts of toxin present are not known, but this was determined in 2009, following 11 outbreaks of DSP poisoning involving 45 individuals who had consumed mussels harvested in northwestern France. The incriminated batch was still available and

consumption data were known. Both okadaic acid (OA) and DTX-3 (closely related to OA) were present. The batch of source mussels contained 1261 µg OA equivalents/kg shellfish, eight times higher than the European regulatory limit of 160 µg OA eq/kg shell-fish flesh. This analysis also supported the lowest observable adverse effects level (LOAEL) to be 50 µg OA eq per person.

Domoic acid is found in shellfish worldwide, but has only caused one outbreak. This was in Canada in 1987, when contaminated mussels affected at least 107 persons, several of whom were left with permanent memory damage, and caused three deaths. As for PSP and DSP, monitoring programs are in place for domoic acid in many countries, either in shellfish or for the phytoplankton themselves. A more recent biotoxin is azaspiracid, which was first identified after an outbreak in the Netherlands caused by Irish mussels in 1995, and over 20 different analogs of the polyether compound have since been identified. The dinoflagellate responsible, *Azadinium spinosum*, was discovered only recently in 2009. Azaspiracids not only cause gastroenteritis but may have carcinogenic properties, which is giving concern to regulators. Ciguatoxin is a seafood toxin present in many varieties of tropical fish but currently there are no routine diagnostic tests to detect it. Although its effects are mainly in regions where these fish are caught, consumption of exported fish in temperate regions has resulted in several outbreaks. For instance, in Texas, 17 crew members of a Norwegian ship suffered from ciguatera poisoning after eating a barracuda caught off the Bahamas, and medical help had to be sought. There is some evidence that toxic plankton are spreading throughout the world, though climate change affecting water temperatures, ballast water being dumped in harbors or increased aquaculture. This may have happened in the Hong Kong area recently, when the causative organism of ciguatera poisoning, *Gambierdiscus toxicus*, was found for the first time in 1998. That same year, at least 71 suspected ciguatera cases occurred after they ate tiger grouper; most were hospitalized briefly. Authorities warned people not to eat fish larger than 1.5 kg. Illnesses typically occur where tropical fish are frequently eaten, as in the Pacific and Indian Oceans and the north Caribbean Sea. However, such fish served in restaurants have also caused cases in temperate regions where the disease is less well-known.

Toxins are increasingly being found in new areas and new toxins are discovered. In New Zealand in 1993, illnesses from neurotoxic shellfish poisoning (NSP) and paralytic shellfish poisoning (PSP), previously unrecorded, were documented because of water temperature changes caused by the El Niňo Southern Oscillation (ENSO) effect. One incident of apparent ciguatera poisoning affected over 500 people who ate a shark in 1993 in Madagascar, causing 98 deaths. In fact, it was two heat-stable liposoluble toxins, carchatoxin A and B, isolated from the shark that seemed to be the cause of the illnesses. In Australia, 20 cases of mild foodborne disease were associated with the consumption of small cockles from New South Wales beaches hundreds of miles apart. An unknown algal toxin was suspected, and a ban was placed on harvesting. Haff Disease was first reported by people living by the Baltic Sea and associated with ingestion of burbot in the 1930s. The same disease affected people in the US from 1984 to1986 after they ate buffalo fish. In 1997, six cases developed muscle aches, rigidness or stiffness, and weakness. One patient had muscle weakness for six months. A heat-stable toxin was suspected.

## 1.5.3   Egg products

In recent years, *Salmonella* in eggs has been a major problem for public health agencies. *Salmonella* Enteritidis (SE), particularly phage type (PT) 4, infects egg-laying poultry

flocks and some of the eggs contain the organism. The risk of illness is more when the egg is used as an ingredient in a food eaten by many people rather than when it is a single egg. Many outbreaks have been reported around the world, and have been associated with foods such as omelets, quiche, meringues, desserts and cakes with egg ingredients, egg nog, and ice cream. For instance, five persons suffering from SE PT 4 infection in England had attended the same gym and consumed a protein-based beverage composed of milk powder and a raw egg as a body-building drink. The value of the drink to these persons outweighed any risk of enteric illness. In one of the largest SE outbreaks, in 1994 there were an estimated 224,000 cases in several US states, following consumption of ice cream. The premix had been contaminated during transport in tankers which had previously been used for carrying raw liquid egg. It was determined that, for some cases, the infectious dose was no more than 28 cells. A risk assessment in 2002 for *S*. Enteritidis in eggs estimated that only 1 in 20,000 (0.005%) eggs were contaminated with S. Enteritidis. However, with over 90 billion eggs produced each year in the US there is a potential for 4.5 million contaminated eggs entering the market each year. Based on storage, consumption and dose response data from a 2005 risk assessment, there are an estimated 175,356 SE illnesses each year from eggs, of which 1440 are hospitalized, and 75 die.

Between 1986 and April 1990, in Argentina, 35 outbreaks of SE affected 3500 persons, largely through consumption of insufficiently-cooked poultry and eggs used in mayonnaise. A similar type problem occurred in Brazil where, in 1993, 280 of 400 patrons of a restaurant suffered from SE infections after eating potato salad with a raw egg dressing; $10^6$ *Staphylococcus aureus* cfu/g was also found in the salad which probably contributed to the discomfort of those ill. Even as late as 1998, the use of raw eggs was still leading to outbreaks in the UK after over a decade of public awareness; one family was ill after a member prepared a lemon soufflé at a cookery course in a college. It was only after the outbreak that the college agreed not to use raw eggs as ingredients in prepared foods. Although the risk of contamination of eggs has been reduced in recent years, one large outbreak shows that, unless the producers are vigilant, large outbreaks are still possible. Beginning in May 2010, the CDC identified a nationwide, four-fold increase in the number of SE isolates through PulseNet, with reports of approximately 200 SE cases every week during late June and early July (Fig. 1.1). Epidemiologic investigations conducted by public health officials in California, Colorado and Minnesota revealed several restaurants or events where more than one person ill with this type of SE had eaten, and that shell eggs were the most likely source of the infections. These eggs had come from a single egg company in Iowa, and subsequently 500 million eggs were recalled nationwide. From 1 May to 30 November 2010, approximately 1939 SE illnesses were reported as likely to be associated with this outbreak. The epidemic curve demonstrates the extent of the outbreak over more than six months. From the nearly 600 environmental samples taken, 11 were found positive for the same strain of SE in feed, egg wash water, farm traffic areas and manure; rodents were also found in the egg-laying area that could have spread the contamination.

Factors contributing to the contamination were: failure to prevent unwanted animals (including rodents) coming into the poultry houses; manure pits too close to the chickens, blocking doors; fly maggots in the manure pits and multitudes of flies in the houses; no practices against the introduction or transfer of SE between and among poultry houses; no changing of protective clothing when employees moved from one house to another; and failure to clean and sanitize equipment.
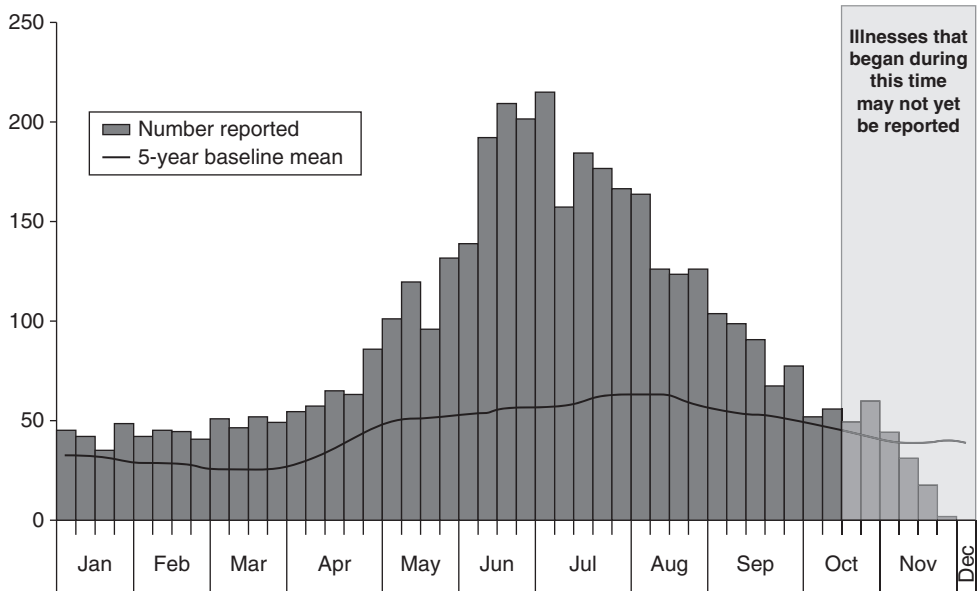
**Figure 1.1**   Number of *Salmonella* Enteritidis cases matching PFGE pattern JEGX01.0004 reported to PulseNet, United States, 2010. http://www.cdc.gov/salmonella/enteritidis/epi_curve.html

## 1.5.4   **Dairy products**

Outbreaks from untreated milk have been well documented, with *Salmonella*, *E. coli* O157 and *Campylobacter* being the most frequent pathogens. A large *Campylobacter* outbreak of 72 cases occurred in 1992 at an outdoor festival at an English farm at which local unpasteurized milk was sold. A similar sized outbreak occurred in 2001 in Wisconsin when 75 people drinking raw milk from one organic dairy farm with 36 cows suffered from *Campylobacter* gastroenteritis. Those ill had participated in a cow leasing program to try and avoid regulatory action since the state prohibits the sale of raw milk. The farm also gave away unpasteurized milk to tour groups, and more cases could have occurred but were undocumented. In a smaller campylobacteriosis outbreak in South Carolina in 2011 eight persons were ill after drinking raw milk. Since raw milk sales are permitted in that state, the farm continues to sell its product, especially as FDA could not find any *Campylobacter* in the samples the inspectors took. The farm produces about 80 to 100 gallons of milk per day and sells raw milk for $6 a gallon.

Occasionally, pasteurized milk has been implicated in outbreaks. In 1985, over 200,000 persons were ill from milk contaminated with *Salmonella* Typhimurium produced in one large Illinois dairy. The cause was never discovered but assumed to be a cross-connection fault that allowed contamination of the pasteurized milk. The dairy closed permanently and there were high settlement costs. In 2000, an outbreak of multidrug-resistant *Salmonella* Typhimurium infections occurred in Pennsylvania and New Jersey with 93 confirmed cases. A case-control study implicated pasteurized milk from a dairy, and an inspection indicated the potential for contamination after pasteurization. Dairy cattle were the likely reservoir. The milk had been adequately pasteurized but conditions in the plant likely allowed post-process contamination. There was excessive condensation throughout the processing and packaging areas, allowing condensation droplets to fall into open containers, several machines leaked raw milk onto the floor, and raw skim milk was held in a silo at >10 °C.

Many small outbreaks have been attributed to *E. coli* O157:H7 in raw milk and occasionally raw milk cheese though environmental contamination typically arising from the cows themselves. Risk factors for contracting the associated disease also include direct or indirect contact with farm animals or animal manure. Farming families may develop immunity to the organism. There is also concern for non-O157 serotypes. In 1994, 17 people were ill after drinking a brand of pasteurized milk in Montana. The pathogen was a non-O157 STEC (*E. coli* O104). Coliform counts exceeded 10/100 ml milk, and fecal coliforms were isolated from post-pasteurization pipes and surfaces. One new pathogen of concern is *Streptococcus zooepidemicus*, which causes acute pharyngitis, glomerulonephritis, meningitis, septicemia and death. There have been outbreaks in Romania, Austria, England and Australia from raw milk consumption. In one of these, delivered milk, meant to be pasteurized, was substituted with bottled raw milk because of supply shortages.

Reconstituted skim milk powder containing *Staphylococcus aureus* toxin caused a very large outbreak in Osaka, Japan, in June 2000. Although many staphylococci were seen under the microscope they had all been killed by the processing, but any enterotoxins produced earlier would still be effective. In fact, more than 13,000 persons were affected by the action of staphylococcal enterotoxin A (SEA). The average total intake of SEA per capita was estimated to be approximately 20–100 ng. Since this is considered a low amount to cause illness, other enterotoxins not looked for could have been present and increased the ingested dose. *S. aureus* if present in pasteurized skimmed milk under room temperature will grow rapidly and some kind of processing failure may have occurred, in this case through a power outage, to allow the organism to grow (≥500,000 cfu/ml) and produce toxin.

*L. monocytogenes* was declared an adulterant in RTE food by the US FDA after a major outbreak from Mexican-style soft cheeses produced in California killed 18 adults and 10 newborns in 1985. Since then, several cheese-associated listeriosis outbreaks have occurred in North America, Europe and elsewhere despite more rigorous attention to better cheese-making hygienic conditions and regulatory oversight. A commercial cheese (acid curd) made from pasteurized milk caused a large listeriosis outbreak with 189 cases in Germany from October 2006 through February 2007. The *Listeria monocytogenes* outbreak strain was identified in humans and in cheese samples from a patient's home and from the production plant. Recent European food safety alerts due to *Listeria*-contaminated cheeses more often concerned products made from pasteurized or heat-treated milk than from raw milk. Between June and December 2008, 38 confirmed cases of listeriosis in cheese in Quebec led to 38 hospitalizations, of which 14 were pregnant and gave birth prematurely. Two adults died and there were some perinatal deaths. The trace-back of food eaten found two cheese plants producing soft cheeses contaminated with the same *L. monocytogenes* PFGE type, and in early September recalls were conducted affecting more than 300 retailers which had received the cheeses. The incriminated product was a soft washed rind cheese made with pasteurized milk, but other cheeses made from both pasteurized and raw milk were found to contain nonepidemic strains of *L. monocytogenes* indicating a high degree of insanitation in some facilities. In 2010, in the US, three outbreaks were associated with raw milk cheese products and one with pasteurized cheese (with *Brucella*, *Campylobacter*, *E. coli* O157:H7, and *L. monocytogenes*). In two of these, the cheeses had been stored for the permitted >60 days before sale, indicating that this decades-old safety measure is not that effective. Overall, the types of cheese and etiologic agents involved in outbreaks in the US attributed to cheese varied depending on whether the milk used to make the cheese was pasteurized. Outbreaks associated with unpasteurized cheese were more frequently due to pathogens transmitted from animals and their environments, whereas pasteurized cheese outbreaks were associated

with contamination by food handlers. Understanding the sources and routes of contamination can help to improve the safety of cheese products.

Butter is an unusual vehicle for foodborne illness, but from December 1998 to February 1999, 18 people in Finland contracted listeriosis after eating locally-produced butter. Sixteen of these had septicemias and four died. The same PFGE strain of *L. monocytogenes* serotype 3 was isolated from the cases, the butter and the producing dairy plant. Although botulism from dairy products is rare, some serious outbreaks have been documented. In 1989, the largest outbreak in the UK took place after commercially-prepared hazelnut yogurt contaminated the nut paste containing *C. botulinum* toxin. In Italy, in 1996, eight persons, mostly children, and one death occurred after tiramisu made with mascarpone cheese was eaten. In 2011, korma (curry) sauce made with yoghurt, onions, nuts and spices and stored in glass jars paralyzed two children in Scotland from *C. botulinum* toxin.

## 1.5.5  Vegetables and fruits

Fresh fruit and vegetable consumption has risen 50% in the US since 1970. Premixed salad sales have risen substantially. Processors are now venturing into new areas, sometimes with little understanding of the risks of transmitting pathogens and the requirements for producing safe food. Many products are certified as organic and chemical free but there are no control measures regarding the presence of manure or cattle feces. Fresh produce, therefore, is becoming increasingly a vehicle for transmitting enteric diseases of many different types, and because there is no terminal heat step, consumers are vulnerable even to low doses of pathogens. Control strategies are limited because there are many stages in the production where pathogens can enter the food chain, and for imported products, there is little knowledge of the agricultural practices involved in their production. There have been many multistate and international outbreaks involving fresh produce. In Japan, in 1996, more than 6300 cases of *E coli* O157:H7 occurred among schoolchildren who ate radish sprouts contaminated at four large growers. Sprouts were also implicated in US multistate salmonellosis outbreaks in more recent years (119 ill from alfalfa sprouts in 1999; 648 ill from mung beans in 2005; 25 ill from alfalfa and spicy sprouts in 2011). Also, in the summer of 2011 a massive outbreak in Germany caused by sprouts was eventually traced back to fenugreek seeds imported from Egypt; the causative organism was *E. coli* O104:H4, a particularly virulent strain that affected over 4100 persons in 15 countries and caused the deaths of 50 of them. The source of the outbreak was particularly difficult to trace and originally cucumbers from Spain were suspected and Europeans refused to eat them and much other produce; eventually a sprouting operation in Germany was the most likely source but no *E. coli* O104 was found there, and the Egyptians denied that the fenugreek seeds were the contaminated source. The EU government offered €210 million ($306.2 million) to affected European farmers who lost money during the outbreak but the economic impact was far greater than the estimated at $2840 million for human losses from sick leave, etc., alone.

Cantaloupes have been contaminated with *Salmonella* for decades. A case-control study showed that 22 persons were ill from May to June 1998, after eating *Salmonella* Oranienberg-contaminated cantaloupes imported into Ontario from the US, Mexico and Central America. Advice to consumers was to thoroughly clean melons with potable water before cutting, prepare cut melons using clean and sanitized utensils and surfaces, and to hold cut melons at ≤7 °C until served or sold. In 2011 cantaloupes were implicated in two multistate outbreaks in the US (Del Monte melons with 20 ill in 10 states, and Jensen Farms product affecting 139 persons in 28 states). The second outbreak, which lasted from July to October

with a recall in September, was considered the worst outbreak in US history since 1924 (when typhoid in raw oysters from New York City killed 150 people) with 29 deaths from *L. monocyogenes*. Other recent large multistate outbreaks were 202 ill (four dead) from *E. coli* O157:H7 in bagged spinach in 2006; 1438 ill from *Salmonella* Saintpaul in tomatoes/peppers in 2008; 106 ill from *Salmonella* Agona in papayas imported from Mexico in 2011). The spinach outbreak, coupled with previous outbreaks from lettuce, triggered the US FDA and the leafy green industry to reduce the risk of contamination. However, as with any crops grown outdoors, there are multiple potential sources of contamination (e.g., irrigation water, flooding, runoff from nearby fields with domestic animals, wild bird and animal droppings, fecal contamination by workers and other humans or pets). Chlorination in the wash water has a limited impact on pathogen decontamination during processing. The human element is always a possibility for contamination as demonstrated in a 1986 *Shigella* outbreak in Texas, where an infected food worker was responsible for contaminating lettuce during its shredding. Another issue is the vigilance of distributors along the supply chain. In 2009, outbreaks in Sweden, Norway and Denmark were caused by *Shigella dysenteriae* type 2 in sugar snaps imported from Kenya. The supply chain involved several companies and distributors with international certification and quality standards that failed to detect potential contamination scenarios at the source or to isolate the pathogen from the product until the outbreak was over. *Shigella* must come from a human source since this pathogen does not colonize animals.

In 1996, 1400 *Cyclospora* infections in 15 US states and Ontario resulted from the consumption of fresh Guatemalan raspberries. Similar outbreaks occurred in 1997 with over 500 persons ill from fresh raspberries, also from Guatemala. In 81% of the 1996 outbreaks and most of the 1997 outbreaks, the raspberries had been rinsed before they were eaten. In 1998, the US banned imports of Guatemalan raspberries but Canada did not follow suit, and in May 1998, 13 clusters of 192 *Cyclospora* cases occurred in southern Ontario. Control measures on farms either were not effective or they were not directed against the true source. The Guatemalan industry had to spend funds to use chlorinated water irrigation systems, develop HACCP plans, and improve worker hygiene, but the market for North America was essentially lost. *Cyclospora* outbreaks are not limited to berries. In 2005, a *Cyclospora* outbreak affected 122 persons who ate an uncooked tomato and cheese appetizer containing fresh basil originating from a Mexican farm in a Quebec restaurant. The attack rate was estimated at nearly 90% with diarrhea, nausea, fatigue and abdominal cramps being the main symptoms. Similar outbreaks with contaminated basil were reported from the Washington, DC, area in 1997, Missouri in 1999, and British Columbia in 2001. *Cyclospora* outbreaks have also been associated with snow peas and mesclun lettuce.

A product not normally associated with illness is nuts. Yet there are many examples where different types of nuts have been contaminated with *Salmonella* and were responsible for illnesses in the US and Canada over the last 10 years. California almonds caused *Salmonella* Enteritidis outbreaks in 2001 and 2003/2004. In the first outbreak with five cases in West Virginia, *Salmonella* was traced back to three California farms. In the second outbreak, 29 cases occurred in 12 states and Canada, and the *Salmonella* was found to be present on two-huller-shellers used to shell almonds during the period the contaminated almonds were produced. There is now a mandatory program requiring all raw almonds to be pasteurized. In 2009, two million pounds of California pistachios were recalled because of contamination by four different serotypes; no known illnesses were associated with consumption of the pistachios which were present in a wide range of foods, including cakes, cookies, puddings, trail mix, snack bars and ice cream. In 2010, an outbreak of *E. coli* O157:H7 was associated

with eating in-shell California hazelnuts (also known as filberts) mostly purchased from bulk bins in grocery stores; the outbreak strain was also found in mixed nut samples. Another outbreak of *E. coli* O157:H7 with 13 cases occurred this time in three Canadian provinces from walnuts imported from the US from January to April 2011. Raw shelled walnuts were sold in bulk and packages. In November 2011 there was a confirmed outbreak of *Salmonella* Enteritidis with 42 cases associated with eating Turkish pine nuts purchased from bulk bins at a grocery chain. As expected, some of the pine nuts were eaten as an ingredient in pre-pared foods, such as Caprese salad, asparagus with pine nuts and pesto. Shopper card records were helpful in identifying pine nut purchases.

In February 2010, 272 people in 44 states and DC were infected with *Salmonella* Montevideo in an Italian-style sausage. On investigation it was found that the black pepper in the salami carried the *Salmonella*. As a result, not only was the meat recalled but also all the batches of black and red pepper from the contaminated source. Because the main *Salmonella* Montevideo outbreak PFGE pattern commonly occurs in the US, there was difficulty separating outbreak strains from unrelated sporadic cases. Interestingly, the USDA regulates salami, and the FDA oversees black pepper and other additives.

Processed vegetables and fruits have also been implicated in foodborne disease, but to a lesser extent than fresh or minimally processed products. Botulism has been documented from several types of vegetable products. In Japan, in July 1998, five people were ill after eating bottled green olives containing type B toxin, with another six ill from the same product in August. In Italy, in August 1998, a woman drank a soup bottled locally and suffered from type A botulism. In the UK, in April 1998, two people were ill and one died after eating home-bottled mushrooms from Italy containing type B toxin. Four persons were ill in two US states in 2007 from consuming canned hot dog sauce which was underprocessed, allowing survival and subsequent growth of *C. botulinum*. Scottish health officials recalled 47,000 jars of a curry sauce made in England after two persons suffered from botulism in November 2011. Botulism from home-bottled low-acid vegetable product and occasionally from com-mercially-canned products has been documented for decades, but it still occurs as illustrated above. Less typical is botulism from baked potatoes. In El Paso, Texas, in 1994, 30 people suffered from botulism after eating a potato dip or egg plant dip at a Greek restaurant. The potatoes had been baked in the oven and left at room temperature, still in the foil, for 18 hours before being used in the dip. Foil allowed anaerobic conditions for surviving *C. botulinum* spores on the skin to germinate and grow into the potato. Shared utensils spread the organism to the eggplant dip. There have been five other botulism outbreaks from baked or boiled potatoes in the US since 1978. These items are now considered a potentially hazardous food.

There have been many peanut butter-related outbreaks and recalls in the last 20 years. The first outbreak was in Australia in 1996 with 15 cases. In the Australian outbreak, 57 cases of *Salmonella* Mbandaka in three states were associated with consumption of the implicated peanut butter. Settlements in 1998 ranged from $500 to $50,000, totalling $7 million for the most seriously affected. The first US instance of *Salmonella* traced to peanuts was a ConAgra brand in 2006–2007 with 648 people in 47 states culture-confirmed with *Salmonella* Tennessee. In 2008/2009 the Peanut Corporation of America was the source of the 2008–2009 outbreak of *Salmonella* Typhimurium, involving thousands of products with peanut butter as an ingredient. There were 691 illnesses, nine deaths, and a recall of over 3800 products costing billions of dollars. In both cases, water in the processing area allowed growth of the *Salmonella*. An outbreak of salmonellosis caused by *Salmonella* Agona affected 211 per-sons with 47 hospitalized in 11 states from April to May 1998. Most cases were aged under

10 or over 70. Toasted oats cereal was the implicated food. A listeriosis outbreak in 1997 with 1594 cases arose from consumption of salad with canned corn served in Italian schools by a mass catering establishment. The same strain of *L. monocytogenes* (group 4) and DNA profiles were found in 123 hospitalized patients, left over corn, and floor and sink drains of the catering establishment. The pathogen grew in corn at 25 °C after six hours.

Fruit products are also being increasingly implicated in outbreaks involving different pathogens. In 1995, 62 cases of salmonellosis were associated with unpasteurized orange juice. Four *Salmonella* serovars were found in juice samples, unwashed fruit surfaces and amphibians beside the processing facility.

In 1993, consumers of apple juice in Massachusetts contracted cryptosporidiosis seemingly from cysts in cattle manure contaminating the apples. Apple juice outbreaks were also documented in Canada and the US in 1980, 1996 and 1998 where *E. coli* O157 was the confirmed or suspected agent. In the largest of these with over 61 cases and one death, a widely-marketed commercial product was involved. A batch of rotten apples had been included in the lot pressed on the day the juice was contaminated, despite the inspector's advice to reject it. The impact on the company was high with a fine of $1.5 million, payments in millions of dollars made to the families of the ill people, and over $1 million in business losses. Several of the parents of the ill children become lobbyists for stricter food safety.

Hepatitis A virus (HAV) outbreaks have been linked to strawberries and green onions. In the 2001 Pennsylvania outbreak associated with one restaurant, 555 persons with hepatitis A were identified, including at least 13 foodservice workers. Several characteristics of the way food was prepared and served in the restaurant could have contributed to the size of the outbreak, including, (1) multiple opportunities for intermingling of uncontaminated and contaminated green onions kept in a common bucket for five days with the ice in which they had been shipped, and (2) serving contaminated items with a relatively long shelf life (e.g., mild salsa) to a large number of patrons over several days. Since HAV is only transmitted through the human fecal-oral route, workers as sources are typically suspected. Green onions require extensive handling during harvesting and preparation for packing. Contamination of green onions could occur (1) by contact with HAV-infected workers, especially children, working in the field during harvesting and preparation, and (2) by contact with HAV-contaminated water during irrigation, rinsing, processing, cooling, and icing of the product. Green onions and strawberries might also be more vulnerable to contamination because plant surfaces are particularly complex or adherent to viral or faecal particles.

## 1.6   High-risk populations

Susceptible persons include children under the age of five, pregnant women, the elderly, persons who have an impaired immune system (e.g., people taking immuno-suppressive drugs, undergoing cancer therapy or infected with the human immunodeficiency virus (HIV), which causes AIDS). These persons should avoid raw foods of animal origin such as undercooked poultry, eggs and meats, unpasteurized milk or cheese or yogurt made with raw milk, dishes prepared with raw or undercooked eggs, raw or undercooked molluscs and crustacea, any cooked food which has been cross-contaminated by raw food after cooking or as a result of poor personal hygiene, and any food stored above the recommended safe temperature. Other populations at risk are those in refugee camps and camps for alien workers, and those with specific customs that compromise food safety, such as eating feasts at funerals even during cholera epidemics, where people share plates, eat with their hands and have poor standards

of hygiene. Some high-risk populations are unpredictable, such as those exposed to bioterroism where enteric pathogens may be put into food in random locations. Travelers to countries where food hygiene is not understood or practised well are exposed to a higher risk of infection than at home, even if the location they are staying in seems to be high class.

## 1.7 Policies to reduce foodborne disease

Policies around the world on how to reduce foodborne disease vary. Most traditional regulations may have specific bacterial counts that cannot be exceeded, and a few may ban products, for example if pathogens like *Listeria monocytogenes* are in RTE foods or *E. coli* O157:H7 is found in ground meat (zero tolerance). The emphasis today is more on preventative measures such as HACCP, either voluntary or mandatory in meat, poultry and/or seafood operations. Warning labels are now being put on packages of raw meat and poultry to advise consumers to cook well, avoid cross-contamination, etc. For juices, these may be pasteurized or a warning label may indicate the risks of drinking unpasteurized products. Because of the concern over high numbers of foodborne illness relating to products prepared in the home from ingredients purchased in retail stores, a few supermarket chains are promoting food safety by educating consumers to keep food safe at home, and working with government to prevent contaminated food from entering the distribution system. In Sweden, great emphasis is put upon eliminating the source of the pathogen, e.g., depopulating flocks with *Salmonella*, testing animals and feed, sanitary slaughter, hygienic practices, and import control. These types of controls come with a substantial cost factor.

  Governments are recognizing there has to be improved surveillance and new approaches to this are being undertaken in different countries. For example, such approaches include measuring the burden of infectious intestinal disease or establishing sentinel sites for case-control and other epidemiological and laboratory studies, including DNA fingerprinting of strains. In the UK, the government is considering making farmers directly liable for compensation for any illness caused by the food they produce. This means that a victim of foodborne disease could sue a farmer if the source of the infection can be traced to a particular foodstuff and farm; the plaintiff would be required merely to establish a causal link but not prove any negligence on the part of the farmer. The principle of strict liability has applied to manufacturers but up until now not to primary producers. This raises a number of questions, such who would be responsible for illnesses arising from bulked food or where raw products of animal origin were mishandled in a home or a restaurant. Certainly, the role of the food worker to have more effective hygienic practices is important, an issue that involves management culture and practices as well as the convenience of hand hygiene sites and new disinfectant and pathogen barrier technologies.

  The Council for Agricultural Science and Technology has issued two publications on foodborne pathogens. The first has been widely quoted and contributed to US policy. The second, published in 1998, gives specific recommendations in goal setting, research needs, production control and education. Some are listed below, and although some progress has been made in the last 14 years, these are still valid today.

*Goal setting*

1. Base food safety policy on risk assessment and include risk management and risk communication strategies.

2. Base food safety regulations on risk assessment and risk management.
3. Set federal food safety goals and priorities: criteria include
   – the numbers of acute illnesses
   – numbers of chronic complications
   – numbers of deaths and disabilities
   – types of food products implicated
   – types of production, harvesting, or processing deficiencies or handling errors identified
   – impact on high-risk populations
   – economic losses to society.

*Research recommendations*

1. Improve reporting of foodborne disease by pathogen, by food, and by contributory factors.
2. Expand existing database on food animals, foods and pathogens.
3. Conduct epidemiologic studies to establish the cause of illness.
4. Improve and regularly update foodborne disease estimates and their costs.
5. Find mechanisms of chronic illnesses and populations at elevated risk from chronic disease associated with foodborne pathogens.
6. Develop rapid, accurate detection methods for pathogen detection in foods.
7. Use dose-response modeling in the risk assessment process.
8. Identify food and pathogen/toxin associations in order to establish controls to minimize the risks.
9. Support pathogen research to understand more about the agents causing foodborne disease, e.g., biofilms, virulence factors, factors contributing to contamination, survival and growth

*Production control*

1. Require producers to adopt effective preharvest intervention strategies to enhance public health, including foodborne pathogen control practices from food source to consumption
2. Harmonize international food safety standards

*Education*

1. Educate the general public and food handlers for safe food preparation and handling, especially for high-risk populations.
2. Use and evaluate food labelling to communicate safe food preparation.
3. Provide risk information on food choices to susceptible persons.

## 1.8 Conclusion

Foodborne disease is an increasing concern in all countries. Because of lack of investment in surveillance and research in infectious diseases over decades, we have to react to problems today rather than anticipate them. For instance, we have limited knowledge on virulence

factors and their transfer between organisms, such as Shigatoxin/verotoxin production from *E. coli* to *Citrobacter* and enteroinvasive properties from *E. coli* to *Klebsiella*. Antimicrobial resistance to antibiotics is preventing adequate means of eliminating enteric pathogens from the gut. Therefore, we are seeing new varieties of pathogens, some of which become important such as *E. coli* O157:H7 and increasingly non-O157 STEC/VTEC strains, and others we are still not certain about for the normal population but can infect the immunocompromized person, such as *Aeromonas*. Governments need to collaborate with limited resources within each country and within blocks of countries, and work with interested stakeholders to develop meaningful policies. Countries need to take recommendations and research conclusions from other countries and adapt these to their own situation, through scientific experts with appropriate resources to produce the relevant policies, risk assessments, and methods to reduce foodborne disease. At present, no country can claim that the battle against the foodborne pathogen is won. Can existing long-term strategies of production control (on farm and HACCP), import inspection, trade agreements, third-party auditors and consumer education substantially reduce foodborne disease or will new problems continually arise to keep the numbers up? If governments are not committed to put resources into new ways to reduce, or at least stabilize, the impact of enteric pathogens, the world's population of 7 billion persons will be continually exposed to new and old hazards. One issue that can be expected is that as surveillance methods improve, we can expect more outbreaks to be identified and this may be an exercise in risk communication to inform the public that these advances are eventually going to improve food safety. Will the 21st century be looked upon as a time when foodborne disease is effectively contained, or will it expand to new products and from unexpected sources and economic downturns limit research and control polices, so that it will become in unpredictable ways a threat to the lives of our children and the increasingly aging population?

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