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FOODBORNE DISEASES

Overview of Biological Hazards and Foodborne Diseases

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Glossary

Cysticercosis  Infection caused by the tapeworm, *Taenia*; the infection occurs when the tapeworm larvae enter the body and form cysticerci: these resemble cysts whose walls are retracted in one place to form heads; each head has suckers and sometimes hooks. The cysticercus develops from an oncosphere in any organ (frequently in muscles, brain, and eye) of its intermediate host, such as swine and cattle. After entering the definitive human host, the cysticercus protrudes from the cyst and the larva is transformed into an adult worm.

Endemic disease  A disease occurring within a particular area.

Enterotoxin  A toxin produced by a microorganism specific for the cells of the intestinal mucosa and causing vomiting and/or diarrhea.

Epidemic  Outbreak of disease that affects a much greater number of people than is usual for the locality or that spreads to regions where it is ordinarily not present.

Hemolytic-uremic syndrome  A disorder that usually occurs when an infection, for example, from *Escherichia coli* O157:H7, in the digestive system produces toxic substances that destroy red blood cells, causing kidney injury.

Immunocompromised  Having a weakened or impaired immune system.

Mastitis  Inflammation of an animal’s udder, usually as a result of bacterial infection.

Oocyst  A fertilized gamete of parasitic sporozoans that is enclosed in a thick wall resistant to the environment.

Pandemic  Worldwide epidemic, a global disease outbreak.

Plasmapheresis  A process in which blood taken from a patient is treated to extract the cells and corpuscles, which are then added to another fluid and returned to the patient’s body.

Retinochoroiditis  Inflammation of the retina and choroid (a brownish membrane between the retina and the white of the eye).

Septicemia  Illness with toxicity due to invasion of the bloodstream by virulent bacteria coming from a local seat of infection (also known as blood poisoning).

Sequela  A disease or disorder that is caused by a preceding disease or injury in the same individual.

Thrombotic thrombocytopenic purpura  Blood disorder that causes blood clots to form in small blood vessels around the body, and leads to a low platelet counts; these clots composed of platelets can block blood flow to the brain, leading to bleeding under the skin forming purple-colored spots called purpura.

Toxicoinfection  Type of foodborne illness that occurs due to ingestion of a bacterium that produces toxins within the gut, which are absorbed to produce gastrointestinal symptoms.

Zoonotic disease  An infectious disease that can be transmitted from wild and domestic animals to humans.
Introduction

Foodborne diseases can be both acute and chronic, and stem from three sources: biological, chemical, and physical. Bacteria, viruses, and parasites, are the main biological hazards causing acute foodborne diseases. Certain biological toxins can also be considered as causing acute effects, such as most seafood toxins, and these are discussed separately in another overview on chemical hazards. Acute can be defined as an incubation period between ingestion and an adverse effect of a determined time, usually from hours up to months, resulting from a single exposure. The impact of these agents results in toxins absorbed by the gastrointestinal tract or in infections produced in the intestines, but can subsequently affect other parts of the body. The whole human population is exposed to these types of agents, but more frequently where there is poor sanitation and lack of temperature control of food, as it occurs in many parts of the developing world. In developing countries and city slums, exposure to such agents on a continual basis may result in chronic diarrhea, which can be fatal for infants and young children. Foodborne bacterial agents include the following: *Bacillus cereus*, *Campylobacter*, *Clostridium perfringens*; different types of pathogenic *Escherichia coli*, *Salmonella*, *Shigella*, *Staphylococcus aureus*; and various types of vibrios, especially *Vibrio cholerae* and *Vibrio parahaemolyticus*. Less well investigated are illnesses caused by enteric viruses, such as hepatitis A and E viruses, caliciviruses, for example, norovirus (NoV), rotavirus, and possibly adenoviruses and astroviruses. More than 70 species of protozoan and helminth parasites can affect humans who consume contaminated food and water; most of these infections occur because of poverty, limited available sanitation, and improper food storage and preparation habits. Those most often investigated include the protozoa: *Cryptosporidium*, *Cyclospora*, *Toxoplasma*, *Entamoeba*, and *Giardia* and helminths: *Clonorchis*, *Echinococcus*, *Fasciola*, *Opisthorchis*, *Paragonimus*, *Taenia*, and *Trichinella*. Diagnostic and detection methods are routinely available only for a few parasites in food and water, mainly those that occur in industrialized countries, for example, *Cryptosporidium*, *Giardia*, *Toxoplasma*, *Anisakis*, *Trichinella*, and *Taenia*.

Epidemics and Pandemics

The major etiological agents that account for the estimated 1.5 million gastrointestinal deaths each year are enterotoxigenic *E. coli* (ETEC), rotavirus, *V. cholerae*, and *Shigella* spp.; all are known to be endemic in the vast majority of developing countries. Whereas *V. cholerae*, *Shigella*, and rotavirus can be detected by standard assays, ETEC is more difficult to recognize, and therefore is often not appreciated as being a major cause of either infantile diarrhea or of cholera-like disease in all age groups. It also causes traveler’s diarrhea in visitors to endemic areas. In fact, ETEC is the most important pathogen of these four pathogens for diarrhea in infants, children, and adults, accounting for 280 million episodes and more than 400,000 deaths annually.

In the historical past, long-lasting plagues and pandemics of infectious disease agents were regularly documented. The latest of these, the seventh cholera pandemic of *V. cholerae* O1, has been the most thoroughly investigated, and has been shown to be transmitted by many related strains through water and food. It began in 1961 in Indonesia, and by 1966, it had affected most of Asia. Cholera incidence then decreased slightly until 1971, when an upsurge was observed in Africa and Europe, which had been free of cholera for >100 years. Cholera rates remained relatively low during the 1980s, with the disease confined to Asia and Africa. However, two major cholera outbreaks appeared in the 1990s: first, a resurgence of cholera in Africa and second, outbreaks starting in Peru that became the first cholera epidemic in Latin America since 1895. The cholera epidemic in Latin America was originally suspected to have come from Asia through the discharge of contaminated ballast water into Peruvian ports, but the isolates from Latin America were closely related to isolates found in Africa in the 1970s and 1990s, indicating that the strain that caused the epidemic in Latin America came from Africa rather than Asia. In addition, a novel serotype, O139, caused major outbreaks on the Indian subcontinent in 1992, and was later shown to be a variant of the seventh pandemic clone; in fact, 9 of the 12 O139 strains actually arose from the O1 precursor strain and were present in seventh pandemic isolates as early as 1979. In 2005, 56 countries had officially notified World Health Organization (WHO) of cholera cases for a total of 131,943 cases and 2,272 deaths. The actual number of cases is probably much higher because of poor surveillance systems and frequent underreporting. Food, as well as water, is frequently implicated in the transmission of cholera, and the reporting of cholera resulted in trade embargoes and lost tourism income in many affected countries. Fecally contaminated water may be the main source but increasingly foods have been implicated. Foods are likely to be fecally contaminated during preparation, particularly those associated with water such as fish and shellfish or handled by infected food workers in an unhygienic environment. WHO estimates that the officially reported cases represent only approximately 5–10% of actual cases worldwide. Many of the *V. cholerae* groups/genotypes spread easily and widely to multiple countries or regions. This finding suggests that cholera epidemics or upsurges, which often occurred at the same time in many countries, are caused by the spread of newly arisen genotypes. Additionally, a genotype can also persist for long periods in an endemic region, as was demonstrated in Southeast Asia and Africa. Detailed analysis of how specific diseases are spread may be useful to monitor and control future potential pandemics.

The Burden of Foodborne Disease

With today’s improvement in standards of personal hygiene and basic sanitation, safe water supplies, effective vaccination programs, food control infrastructure, and the wide application of newer food processing technologies and hazard analysis and critical control point (HACCP), many foodborne and waterborne diseases (*e.g.*, poliomyelitis, brucellosis, cholera, scarlet fever, typhoid, and paratyphoid fevers) have been either eliminated or considerably reduced in industrialized countries. Nevertheless, most countries experienced important increases in several other foodborne diseases.
Zoonoses are particularly difficult to eradicate or even control because of an ever-present reservoir; these include both domestic and wild animal populations. In most developing countries, foodborne and waterborne zoonotic diseases have been so poorly investigated that even the crude burden of illness cannot be estimated with any degree of certainty. Although the situation regarding foodborne diseases is most serious in developing countries, industrialized countries have experienced a succession of well-publicized outbreaks, which have led to better estimates of foodborne disease cases in recent years. The annual estimates are 2.37 million cases of foodborne gastroenteritis in the UK, 5.4 million cases in Australia, and 11–13 million in Canada. The equivalent number for the USA (48 million; range 28.7–71.1 million in 2011) indicates there is considerable uncertainty in determining such estimates. The above data indicate that from 1 in 3 to 1 in 26 are ill from foodborne disease every year.

### Bacteria

**B. cereus**

*Bacillus cereus* causes two types of mild foodborne illnesses. The emetic type is characterized mostly by nausea and vomiting and some abdominal cramps occurring 1–6 h after ingesting the food containing a heat-stable emetic toxin, similar in effect to staphylococcal food poisoning but typically milder. The other type is the diarrheal form which causes abdominal cramps and diarrhea after an incubation period of 8–16 h; the effect is similar to *Clostridium perfringens* toxicoinfections. This type produces a heat-labile diarrheagenic enterotoxin and/or a hemolytic enterotoxin, which cause intestinal fluid secretion. The symptoms of those experiencing either type of illness typically resolve themselves within 24 h. *Bacillus cereus* is commonly found in soil, crops, and dust, and therefore spores are frequently present in many foods. However, the spores themselves can be ingested without adverse effects; it requires their germination and toxin production in food to cause the above-mentioned symptoms. The emetic type is most often associated with rice dishes that have been cooked and then held at warm temperatures for several hours, such as fried rice. The diarrheal form is more associated with vegetable dishes or puddings containing cereal products that have been cooked and stored. However, both types of *Bacillus* spores may be present in the same food (or one strain can produce both types of toxin), and a mixture of incubation periods and symptoms may occur, and less typical foods like cooked poultry dishes may also be vehicles. Spices may be overlooked as a source of spores. Generally, illnesses from *B. cereus* (and other *Bacillus* species) are overlooked and rarely reported and they may be much more frequent than currently documented. Thus, there may be foods that may be important vehicles for which there is no record. Since most recognized cases have been confined to developed countries, the extent of *B. cereus* intoxications in much of the world remains unknown.

**Campylobacter**

*Campylobacter jejuni* is a major cause of bacterial diarrheal illness in the USA and in many other countries, with an estimated 845 000 foodborne cases per year in the USA alone, third in the number of estimated bacterial foodborne disease cases after *Salmonella* and *Clostridium perfringens*. Part of the reason for the high numbers is the low infective dose. However, this foodborne illness occurs mainly as sporadic cases and not as common-source outbreaks. Most cases of diarrhea, vomiting, fever, and abdominal cramping usually appear within 2–5 days of exposure and resolve within days, but one sequel of concern is Guillain–Barre syndrome (GBS) which is discussed under Section Autoimmune Sequelae to Gastrointestinal Infections, *Campylobacter* grows optimally at 42 °C under low oxygen concentrations, such as would be found in the intestines of warm-blooded birds and mammals. Thus, one of the most frequent sources of *Campylobacter* for consumers is raw meat and poultry, particularly the latter. A large proportion of chicken and turkey carcasses entering the kitchen contain this pathogen in higher numbers than *Salmonella*. Most *Campylobacter* organisms originate in poultry flocks. For instance, an European Union (EU) study shows it is approximately 30 times more likely that a *Campylobacter*-colonized broiler flock will yield positive carcasses for *Campylobacter*, compared with a noncolonized flock. Risks for contamination increase with the age of the slaughtered broilers and time of year. The handling, preparation, and consumption of broiler meat may directly account for 20–30% of human cases of campylobacteriosis in the EU. Outbreaks are not only associated with poultry but also with unpasteurized milk, cheese made from unpasteurized milk, undercooked beef, pork, lamb, shellfish, lettuce, and water, with many of these infections caused by cross-contamination in the kitchen, for example, from raw poultry to cooked poultry or salads or other ready-to-eat foods. Poultry growers have yet to significantly reduce infections in chicks because of their close contact and access to fecally contaminated drinking water. Thorough cooking of raw meats and poultry, avoidance of preparation practices that encourage cross-contamination, and proper hand washing will all reduce the risk of consumer infections.

**Clostridium botulinum**

*Clostridium botulinum* is a Gram-positive, rod-shaped anaerobic spore-former that produces oval endospores commonly found in soil, and marine, brackish, and freshwater sediments. Most spores are sufficiently heat resistant that the canning industry stipulated many decades ago that commercial canning for low-acid canned foods (e.g., foods having a pH > 4.6) must be subjected to a 12-D log reduction heating step to ensure there are no surviving spores that could germinate and outgrow to flourish in the anaerobic environment. Although fairly frequent in the past, outbreaks involving commercial canned products (cans/tins and jars) are rare today. However, as recently as in 2007, eight cases of botulism were reported in US residents who had eaten commercially prepared hot dog chili sauce. In this outbreak, the spores apparently survived the retorting process through a processing failure. However, it is not the spores or organism themselves that are the concern but the neurotoxin(s) which are produced and ingested with the food. Seven different toxins (A–G) can be produced but...
the most common affecting humans are A, B, and E. On ingestion the toxins can manifest themselves in several different types of symptoms from diarrhea to constipation, but the main effect is flaccid muscular paralysis that can cause respiratory failure, and eventual death. Some specific symptoms are double vision, blurred vision, drooping eyelids, slurred speech, difficulty swallowing, or muscle weakness. Symptoms generally begin 18–36 h after eating a contaminated food, but they can occur as early as 6 h or as late as 10 days. If untreated, the whole body may become paralyzed affecting the face, arms, breathing muscles, trunk, back, and legs. All Type A and some Type B strains are proteolytic with the food often spoiling with strong odors making it less likely to be consumed; whereas some Type B and all Type E strains are non-proteolytic and will not spoil the food. Infant botulism is an unusual syndrome affecting only very young children suspected of eating honey or other sweeteners, but the source of spores in such illnesses is rarely determined. Spores present in the low-oxygen, low-acid digestive system of an infant can outgrow in the partly anaerobic environment. Although outgrow in the partly anaerobic environment. Although outgrow in the partly anaerobic environment. Although outgrow in the partly anaerobic environment. Although outgrow in the partly anaerobic environment. Although outgrow in the partly anaerobic environment. Although outgrow in the partly anaerobic environment. Although outgrow in the partly anaerobic environment.

Clostridium botulinum

Clostridium botulinum is a Gram-positive, rod-shaped, anaerobic, spore-forming bacterium found as a normal component of soil, decaying vegetation, marine sediment, and the intestinal tract of animals including humans. Thus, it is present in areas subject to human or animal fecal pollution, and has sometimes been used as an indicator of fecal contamination of water and food facilities. Type A Clostridium perfringens is a frequent cause of foodborne illness in many countries, including the UK and the USA. It is the second most frequent cause of bacterial foodborne outbreaks in recent estimates for the USA. This strain possesses a chromosomal enterotoxin (cpe) gene and vegetative cells are relatively heat-resistant compared with other Clostridium perfringens strains. Symptoms of abdominal cramps and diarrhea begin 8–22 h after consumption of foods containing large numbers (>10⁷) of vegetative Clostridium perfringens cells capable of producing an enterotoxin, during sporulation in the gut. This is a type of a toxicoinfection. Clostridium perfringens-associated illness is normally mild with a duration of <24 h but some symptoms may persist occasionally for 1 or 2 weeks, and deaths only rarely occur as a result of dehydration and other complications in elderly persons. Most illnesses occur after cooked soups or stews have been left too long at ambient temperatures or in too large quantities in coolers that would allow spores naturally present to germinate and produce vegetative cells. The cooking stimulates spore germination and once the temperature has dropped below 50 °C, the vegetative cells can multiply rapidly, growing to large numbers, especially in high protein foods. Because Clostridium perfringens can double in number every 7–10 min under optimal temperature (43–45 °C) and nutrient conditions after spore germination, even low numbers of spores can increase to illness-causing levels in a few hours. Thus, outbreaks are most frequently associated with catering companies and others who prepare and then improperly store large quantities of food; social events at churches or large family gatherings are also occasions where meat or poultry dishes may become vehicles for growth of the pathogen. If such stews, soups, gravies, roasts, and other meat or poultry products are served hot or are rapidly cooled before reheating and serving, the risk of Clostridium perfringens food poisoning is much reduced.

A more serious but rare illness, however, is caused by ingesting food contaminated with large numbers of Type C Clostridium perfringens strains, resulting in infection and necrosis of the intestines and subsequent septicemia. This illness is known as enteritis necroticans or pigbel disease in developing countries, particularly Papua New Guinea, but was also documented in post-Second World War Germany (where it was called Darmbrand). The main reason for the development of the disease is protein deprivation because of

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starvation, episodic meat feasting (mainly pork), and staple diets containing trypsin inhibitors, such as sweet potatoes, combined with poor food hygiene. The enterotoxin is normally destroyed by trypsin protease, but production of this enzyme is inhibited if there is lack of protein synthesis. Consumption of spores alone has typically no adverse consequences for foodborne illness. However, *Clostridium perfringens* and other *Clostridium* can also cause gas gangrene in damaged tissue without medical aid, such as which occurs during military conflicts where spores from the soil can be trapped deep into body tissue where partially anaerobic conditions are present. If the pathogen can multiply in the tissue, it can spread rapidly to other parts of the damaged limb, and cause a systemic infection resulting in death. Interestingly, one reason for embalming dead bodies is to prevent the action of *Clostridium perfringens* from producing tissue gas, resulting from spores germinating and outgrowing into the increasingly anaerobic conditions of the corpse. Strangely, *Clostridium perfringens* has an apparent beneficial use. It is believed to be one of the starter cultures components used as the leavening agent in artisanal salt-rising bread. This bread is rarely made today but has been reported to be made in Scotland and Ireland and by immigrants to Canada and the USA centuries ago. Salt-rising bread is different from other breads because yeast does not play any part in the fermentation process. It is thought that the salt used in the starter suppresses any yeast growth and provides an environment to allow naturally present *Clostridium perfringens* and other microorganisms to produce gas and a flavor different from that produced by yeast and lactobacilli (or baking soda). However, the ferment has not been scientifically investigated to understand its beneficial qualities.

Other clostridia apart from *Clostridium botulinum* may occasionally be implicated in foodborne illness but the one of current interest is *Clostridium difficile*, most commonly linked to hospital-acquired infections. These can be chronic and life threatening in humans and hard to eliminate from already-ill patients. *Clostridium difficile* is recognized as both a gut colonizer and cause of diarrhea in food animals, including cattle and poultry. *Clostridium difficile* epidemic infections also occur in piglets. Because *Clostridium difficile* has been isolated from retail foods intended for human consumption in the USA, Canada, and Europe and from meat products intended for consumption by pets, this may be a future concern for contamination of the food supply. At present, there are no analytical methods for testing foods for this pathogen, but these and standards may eventually have to be established to reduce the risk of infections though foods.

**E. coli O157:H7 and Other Pathogenic E. coli**

*Escherichia coli* O157:H7 was first recognized as a pathogen in 1982 as a result of an outbreak involving hamburgers in two US states. The organism was found to produce a toxin that is capable of damaging the kidneys (hemolytic-uremic syndrome (HUS)) and affecting the central nervous system (CNS) (thrombotic thrombocytopenic purpura (TTP)), often preceded by bloody diarrhea (hemorrhagic colitis (HC)). The toxin has been called both a verocytotoxin (affecting thevero kidney cells of the green monkey) and a Shiga-like toxin (similar to a toxin produced by *Shigella*). In fact, there are a number of similar toxins that make up the verotoxin/Shiga toxin category. This division of nomenclature remains today with both terms verotoxin-producing *E. coli* (VTEC) and Shiga toxin-producing *E. coli* (STEC) being used to describe the same group of organisms. Other virulence factors that may be necessary for severe infections are an attaching and effacing protein (adhesin/intimin) and a hemolysin. An earlier outbreak in Canada with one death in 1980 was likely caused by the same organism after apple cider (unfermented squeezed apple juice) was consumed at a farmer’s market. Verotoxin was found in the intestines of the dead youth but VTEC were not successfully isolated. Outbreaks from apple cider have since been occasionally reported in Canada and the USA. In fact, outbreaks of *E. coli* O157:H7 and similar organisms producing verotoxin/Shiga toxin have occurred in many countries outside North America including Australia, Japan, the UK, and many other European countries. It is interesting that the more northerly countries seem to have the higher case rates, for example, Canada, Scotland, and Scandinavian countries. This may relate to farm practices for cattle and sheep that are the main reservoirs for the pathogen. Supershedders (animals that excrete large numbers of the pathogen in their feces) may be a major transmission factor in the field or feedlot and during and after transportation to the slaughterhouse.

In 1993, a major outbreak of *E. coli* O157:H7 infection affected approximately 500 people in four northwestern US states. Many children developed HUS, and four died as a result. This episode forced the US government to declare that *E. coli* O157:H7 was an adulterant in ground beef (and later in nonintact beef). Another large outbreak caused by this pathogen occurred in Africa in 1992, affecting probably thousands of people, with an undocumented number of cases of HUS; drinking water and cooked maize were the identified vehicles of transmission. In 1996, in an outbreak of *E. coli* O157:H7 in Japan, 6309 schoolchildren and 92 school staff members were affected, with two deaths; the epidemiological investigation identified fresh radish sprouts (kaiware-daikon) as the probable cause. This was the largest outbreak ever recorded from this pathogen. However, subsequently two very large and well-publicized outbreaks occurred in Scotland in 1996–97 (400 ill with 20 deaths of elderly persons) and Wales in 2005 (> 170 schoolchildren and others, and 1 child died). Both were caused by butchers who managed catering businesses and also worked with slaughtered animals. The outbreaks were traced to contaminated meat dishes that were delivered to residences and schools. Another well-published outbreak originated from Californian bagged fresh spinach in 2006, with 205 confirmed cases of illness and 3 deaths. The ultimate source was not determined but upstream cattle, feral pigs, and irrigation water may have been involved in field contamination, and the washing and disinfection processes for the spinach were insufficient to prevent *E. coli* O157:H7 from entering the bags. This was one of the three leafy green outbreaks caused by *E. coli* and other pathogens in 2006 involving leafy greens, mainly lettuce, over the previous decade. The leafy greens industry is assessing how to minimize risks to avoid similar problems. Other outbreaks have been linked to
contaminated unpasteurized milk, alfalfa sprouts, salami, recreational water, and contact with infected live animals.

VTEC/STEC that are not of the O157:H7 serotype also pose a risk to human health. Several of these have caused outbreaks (O26, O103, O104, O111 O126, O145, and O157:NM), whereas many more have been reported as causes of sporadic illness. In Europe, infections by non-O157 VTEC/STEC, caused by 37 different O serogroups, are more common than those by O157:H7 strains. In the USA, though outbreaks associated with STEC of serotypes other than O157:H7 are rare, they account for approximately 30% of STEC illness reported to the Centers for Disease Control and Prevention. In 2011, the US Department of Agriculture announced that an additional six serotypes of STEC (O26, O45, O103, O111, O121, and O145) would be considered to be adulterants in nonintact raw beef products. Once implemented, it is likely that decision will have an impact on the meat and other food industries, with the potential for increasing the number of recalls. Industry is concerned that testing for more pathogens that may be controlled by the same means as for E. coli O157 may be an unnecessary burden.

Apart from VTEC/STEC, there are other enteric E. coli pathogens that have been implicated in foodborne outbreaks traced to fecally contaminated food or water, and also through person-to-person contact. These E. coli strains have been classified into four major groups based on their virulence mechanisms: ETEC; enteropathogenic E. coli (EPEC); enteroinvasive E. coli (EIEC); and enterohemorrhagic E. coli (EAEC). Though considered less significant than VTEC/STEC in the developed world, these pathogens are a major cause of infant mortality in regions with poor sanitation and untreated water supply. ETEC is a frequent cause of traveler’s diarrhea in people from industrialized nations during visits to developing countries. Infected infants and children in developing countries suffer from dehydrating diarrhea and create a reservoir of the pathogen in these communities. Both heat-labile and heat-stable enterotoxins are produced when these organisms proliferate in the small intestine to induce watery diarrhea similar to cholera. EPEC and EIEC are most common among young children in the developing world. EIEC is very similar to Shigella and has a low infectious dose; infections are endemic in developing countries. EAEC is increasingly recognized as a cause of persistent diarrhea in children and adults in developing countries and is also a cause of chronic diarrhea among immunocompromised persons, such as those infected with the human immunodeficiency virus (HIV). Typically, EAECs originate only from human sources. However, an EAEC strain of the serotype O104:H4, possessing the genes for a verotoxin, was responsible for an outbreak in Germany in 2011 that involved approximately 4000 cases of illness and caused 50 deaths. This indicates that pathogenic E. coli strains have the ability to exchange genetic material between pathogens from different sources, a concern for future prevention and control measures.

A widespread related opportunistic pathogen, Cronobacter (formerly Enterobacter) sakazakii can cause bacteremia, meningitis, and necrotizing enterocolitis. The main concern is for infants consuming powdered infant formula with some strains able to survive in a desiccated state for more than 2 years.

**Listeria monocytogenes**

Surveillance of listeriosis has until recently been restricted to developed countries, with most incidence rates by country ranging from 0.3 to 0.5 per 100 000, irrespective of the regulatory system and industry control programs that have been in place. Listeria monocytogenes can no longer be called an emerging pathogen as foodborne outbreaks have been documented since 1981, and we already know much about the pathogen and its means of transmission. The incubation period may range from a few days to 2 months before flu-like symptoms progressing to more serious conditions such as meningitis and stillbirths, and usually those infected are hospitalized. Those who are highly immunocompromised are at greatest risk of infection and death, for example, the elderly, fetuses, acquired immunodeficiency syndrome (AIDS), and organ transplant patients. Ready-to-eat foods, such as meat, poultry, and dairy products, are the main vehicles documented in outbreak investigations, particularly deli meats and soft cheeses. These products have been most frequently implicated, but other foods including fresh-cut fruits and vegetables may also be transmission vehicles. Large outbreaks have mostly occurred through consumption of deli meats as cold cuts or in sandwiches, and linked to errors in food-processing plants, such as lack of proper sanitation of contaminated slicing machines, followed by opportunities for growth of the pathogen. Because the organism can grow at cold temperatures and can persist in damp environments, home refrigerators may be both reservoirs for cross-contamination and incubators for the slow growth of the pathogen. Because Listeria is ubiquitous in the environment, it is understandable that infrequently sanitized refrigerators can be continual sources of contamination if the opportunities for cross-contamination arise. One major concern in the USA is the illegal sale of raw-milk Hispanic cheeses which may contain several pathogens including Salmonella, Brucella, and Mycobacterium, as well as Listeria. In addition to outbreak investigations, case-control studies, risk assessments, food attribution studies, and expert elicitation, can help focus on areas of greatest risk for prevention and control measures throughout the food chain. Many governments have strict control policies for this organism in food because of its high mortality rate, with either a tolerance of no more than 100 cfu g⁻¹ or no detection at all in the sample taken (usually 25 g). Recalls are relatively common for ready-to-eat food, especially if the zero tolerance policy is in place. Food processors are encouraged to determine if their foods permit the growth of the pathogen or not, and if so limit the opportunities for its growth by adding organic acids, freezing, or destroy any surviving organisms through postpackaging pasteurization. Despite industry awareness and government regulations and controls, the USA has not managed to achieve the Healthy People 2010 goal of 0.25 cases per 100 000 population (this was 0.34 in 2009), and outbreaks continue to occur. In the USA, food processors are encouraged to determine if their foods permit the growth of the pathogen or not, and if so limit the opportunities for its growth by adding organic acids, freezing, or destroy any surviving organisms through postpackaging pasteurization including high pressure. This was not the policy in Canada in 2008, when a large outbreak in Canada resulted in 58 cases and 22 deaths...
Some small outbreaks may not be detected. However, most risks involving auditors did not identify any major problems in the previous months. Another issue was that third-party used to wash the cantaloupes; and there was no means to place into cold storage. Another issue was that third-party auditors did not identify any major problems in the previous months. However, most risks involving L. monocytogenes of ready-to-eat food, particularly deli meats occur more at retail than at processing; the exposed public is likely to be much less than at processing; the exposed public is likely to be much less risk. The 2011 Colorado cantaloupe outbreak with 30 deaths led to a congressional investigation that indicated that condensation from cooling systems drained directly onto the floor; poor drainage resulted in water pooling around the food processing equipment; inappropriate food processing equipment was difficult to clean; no antimicrobial solution was used to wash the cantaloupes; and there was no means to remove field heat from the cantaloupes before they were placed into cold storage. Another issue was that third-party auditors did not identify any major problems in the previous months. However, most risks involving L. monocytogenes of ready-to-eat food, particularly deli meats occur more at retail than at processing; the exposed public is likely to be much less and some small outbreaks may not be detected.

**Mycobacterium avium Subspecies paratuberculosis (MAP)**

Johne’s disease, or paratuberculosis, is a chronic intestinal infection in ruminant animals believed to be caused by MAP. The usually fatal and slowly developing infectious disease is characterized by chronic intestinal inflammation, reduction of milk yield, wasting syndrome with loss of weight, muscle atrophy, fatigue, and weakness (cachexia), and in some species diarrhea, after a long preclinical phase. Treatment is ineffective and economically impracticable, which forces the farmer to cull these animals prematurely. Detection methods include serological testing, and culture followed by molecular typing of the isolated strains. The infection primarily affects domestic and free-ranging ruminants (cattle, sheep, goats, deer, elk, and bison), but has also been reported in primates, rabbits, stoats, foxes and other nonruminant wildlife. Wildlife reservoirs contribute to the persistence and spread of infection, which is widespread in domestic animals in Europe, North America, Australia, and other parts of the world. Because the pathogen can also exist subclinically in these animals for years without necessarily causing clinical disease, not only is the disease underreported but also these animals represent a reservoir for other animals as well. For instance, bovine herd prevalence has been reported from 7% to 55% in Europe and 9–22% in Australian dairy cattle. In the USA, 34% of lymph nodes and 80% of hide coats of culled beef and dairy cattle were positive for MAP as detected by polymerase chain reaction, but the rate was under 1% in the final processed carcasses. However, true animal prevalence is difficult to compare across studies as only a very few of them adjusted prevalence for factors such as test sensitivity and specificity. Even if animals do not die, there is economic loss because of premature culling and increased veterinary costs. Subclinically infected cows secrete *M. paratuberculosis* in their milk. MAP may also enter the milk by fecal contamination in the milking parlor, and one of the concerns is that it may survive commercial pasteurization as it is more thermostolerant than *Mycobacterium bovis*. It has been cultured from 2% to 3% of retail pasteurized milk units in the UK and the USA, and in the Czech Republic, it has been found in raw milk from fecal culture-positive cows (10–20% of animals), and 1.6% of commercially pasteurized retail milk. Because the population is exposed to this pathogen through the consumption of milk and meat from infected animals, it has been postulated that this organism may be a cause of, or at least a contributor to, Crohn’s disease, a chronic inflammatory condition of the human intestine. Other possible routes of human exposure to MAP are dairy products, infant formula, vegetables, fruits, and contaminated water supplies. However, presently, the role of MAP in Crohn’s disease is still debated and there is no consensus within the scientific community. Nevertheless, *M. paratuberculosis* is a pathogen of concern, with subclinically infected dairy cows being a major source of the pathogen. The fact that the milk containing MAP has been pasteurized to the required national and EU standards means that humans are likely being exposed to low levels of this chronic enteric pathogen on a regular basis.

**Salmonella**

The family Enterobacteriaceae, including the closely related genera *Cronobacter*, *Escherichia*, *Salmonella*, *Shigella*, and *Yersinia*, is responsible for most foodborne illnesses. Of these, *Salmonella* is the most widespread in the environment with over 2800 serovars now recognized. The ones most frequently associated with foodborne outbreaks are *Salmonella enterica* subspecies enterica with serovars such as Enteritidis, Montevideo, and Typhimurium (typically written *Salmonella* Enteritidis, etc.). In countries where enteric notifiable diseases and foodborne disease outbreak surveillance statistics are recorded, salmonellosis typically ranks first or second in terms of outbreaks and case numbers in most countries. In the USA, there are estimated 644 786–1 679 667 foodborne cases each year and 378 deaths. Symptoms of salmonellosis are typically moderate with gastroenteritis lasting several days, but sequelae can follow an infection, and *Salmonella* is the largest cause of foodborne disease-related deaths in the USA. Part of this is because the pathogen can persist in different environmental niches that range from wet to very dry and in highly nutritious foods to powders. A wide range of foods, including raw meats, poultry, eggs, milk and dairy products, fish, shrimp, frog legs, tomatoes, cantaloupes, leafy greens, yeast, coconut, sauces and salad dressings, cake mixes, cream-filled desserts and toppings, dried gelatin, peanut butter, cocoa, chocolate, tahini, and other foods have been identified as being contaminated with *Salmonella* spp., and, subsequently, serving as vehicles for the transmission of this pathogen in outbreaks. Where the organism is protected from stomach acids, such as in cheese, chocolate, or hamburger, the infectious dose can be quite low (1–100 cfu). As a result of industrialization, large-scale centralized production, and an increasing export market, large outbreaks have been reported. Recent examples of large outbreaks in the USA and Canada include *Sa. Enteritidis* in mung beans in 2005 (648 cases).
Salmonella Tennessee in peanut butter in 2006 (>600, two deaths); Salmonella I 4,[5],12:i– (no serovar name) in pot pies in 2007 (425 cases); Salmonella Saintpaul in tomatoes/peppers in 2008 (1438 cases); Salmonella Typhimurium in peanut butter and peanut butter products (>3900 were recalled) in 2008–09 (691 cases, 9 deaths); Salmonella Montevideo in meat with pepper in 2009–10 (272 cases); and Sa. Enteritidis in shell eggs in 2010 (1500 cases and 550 million eggs recalled).

Eggs were also a key vehicle for Salmonella outbreaks in Europe, both as the main ingredient and in buffers and inadequately heated bakery items, as well as through contaminated pork products. Some of these were reported from only one country, as 225 cases (62 hospitalized) of Sa. Typhimurium from hard cheese in the Netherlands in 2007; 42 cases of Salmonella Kedougou from infant formula in Spain; and 53 cases of Salmonella 4, 12:i from dried pork sausage in France in 2008. However, in the EU community food items are regularly shipped across borders, and multinational outbreaks are also frequent. From 2001 to 2005, four outbreaks of salmonellosis (Salmonella Newport and Sa. Typhimurium) in the UK were traced to lettuce but the source of contamination was never conclusively identified because of the complex distribution system. In Finland, in 2005, however, 60 Sa. Typhimurium cases were linked to lettuce from a supplier in Spain. An outbreak occurred from chocolate in 2001–02 with 439 cases, not only in Germany but also in 6 other countries; Salmonella Oranienberg was found in 5% of 381 chocolates tested at levels of 1.1–2.8 MPN g⁻¹. In 2005, the largest outbreak documented in Spain occurred when precooked chicken contaminated with Salmonella Hadar infected 2883 persons; residents in four other EU countries were also affected. In 2007, 55 persons in England/Wales and a further 19 in 5 other countries were infected with Salmonella Senftenberg from fresh basil imported from Israel. Also, in 2007, a Salmonella Weltevreden outbreak was documented involving 45 persons in Norway, Denmark, and Finland from alfalfa seeds originating in the Netherlands and possibly Italy. In 2008, an outbreak occurred in Norway, Sweden, and Denmark with 37 cases and 4 deaths from Sa. Typhimurium in raw Danish pork products. Also in 2008, a similar Sa. Typhimurium outbreak, but involving two separate strains, occurred in Switzerland where approximately 150 persons were affected after eating pork, typically barbecued. One strain was more associated with cases from Denmark and France than with Switzerland based on molecular typing, thus indicating that the strain isolated from pork samples was not persistent in the factory but was introduced by pork imported from other European countries. Today, with the use of more sophisticated molecular strain identification systems, it is now possible to link human and food isolates across different countries. This has been facilitated through PulseNet using pulsed-field gel electrophoresis (PFGE) patterns shared on the Web.

In many Salmonella outbreaks multiple strains may be present, but typically only the dominant strain is recorded in most reports. An example of this occurred in September 2001, where an outbreak in Australia from imported peanuts resulted in a wider investigation in Canada, England/Wales, and Scotland. Patients infected with Salmonella serotypes known to be isolated from peanuts and reported to surveillance systems were interviewed to determine exposure histories, and PFGE patterns of Salmonella isolates were shared electronically among laboratories. From this there were 97 cases of Salmonella Stanley and 12 cases of Sa. Newport infection; one family in Canada had separate members infected with Sa. Stanley, Sa. Newport, and Salmonella Kottbus. Mainly persons of Asian ethnicity were affected: 66% of patients in Australia, 90% in England/Wales and Scotland, and 79% in Canada.

This was because dry-flavored Asian-style peanuts were typically consumed by persons of Asian origin. Laboratories isolated Sa. Stanley, Sa. Newport, Sa. Kottbus, Salmonella Lexington, and Salmonella Unnamed from Brand X peanuts, and isolates of Sa. Stanley from peanuts and human patients were indistinguishable by PFGE. The concentration of Salmonella was generally very low, ranging from <0.03 to 2 MPN g⁻¹ of peanuts in the shell. This international outbreak was detected because of rapid sharing of electronic deoxyribonucleic acid PFGE images.

Despite government vigilance and industry prevention and control procedures, Salmonella outbreaks remain a major issue for all countries, and with global trade, a wide variety of serovars exist to contaminate many different types of foods to cause illnesses all around the world. It is heartening to discover, however, that adherence to HACCP principles, at least in the US broiler industry, is now linked to fewer salmonellosis cases.

Shigella

There are four Shigella species that can be transmitted by food (as well as by water): Shigella dysenteriae, Shigella sonnei, Shigella flexneri, and Shigella boydii. Shigella dysenteriae Type 1 is primarily associated with serious life-threatening dysentery in epidemics (fatality rate 5–15%), and is mostly encountered in developing countries. A major virulence factor produced by Sh. dysenteriae is Shiga toxin (Stx), similar to the Shiga-like toxins produced by STEC/VTEC. Shigella flexneri predominates in areas of endemic infection, mainly in developing countries. More outbreaks of Sh. boydii occur in Central and South America than elsewhere. Shigella sonnei has been implicated in more foodborne outbreaks in developed countries than any other species. Outbreaks have been most often associated with food, water, and child care centers. However, most cases are sporadic and of unknown origin; in the USA, sporadic shigellosis is most likely to occur among young children and Hispanics. Common exposures include international travel and contact with ill persons or child care. Based on the many outbreaks identified, drinking untreated water, or recreational exposure to water, and eating ready-to-eat vegetables washed with untreated water remain important sources for the pathogen; more than one-third of US shigellosis cases could be foodborne. Shigella has been isolated a wide range of foods including potato salad, ground beef, bean dip, raw oysters, fish, and raw vegetables. Ready-to-eat foods are most commonly contaminated with Shigella by an infected food handler who practices poor personal hygiene, or products harvested from sewage-polluted areas. Shigella is salt tolerant, and can survive in many types of ready-to-eat foods including fruits and vegetables, and those subject to modified atmosphere or
vacuum packaging. Cold temperature storage increases their chances for survival. Outbreaks have implicated the following foods as vehicles: tossed salad, potato salad, tofu salad, pasta salad, bean salad, bean dip, shredded and iceberg lettuce, parsley, watermelon, fresh pasteurized milk, cheese, and oysters, mostly caused by Sh. sonnei. In one Sh. sonnei lettuce outbreak in Texas in 1986, an infected food worker at a lettuce-shredding facility likely infected 347 persons across two counties. In 1988, 240 passengers and also flight crew on 13 national and international flights originating in Minneapolis were infected with Sh. sonnei after eating cold items prepared by an airline flight catering kitchen. In 2005, in Bangkok, Thailand, 103 schoolchildren suffered from Sh. sonnei shigellosis (and possibly also salmonellosis) whose origins were likely a mixed chicken and rice dish served for lunch on 1 day. In 2009, in Sweden, there was an unusual outbreak of Sh. dysenteriae Type 2 infections involving 47 cases. The contaminated vehicle was sugar snaps imported from Kenya. Outbreaks in Norway and Denmark were also linked to the snaps. Trace back of the implicated sugar snaps showed that several import companies and distributors were involved, and the international certification and quality standards were questioned as to their utility in preventing such contaminated product from reaching consumers.

Staphylococcus aureus

Staphylococcus aureus has been recognized as a cause of foodborne illness since the 1940s, mainly through ready-to-eat foods contaminated by the pathogen from the human skin or nasopharynx. Typically, St. aureus will grow to numbers in excess of 500 000 cfu g⁻¹ and produce heat-resistant enterotoxins which cause nausea, vomiting, abdominal cramps, and diarrhea within a few hours of ingestion. Cream-filled bakery products displayed unrefrigerated in shop windows were a major vehicle for this pathogen allowing rapid growth in the filling where the high sugar content may inhibit growth of other organisms leading to rapid growth and enterotoxin production. This is less likely today with the use of more synthetic creams and better temperature control of the storage areas. Cooked poultry, egg, and seafood products have also been frequently implicated where worker contamination followed by storage at improper temperatures before consumption has occurred. Less frequent foods are fermented meats and cheese where the starter culture has been contaminated and the fermentation does not proceed rapidly. In South America, soft white cheese is frequently eaten and has been implicated in many outbreaks because of the use of raw milk from mastitic animals, manipulation by asymptomatic workers, and the use of unhygienic production and improper storage practices. Another occasional vehicle leading to outbreaks is dry pasta where the dough is typically extruded at warm temperatures on equipment that is difficult to clean, permitting growth of St. aureus and subsequent toxin production. The organism which competes poorly with other microorganisms can grow rapidly in ready-to-eat processed foods such as ham to produce one or more heat-resistant toxins. Generally, in most developed countries, illnesses from St. aureus intoxication have been declining. Part of the reason may be that it is not considered a major pathogen and is not looked for in many surveillance programs, and therefore may be underreported. One large outbreak in Brazil in 1998 with 4000 cases and 16 deaths, however, indicates that this is a pathogen not to be ignored. Methicillin-resistant St. aureus is an important hospital-acquired infection but has not yet been linked to foodborne outbreaks.

V. cholerae

Cholera is a severe infection caused by V. cholerae, which primarily affects the small intestine. The main symptoms include profuse watery diarrhea and vomiting. Transmission is primarily through contaminated drinking water or food. Severe diarrhea and vomiting can lead to rapid dehydration and electrolyte loss, which can result in death if rehydration with salts is not available. Cholera is a major cause of illness and death in the world, and seven pandemics have been documented since the early 1800s, with the latest major event in the early 1990s in South America. Despite our greater awareness of the disease today, and its means of spread, cholera shows no signs of diminishing, and in fact, new strains are encountered on a regular basis. Vibrio cholerae O1, the causative agent of epidemic cholera, has two biotypes (classical and El Tor). However, three variants of the El Tor biotype have been described after 2000 in Bangladesh, India, Vietnam, and Mozambique; hybrid vibrios have also been described in other regions of Asia and Africa. Also, V. cholerae O139 first identified in 1992, continues to cause outbreaks in India. Many of these strains are resistant to drugs such as trimethoprim, sulfamethoxazole, and streptomycin. Thus, this pathogen is generating different genetic recombinants, which may change the epidemiological role of the disease. Many large outbreaks have been reported in different regions of the world between 2000 and 2010, for example, in 2000, there were 140 000 cases around the world but mainly in Africa; outbreaks in India in 2007 and in Iraq in 2007 and 2008, with thousands of cases (lack of potable drinking water being one of the factors); Vietnam, Congo, Zimbabwe in 2008; South Africa in 2009; and Nigeria in 2010 (the Nigerian outbreak was blamed on heavy seasonal rainfall and poor sanitation). The epidemic in Zimbabwe lasted 9 months and spread to Botswana, Malawi, Mozambique, South Africa, Zambia, and other sub-Saharan African countries. By January 2010, there had been at least 98 000 reported cases and 4200 deaths making it the deadliest African cholera outbreak in the past 15 years. The Zimbabwean government declared the outbreak a national emergency and requested international aid. The epidemic had an unusually high fatality rate because Zimbabweans were seriously weakened by hunger, HIV, and AIDS. A major contributing factor to the severity of the outbreak was the collapse of Zimbabwe’s public health system. In Malawi, 104 deaths were recorded, making it the worst outbreak since 2001–02 when 960 people died. Starting in 2006 and continuing to this day, cholera spread to many parts of Haiti and other countries. It likely originated from a peacekeeper encampment upstream of the first case, and perhaps could have been controlled earlier but neither the local or international agencies had surveillance and medical care facilities in place soon enough.
because of other priorities. *Vibrio cholerae* non-O1 (nonepidemic) naturally occurs in the Gulf of Mexico causing most US cases from contaminated shellfish but the disease is relatively mild.

**Vibrio parahaemolyticus**

*Vibrio parahaemolyticus* was first isolated in 1950 from clinical samples and dried sardines during an outbreak of gastroenteritis in Osaka, Japan. Its pathogenicity is correlated with the production of a thermostable direct hemolysin, known as the Kanagawa phenomenon. Since the 1950s, *V. parahaemolyticus* infections have increased globally; they are usually associated with eating raw, improperly cooked, or cooked, recontaminated fish and shellfish. *Vibrio parahaemolyticus* is the leading cause of seafood-associated gastroenteritis in the USA, and typically associated with the consumption of raw oysters gathered from warm-water estuaries. A correlation exists between the probability of infection and warmer months of the year. Improper refrigeration will allow the proliferation of this organism in seafood, increasing the possibility of infection. The largest culture-confirmed outbreak in North America occurred during the summer of 1997 when 209 people became infected (one death) after eating contaminated raw oysters harvested from California, Oregon, Washington, and British Columbia. A more recent *V. parahaemolyticus* outbreak in 2005 with 22 cases occurred in Alaskan cruise ship passengers after they ate Prince William Sound oysters, extending by 1000 km to the northernmost documented source of oysters that previously had caused *V. parahaemolyticus* illnesses. All oysters associated with the outbreak were harvested when mean daily water temperatures exceeded 15 °C. Since 1997, mean water temperatures in July and August at the implicated oyster farm increased 0.21 °C per year; and in 2004, the mean daily water temperatures in July and August at the shellfish farm did not drop below 15 °C.

*Vibrio parahaemolyticus* causes approximately half of the foodborne outbreaks in some Asian countries, and is the leading cause of foodborne disease outbreaks in Taiwan with most infections from the O3:K6 strain. This strain also accounted for the majority of diarrhea cases in patients in Calcutta, India, between September 1996 and April 1997. Pandemic O3:K6 clone of *V. parahaemolyticus* appeared in Asia around 1996. Since its emergence, it has accounted for most *V. parahaemolyticus* infections in Asia. It then spread to the USA in 1998, and Spain and Chile in 2004, where it has caused hundreds of infections, resulting in the first *V. parahaemolyticus* pandemic in history. This serotype may have a lower infectious dose than other pathogenic *V. parahaemolyticus* strains, accounting for its apparent virulence. *Vibrio parahaemolyticus* has always been a major pathogen documented in Japan because much of the population loves seafood. However, illnesses are usually restricted to relatively small-scale outbreaks involving fewer than 10 cases. From 1996 to 1998, there were 1710 incidents, including 496 outbreaks, with 24,373 cases of *V. parahaemolyticus* reported. The number of foodborne cases of *V. parahaemolyticus* in Japan doubled in 1998 as compared to 1997 and exceeded the number of *Salmonella* cases. Similar to the 1994–95 period, outbreaks were more prevalent in the summer with a peak in August and relatively few outbreaks occurred during winter months. Boiled crabs caused one large-scale outbreak, involving 691 cases. In 1997, the incidence increased to 568 outbreaks and sporadic reports, with 6,786 cases, and in 1998, there were 850 outbreaks and sporadic reports. The increased incidence during 1997–98 has been attributed to an increased incidence of serovar O3:K6. However, since the high of 667 outbreaks and 9,396 cases in 1999 when *Salmonella* and *V. parahaemolyticus* were the main causes of the food poisoning, the incidence of *V. parahaemolyticus* has decreased dramatically to 17 outbreaks and 168 cases in 2008. An extended outbreak in northern Chile in 1997–98 was associated with consumption of shellfish and the exceptionally warm seawater caused by ‘El Nino’ may have favored the growth of the Vibrio. This was the first report of *V. parahaemolyticus* causing an outbreak in Chile. An outbreak in Vietnam with over 500 cases from 1997 to 1999 was associated with fresh seafood eaten by persons of high socioeconomic status. i.e., those who could afford to eat this delicacy.

In Europe, illnesses from *V. parahaemolyticus* have been rare and surveillance programs limited. However, in July 2004, a *V. parahaemolyticus* outbreak with 80 illnesses occurred among guests at several weddings after eating boiled crabs at the same restaurant in Coruna, Spain. *Vibrio parahaemolyticus* O3:K6 was isolated from stool samples. Live crabs were imported to Spain from the UK, processed under unhygienic conditions, and stored at room temperature for several hours before they were eaten. The emergence of this virulent serotype in Europe is a public health concern and emphasizes the need to include *V. parahaemolyticus* in microbiological surveillance and control programs for shellfish-harvesting areas and ready-to-eat seafood. Disease outbreaks caused by *V. parahaemolyticus* in Puerto Montt, Chile, began in 2004 and peaked in 2005 at 3600 clinical cases. Until 2006, every analyzed case was caused by the serovar O3:K6 pandemic strain. In the summer of 2007, only 475 cases were reported and this decrease was attributed to a change in serotype of many pandemic isolates to O3:K59 and the emergence of new clinical strains. There was evidence that pathogenicity-related genes were laterally transferred from the pandemic strain to one of the different *V. parahaemolyticus* groups comprising the diverse and shifting bacterial population in shellfish in this region. Other *Vibrio* species are also important marine and brackish water-related pathogens, particularly *Vibrio vulnificus*, which can cause severe wound infections and death. Symptoms are vomiting, diarrhea, abdominal pain, and a blistering dermatitis, which can lead to septicemia. With changing weather patterns and warming of seawater, we can expect more *Vibrio* infections and outbreaks in future.

**Yersinia and Related Pathogens**

*Yersinia enterocolitica* and *Yersinia pseudotuberculosis* have often been isolated from such animals as pigs, birds, beavers, cats, and dogs. Only *Y. enterocolitica* has been detected in environmental and food sources, such as ponds, lakes, meat, ice cream, and milk. Most isolates are not pathogenic; the exceptions are serotypes O:3, O:5,27, O:8, and O:9, which tend
to show different geographical distributions. These virulent strains most commonly found in pigs and raw milk can cause yersiniosis, characterized by gastroenteritis with diarrhea and/or vomiting, fever, and abdominal pain. The symptoms can be sufficiently painful that appendicitis is thought to be the cause and in many infected persons the appendix has been mistakenly removed. Both Y. enterocolitica and Y. pseudotuberculosis have been associated with reactive arthritis, at a frequency of 2–3%, which may occur even in the absence of frank symptoms. *Versinia enterocolitica* has been transmitted through contaminated unpasteurized milk and milk products, raw pork, tofu, meats, oysters, and fish. Outbreaks have been associated with raw vegetables; the surface of vegetables can become contaminated with pathogenic microorganisms through contact with soil, irrigation water, fertilizers, equipment, humans, and animals. Another feature of *Versinia* spp. is that they can grow at refrigerated temperatures. If product is contaminated during manufacture or preparation, subsequent refrigeration can increase the pathogen load. The food most frequently associated with *Y. enterocolitica* outbreaks and sporadic cases is pork. In Scandinavia, *Versinia* infections are more frequently reported than in most other countries, and this may reflect a high proportion of meat being pork in these countries. For instance, in Norway, yersiniosis is the third most commonly reported cause of acute enteritis after campylobacteriosis and salmonellosis. One outbreak of *Y. enterocolitica* in 2007 was attributed to 11 persons eating a traditional Norwegian Christmas pork dish, brawn, which was probably undercooked. Small outbreaks have also occurred as when children were exposed to raw pork during the making of chitterlings from pork intestines, a traditional winter holiday food in certain US black families (soul food). *Versinia enterocolitica* is transferred from raw chitterlings to infants, particularly to bottle-fed infants, through contact with the hands of the food preparers, and less frequently by direct chewing of the prepared intestines. However, such pork dishes from intestines are common in many countries with similar risks of infection.

Till date, no foodborne outbreaks caused by *Y. pseudotuberculosis* have been reported in the USA, but human infections transmitted via contaminated water and foods have been reported in Japan, Canada, and Europe. At least four outbreaks involving carrots contaminated with *Y. pseudotuberculosis* occurred in Finland in recent years; these mainly affected schoolchildren. The carrots had been stored over winter and a few had spoiled allowing the pathogen to grow. Over a 2-month period in 1998 in British Columbia, Canada, 74 cases of *Y. pseudotuberculosis* were associated with consumption of homogenized milk but no processing, handling, or storage errors were identified during the outbreak investigation. Outbreaks of *Y. pseudotuberculosis* linked to fresh produce have been detected repeatedly in Finland. From 1997 to 2006, there were nine outbreaks documented, including one with carrots as a vehicle where it was shown that shrews were the environmental source of the *Versinia* either during harvesting or over the winter storage period. Kawasaki disease in Japan, characterized by fever, rash, conjunctival infection, cervical lymphadenitis, inflammation of the lips and oral cavity, and erythema and edema of the hands and feet, has been linked to *Y. pseudotuberculosis* infections but the etiology is not well established. *Yersinia pestis*, the plague *Bacillus* or *Francisella tularensis*, causing tularemia, have not been implicated directly in foodborne illness, but hunters and those in the endemic areas may contract these diseases from handling dead or alive infected animals, particularly from skinning rodents, or from flea and deerly bites.

**Viruses**

**Avian Influenza (AI) and Other Respiratory Viral Diseases**

Viral diseases causing influenza and other respiratory problems were prominent in the 2000–09 decade. These attracted considerable attention because of their high numbers of cases, the speed with which the diseases traveled around the world, and the unacceptable fatality rates. None of these was transmitted directly through food, though food animals were involved. However, because viruses rapidly mutate, there is a growing concern that a major zoonotic viral disease can occur that will involve domestic food animals and then be transmitted rapidly person to person. Unfortunately, there is no simple methodology available to isolate and identify viruses in outbreaks and some are not culturable, all of which has limited understanding of the extent of foodborne outbreaks caused by these agents.

AI (or bird flu) is transmitted by the highly pathogenic influenza A virus subtype H5N1 virus. It was first identified in 1987, which subsequently led to global spread in 2003 with the deaths of millions of ducks, geese, and chickens in more than 50 countries, but mainly in Southeast Asia where small flocks of poultry are widely owned on small farms and even in households. A major concern was that it would spread through wild bird populations, especially ducks and geese that migrate long distances. In fact, there was some evidence for this; a few flocks were infected, but did not seem to be a major factor in causing a pandemic. There was no evidence that poultry meat could spread AI to the human population. In periodic outbreaks, infected birds are destroyed to regain public confidence in the poultry products and to revoke bans placed on exported products by other countries. AI has claimed at least 200 human lives in Asia, Turkey, Romania, and Russia. The main concern is that the AI virus could mutate to pass directly from person-to-person rather than from poultry-to-person. The H5N1 virus kills up to 60% of the persons it infects, but most infections occur after direct contact with an infected bird and the disease does not appear to spread well between humans. As long as human-to-human transmission remains rare, the virus cannot cause an influenza pandemic. However, if the virus can be first adapted to pigs, it then could develop the ability to spread among humans. Since 2007, in Indonesia, AI outbreaks have diminished in poultry and in people but pigs are still carrying signs of recent infection. This may mean the virus is still evolving and could eventually spread rapidly through pigs and become another version of the 2009 ‘swine flu’ pandemic. This was a respiratory infection caused by the swine influenza A (H1N1), and contained genetic material from human, swine, and avian flu viruses. Occasionally, pigs transmit influenza viruses to people, mainly hog farm workers and veterinarians. However,
H1N1 flu spread quickly and easily around the world, and in June 2009, the WHO declared H1N1 influenza a global pandemic. However, like AI, H1N1 cannot be transmitted through swine or poultry meat or any other food.

Severe acute respiratory syndrome (SARS) is a respiratory illness caused by a virus. SARS was first reported in Asia in November 2002. It spread worldwide over several months before the outbreak ended in July 2003, with 8096 known infected cases and 774 confirmed human deaths (a case-fatality rate of nearly 10%). The severe illness was marked initially by systemic symptoms of muscle pain, headache, and fever, followed in 2–10 days by the onset of respiratory symptoms. The virus was later isolated from wild animals (palm civet, raccoon dogs, ferret badgers, cats, and bats) which were asymptomatic; the palm civets were sold as food in local markets in Guangdong, China. Because it was thought that the SARS virus crossed the species barrier from palm civet to humans, more than 10,000 of these animals were destroyed in Guangdong Province, alone. In 2005, an almost identical SARS-like coronavirus was found in Chinese bats. It was eventually deduced that the SARS virus originated in bats and spread to humans either directly, or through animals held in Chinese markets; bats are considered the natural reservoir of SARS-like coronaviruses.

**Hepatitis A and E Viruses**

Hepatitis A is one of five human hepatitis viruses that primarily infect the liver and cause illness, and is widespread throughout the world. However, it is declining in countries with adequate potable water and sewage disposal systems. Hepatitis A is one of the few foodborne diseases, i.e., vaccine treatable. The hepatitis A virus (HAV) is transmitted by food and water contaminated with sewage or even urine. Unlike hepatitis B and C, hepatitis A does not develop into potentially fatal chronic hepatitis or cirrhosis; however, HAV infections can still lead to acute liver failure and death (fulminant hepatitis A). Fresh produce contaminated during cultivation, harvesting, processing, and distribution has been a source of hepatitis A. In the USA and other countries, there have been outbreaks associated with frozen strawberries, blueberries, fresh green onions, and lettuce. Restaurant- or caterer-associated outbreaks typically involve an infected food worker who contaminates the food during extensive handling and preparation. Because the incubation period from infection to detectable symptoms can be ≥2 weeks, these infected workers can be asymptomatic excreters for many days or weeks before being identified and removed from preparing and serving food. Shellfish contaminated through polluted water are also an ongoing source of infections; the largest one occurred in Shanghai in 1987 and 1988 when 292,000 persons (with 32 fatalities) were reported in 2 months. These infections were acquired after clams were eaten. These were harvested by boats operating under unsanitary conditions, and from waters containing untreated sewage effluent in previously unharvested clam beds. The contaminated clams were then transported to Shanghai distributors. Clams like other shellfish are filter feeders that can concentrate ingested particles including HAV to cause infections after their ingestion. Steamimg of clams was found to be insufficient to kill the virus, although those who ate raw clams were more likely to be infected than those who consumed cooked clams.

Like hepatitis A, hepatitis E is a disease transmitted between persons via the fecal–oral route, but less is known of the disease transmission. Hepatitis E virus (HEV) may be mainly acquired through water but may originate from animal sources. Domestic animals have been reported as a reservoir for HEV, with some surveys showing infection rates exceeding 95% among domestic pigs. Transmission after consumption of wild boar meat and uncooked deer meat has been reported as well. The rate of transmission to humans by this route and the public health importance of HEV, however, is still unclear. While most often developing into an acute, self-limiting disease, HEV can proceed into a fulminant form in 1–2% of those infected, and up to 20% in infected pregnant women. There is no effective treatment and the only action that can be taken is to relieve the symptoms through rehydration and patient care. When the disease has run its course, HEV is no longer detectable and the liver recovers its regular function. Major outbreaks have occurred in New Delhi, India (30,000 cases in 1955–56); Burma (20,000 cases in 1976–77); Kashmir, India (52,000 cases in 1978); Kanpur, India (79,000 cases in 1991); and China (100,000 cases between 1986 and 1988). More recent outbreaks occurred in 2004, one in Chad with 1442 reported cases and 46 deaths and another in Sudan with 6861 cases and 87 deaths. A larger outbreak occurred in northern Uganda during 2007 and 2008 with 7000 infected and 121 deaths. As these outbreaks demonstrate, HEV often becomes established in camps with displaced persons where sanitation is poor. Specific factors that have been identified with the disease include lack of covers on latrines in the camps; poor hygiene in homes; and lack of routine hand washing with soap before eating or after using the toilet; infections may also occur from eating the raw meat of a contaminated animal. Control measures include improving sanitation and providing adequate supplies of potable water at camps. However, increasingly, HEV is being seen in developed nations with reports of cases in the UK, USA, and Japan. In 2008, one small outbreak occurred with UK passengers on a cruise ship where consuming shellfish and drinking alcohol were risk factors (possibly with contaminated ice), indicating a common-source foodborne outbreak.

**NoV**

NoVs are a group of related, single-stranded ribonucleic acid, nonenveloped viruses that cause acute gastroenteritis in humans. NoV is the official genus name for the group of viruses previously described as ‘Norwalk-like viruses’ or small round structured viruses because of their morphological features. NoVs are part of the larger *Caliciviridae* family, which also includes the genus *Sapovirus*, formerly described as ‘Sapporo-like viruses’, which also cause gastroenteritis in humans. There are five NoV genogroups of which three (GI, GII, and GIV) cause human infections; variants of the GII.4 genotype have been the most common cause of NoV outbreaks. The incubation period is typically 24–48 h and the disease is characterized by acute-onset vomiting (projectile vomiting).
and watery diarrhea. Recovery is usually within 72 h. A major means of transmission is through aerosolization of vomitus, but the fecal–oral route is probably the most frequently encountered, either by consumption of fecally contaminated food or water, or by direct person-to-person spread. As the minimal infectious dose is 10 or fewer particles, one infected person can contaminate a large area and extensive outbreaks have been reported. In the USA and probably many other countries, more foodborne disease outbreaks are caused by NoV than any other pathogen, but the extent is hard to estimate because NoV infections are also transmitted by nonfood means. Asymptomatic infections are common and may play an important role in outbreaks caused by food workers. Also, infected persons can continue to excrete the virus for weeks after they have recovered. The most frequent vehicles in NoV outbreaks include lettuce, cold cuts, hors d’oeuvres, and multicomponent ready-to-eat products where there is much handling and no further cooking step. NoVs are relatively resistant to environmental stress, and are able to survive freezing, some cooking temperatures as high as 60 °C, including after being steamed in shellfish. NoV can survive in up to 30 ppm chlorine, but chlorine-based disinfectant compounds are better for disinfecting contaminated vomitus- or diarrhea-contaminated areas than quaternary ammonium compounds or vacuum cleaners which can actually create an aerosol of infectious particles. Contamination of foods by NoV can be lessened when persons suffering from gastroenteritis avoid preparing or serving food, by frequent hand washing with disinfectants (alcohol-based hand rubs are insufficient), appropriate use of gloves, and by discarding any food that is in the same area where someone has vomited.

**Rotavirus**

Rotavirus is the most common cause of severe watery diarrhea, typically preceded by vomiting, among infants and young children, and is one of several viruses that are attributed to ‘stomach flu’. Infections are very common, and, with each infection, immunity develops, to make subsequent infections less severe, and adults are rarely affected. Dehydration is more common in rotavirus infection than in infections caused by most bacterial pathogens, and is the most common cause of death related to rotavirus infection in young children. As with other enteric viruses, rotavirus is transmitted by the fecal–oral route. The ease of transmission through a population is illustrated by the fact that the feces of an infected individual can contain more than $10^{10–12}$ infectious particles, and the infectious dose is only 10–100 of these particles. Rotaviruses are stable in the environment, and it is difficult to completely sanitize contaminated areas. Outbreaks of rotavirus diarrhea are common among hospitalized infants, young children attending day care centers, and elderly people in nursing homes. Apart from person-to-person spread, water is one known source of the virus, and there have been waterborne outbreaks documented. Food is thought to play a minor role in its transmission, probably <1% of cases. In the USA, it is estimated that NoV accounts for approximately 97% of the virus-caused foodborne outbreaks, with the remainder attributed to hepatitis A and rotavirus. However, a few foodborne rotavirus outbreaks have been described in Japan, the UK, and the USA. One outbreak lasting 4 days occurred in an English boarding school in 1994, and was associated with students eating chicken tikka masala (the median incubation period was 35 h). In 2000, 85 college students in the District of Columbia became ill over a 16-day period after eating sandwiches and deli meats prepared on campus. Some of the preparation staff were carriers. In both the Japanese outbreak from restaurant-prepared food and the above-described campus one, adults (patrons and cooks, respectively) were infected with frank symptoms, which would not have been expected from previous rotavirus studies, as adults are supposed to develop complete immunity. Increased strain virulence may have been a factor.

**Other Viruses**

In the past, poliovirus was one of the disease scourges of persons living under unsanitary conditions particularly in cities causing paralysis and premature death, but vaccination has largely eliminated the disease except in a few areas of Asia. Astroviruses are spread by the oral–fecoal routes and are probably foodborne from time to time. Many other viruses are likely to be recognized at least as occasionally foodborne but the epidemiology has not yet been demonstrated.

**Parasites**

**Protozoans**

**Cryptosporidium**

*Cryptosporidium* species affect different species of mammals and birds. Young animals are those most susceptible to infection. Human cryptosporidiosis is predominantly caused by *Cryptosporidium hominis* and *Cryptosporidium parvum*, which differ in host range; the former infects mostly humans under natural conditions, and the latter infects both humans and many farm animals, such as cattle, sheep, and goats. The main symptoms are watery diarrhea lasting 2–4 days and most people recover quickly, but diarrhea can be prolonged lasting 1–4 weeks, such as in child care centers. In such centers, the spread of infection is highest among young children who are not toilet-trained and their caregivers (those who change diapers). Immunocompromised persons, especially AIDS patients, are at greatest risk for life-threatening conditions, particularly if the cryptosporidiosis develops into a pulmonary form. The infective dose is <10 oocysts. Oocysts are very resistant to normal decontamination procedures, and there is no universally recommended drug for the treatment of the disease, but nitazoxanide is now approved in the USA for diarrhea caused by *Cryptosporidium*. The infective oocyst stage of the organism shed in feces is resistant to most chemical disinfectants, like bleach, but is susceptible to drying and ultraviolet light. Hydrogen peroxide seems to work best. Outbreaks have occurred from infected food workers preparing ready-to-eat foods, and from environmental sources such apple cider from apples contaminated by animal feces or the processing water, and salad items. In 2008, a relatively large outbreak occurred in Finland among...
government employees eating at the same canteen; 72 of them experienced mild gastrointestinal symptoms, mainly diarrhea after eating meals with mixed salad items shipped from Sweden.

There have also been instances of illness related to oocyst survival in milk because of improper pasteurization. Shellfish in polluted waters have been found to contain the oocysts but no outbreaks from consumption of these have been reported. Waterborne outbreaks are the most frequent in communities where there is no filtration system for the potable water supply, and these can sometimes affect thousands of people. For instance, in Milwaukee, Wisconsin in 1993, *Cryptosporidium* oocysts passed through the filtration system of one of the city’s water-treatment plants, and in a 2-week period, 403 000 of the estimated 1.61 million residents became ill with stomach cramps, fever, diarrhea, and dehydration caused by the pathogen; there were 54 deaths attributed to this outbreak, mostly among the elderly and immunocompromised persons, such as AIDS patients. In 2000, three separate drinking water-associated cryptosporidiosis outbreaks occurred in Northern Ireland, each with over 100 cases. One outbreak was caused by the bovine genotype, and two were caused by the human genotype; subgenotyping analyses indicate that two predominant subgenotypes were associated with these outbreaks and had been circulating in the community, probably *Cr. parvum* and *Cr. hominis*, respectively.

**Cyclospora**

The coccidian parasite *Cyclospora cayetanensis* causes protracted diarrhea in humans, and was first identified among expatriates and travelers in Haiti, Guatemala, Nepal, and Peru, where infections are endemic. Since then, *Cyclospora* has been considered a cause of traveler’s diarrhea, linked to consumption of untreated water, lack of adequate sanitation, and soil contact with young children. Asymptomatic carriers have also been identified. One waterborne outbreak occurred in the USA in 1990 where bird feces in a water storage tank might have been the source of the parasite. In the mid-1990s, the first foodborne outbreaks were recognized, when illnesses in both Canada and the USA were associated with raspberries and possibly blackberries imported from Guatemala, likely contaminated during spraying of pesticides. Later, mesclun lettuce and basil were identified as vehicles in other outbreaks, most likely from product imported from Mexico and South America. In 2009, 160 passengers and crew were infected by the parasite after the ship had called in at various ports in South America; no specific vehicle was determined but raw fruits or vegetables brought aboard were suspected. There are no known animal hosts for this pathogen and so human source contamination must be sought during an investigation. In developing countries, transmission most likely occurs through sewage-contaminated water that is sometimes used in the fresh produce industry for irrigation or pesticide application. Unfortunately, the oocysts are relatively resistant to chlorine which makes control difficult.

**Toxoplasma**

Toxoplasmosis is caused by the protozoan *Toxoplasma gondii*. The parasite infects most genera of warm-blooded animals, including humans, but felines represent the only definitive hosts which shed the infective oocysts, with the domestic cat being the animal of greatest risk for humans. *Toxoplasma gondii* is a major cause of abortion and problems with fertility in livestock, especially among ewes, and therefore a significant cause of economic loss in livestock farming. In humans, during the first few weeks postexposure, the infection typically causes a mild flu-like illness or no illness. Thereafter, the parasite rarely causes any symptoms in otherwise healthy adults. However, those with an inadequate immune system, such as AIDS patients, may become seriously ill, and die. The parasite can cause encephalitis and neurological diseases, and can affect the heart, liver, ears, and eyes (chorioretinitis). Transplacental infections with *To. gondii* may occur in some 45% of seroconverted pregnant women. In 10–20% of non-fatal cases, the infants may suffer from damage to the CNS and retinchoroiditis, leading to blindness. It is believed that infected but asymptomatic infants may also develop some sequelae later in life, most commonly retinchoroiditis. It is estimated that, worldwide, in approximately 3 out of every 1000 pregnancies the fetus/infant is affected by toxoplasmosis. In the USA, toxoplasmosis is estimated to be one of the most costly foodborne diseases because of the high lifetime cost of care for surviving but impaired infected fetuses. Unfortunately, there is no completely effective treatment for humans or animals, although pyrimethamine/sulfadiazine are commonly used.

Up to one-third of the world’s human population is estimated to carry a current or previous *Toxoplasma* infection. For instance, the seroprevalence in the USA was 10.8% from 1999 to 2004. Interestingly, 60% of the Inuit of Nunavik (northern Quebec) were seropositive for *To. gondii*. In multivariate analyses, risk factors for seropositivity included increasing age, gender (women > men), lower level of education, consumption of potentially contaminated water, frequent cleaning of water reservoirs, and consumption of seal meat and feathered game. In Doha, Qatar in 2008, approximately 30% of the population was affected where there was a high feral cat population (> 2 million), with the vast majority of the cats living on the streets, scavenging garbage as well as feeding on the rodents near homes and restaurants. The immunoglobulin M (IgM) serological information showed ongoing and repeated exposure to *To. gondii* for all age groups except infants. However, although cats are often blamed for spreading toxoplasmosis (such as children playing in sandboxes where cats have excreted), fecal contamination of hands and consumption of raw or undercooked meat containing tissue cysts are more significant risk factors for human infections. For instance, the seroprevalence in pigs in Guangdong Province, China, in 2008–09 was alarmingly high at 58% in one city region. These results indicate that *To. gondii* infection is a significant health problem in pigs, and pork from these animals represents a public health concern in southern China. This compares with a 2005 USA study where the seroprevalence for pigs was 2.6%, with a herd prevalence of 21.6%, and a mean within-herd prevalence of 2.7%. Analysis of swine management practices indicated that rodent control methods and carcass disposal methods were associated with differences in the number of *To. gondii*-positive samples on the farm.
Entamoeba

Entamoeba histolytica is an anaerobic parasitic protozoan that affects primates. Entamoeba histolytica is estimated to infect approximately 50 million people worldwide although there can be many asymptomatic cases. Symptoms can include fulminating dysentery, bloody diarrhea, weight loss, fatigue, and abdominal pain. The ameba can penetrate the intestinal wall and reach the bloodstream and internal body organs, such as the liver, spleen, lungs, and even the heart and brain. There is approximately 1–3% mortality rate in those with overt symptoms. A closely related species, Entamoeba dispar, is considered to be nonpathogenic but these species are hard to distinguish morphologically. The cysts are transmitted through consumption of contaminated water or food, such as salads, fruits, or vegetables that have been washed in water containing the cysts; handling objects that have been in contact with contaminated soil or animal feces, and by anal sex. More cases occur in the rainy season than in the dry season. One cyst may be enough to initiate an infection with an incubation period of 2–4 weeks. Although water is the primary vehicle for infection, raw fruits and vegetables may also be contaminated, and normal chlorination treatment for potable water is not effective in destroying the cyst. The most dramatic incident in the USA was the Chicago World’s Fair outbreak in 1933 caused by contaminated drinking water. There were 1000 cases and 58 deaths; defective plumbing allowed sewage to contaminate the drinking water. More recently, food workers are suspected of causing a few sporadic infections, but there has been no single large outbreak in industrialized countries. Because asymptomatic persons can excrete large numbers of the protozoan, food workers who have recently returned from endemic areas should be cautioned to follow exemplary hand hygiene, especially when preparing ready-to-eat foods.

Giardia duodenalis (syn. Giardia lamblia, Giardia intestinalis)

Giardia duodenalis is present in many species of wild mammals, as well as in livestock and companion animals (dogs and cats). Thus, consuming water from an apparently pristine wilderness can contain this parasite and cause diarrhea (‘beaver fever’), unless it has first been filtered or boiled. Also, up to 30% of dogs can be affected with diarrhea that can become a source of human infection. Giardiasis is widely distributed throughout the world and may be in both symptomatic and asymptomatic forms at a low prevalence level in the population. Symptoms may include diarrhea, gas or flatulence, greasy stools that tend to float, stomach or abdominal cramps, and occasional nausea. Giardiasis may be expressed as diarrhea within 1 week of ingesting the environmental-resistant cyst. Normally illness lasts for 1–2 weeks, but there are cases of chronic infections lasting months to years. The infectious dose is very low; perhaps even one cyst is sufficient to initiate an infection. Giardiasis is more prevalent in children than in adults, possibly because many individuals seem to have a lasting immunity after infection. However, some adults develop a chronic condition, which is difficult to treat. Infants and other children in child care centers and patients with AIDS are the most vulnerable to infections. Foodborne outbreaks from this parasite are relatively infrequently reported but small episodes may occur but are not typically investigated. In 1979, 29 school employees that ate home-canned salmon developed giardiasis. The wife of the school employee who brought the food had an asymptomatic Giardia-infected grandson, whom she diapered before handling the salmon; her subsequent washing was insufficient to remove all the cysts from her hands. In 1985, in Connecticut, 13 persons at a picnic later suffered from giardiasis after eating a noodle salad dish, which was made by the hostess. Either she or her asymptomatic children were the source of the infection. In New Jersey, in 1986, a similar outbreak occurred in which 9 individuals within a family of 25 developed symptoms after consuming a home-prepared fruit salad. The salad preparer had a diapered child and a pet rabbit at home, both later found to be positive for G. lamblia. The preparer had clearly come in contact with the parasite after diapering the child and/or cleaning up the rabbit cage. In 1998, 22 members of a church youth group in New Mexico suffered from giardiasis with taco ingredients being the most likely vehicle: the source was either contaminated municipal water or one of the parent preparers. In 2005, over a 3-month period, in California, 41 users of exercise equipment in a gym were identified as Giardia cases. There were 12 confirmed cases and 29 other persons with symptoms but no detectable cysts in their stools. The most likely source was the gym water dispenser. The dispenser spigot required substantial hand manipulation to use. Thus, contaminated hands could easily come in contact with the spout, which would be a viable environment for the Giardia. Those infected may have been a continuing source, because many continued to work out at the gym despite their symptoms.

Helminths

Most helminth infections fall under the term neglected tropical diseases (NTDs), as they are not reportable, but affect large segments of the world’s population, mainly the poor and the hard-to-reach population groups where there is lack of access to health care. NTDs represent the fourth most important group of communicable diseases worldwide, behind lower respiratory infections, HIV/AIDS, and diarrheal diseases. Most of these diseases will often result in debilitating complications, contributing to malnutrition, disabilities, anemia, and stunted growth and cognitive development in children. It is estimated that more than 1.2 billion people could be infected with soil-transmitted helminths and/or schistosomes in the Asia-Pacific Region alone. A number of helminth parasites are transmitted to humans through foods. These include the nematodes Trichinella, Anisakis, and Ascaris; the tapeworms Taenia, Echinococcus, and Diphyllobothrium; and the trematodes Fasciola, Clonorchis, Opisthorchis, and Paragonimus. Ascaris alone affects approximately 10–25% of the world’s population, although most of those infected have mild or no symptoms. Severe Ascaris infections cause approximately 60 000 deaths each year, mainly in children. Eating uncooked food grown in contaminated soil or irrigated with inadequately treated wastewater is a frequent source of infection. Anisakis simplex (herring worm) and Diphyllobothrium latum (fish tapeworm) are associated with marine and freshwater
fish, respectively. They can be found in the gut or in other tissue. Most *Anisakis* infections can occur in Japan where fish are more likely to be eaten raw, and they may have to be removed surgically from the stomach if pain like appendicitis or a gastric ulcer persists. Many of these helminths affect wide-spread populations in Asia and Africa and even if they are treated, diseased individuals can be reinfected in endemic regions transmitted through the fecal–oral route.

**Trichinella**

The nematode *Trichinella spiralis*, and other related *Trichinella* species, are acquired through the consumption of raw and undercooked meat containing encysted larvae. The initial symptoms of trichinellosis include diarrhea, abdominal pain, and vomiting, followed later by facial edema, conjunctivitis, symptoms of trichinellosis include diarrhea, abdominal pain, and vomiting, followed later by facial edema, conjunctivitis. *Trichinella* species can infect swine, horses, wolves, bears, skunks, raccoons, rats, and other small mammals, as well as humans. Humans are most likely to be infected by *Tr. spiralis*, *Trichinella nativa*, or *Trichinella britovi*. The cold-adapted species, *Tr. nativa*, found in Arctic mammals is more resistant to freezing; the most frequent strain in bears is *Trichinella murrelli*. Although humans likely were exposed to *Trichinella* spp. long before the domestication of the pig, this host has been the most frequent source of infection in the past few thousand years. Foxes, wolves, and bears have the highest infection rates, but small mammals such as skunks, raccoons, and rats provide the highest risk of infecting the domestic pig. Domestic swine can be exposed to the parasite by the following three ways: (1) feeding on animal tissues containing *Trichinella* cysts; (2) exposure to infected rodents or other infected wildlife; and (3) cannibalism within an infected herd. Vigorous meat inspection programs have effectively lowered infection rates, but small mammals such as skunks, raccoons, and rats are more likely to be eaten raw, and undercooked, occasionally causes small outbreaks, but the population exposed to these is small. However, human trichinellosis is regularly reported in Poland, with 35 outbreaks and 702 cases being reported from 2002 to 2007. The primary source of human infection today has changed from pork to wild boar meat because of stringent control of commercial pork operations. Escaped raccoon dogs (prevalence of 4%) have expanded in both Poland, and Germany and pose a risk to hunters and domestic pigs kept on backyard farms. Wild (feral) pigs are also expanding in the USA, and because the hunting of these animals is encouraged they may represent a source of infection in humans.

**Taenia**

Approximately 50 million people worldwide are infected by either the beef tapeworm, *Taenia saginata*, or the pork tapeworm, *Taenia solium*. Adult tapeworms develop in the small intestine when meat contaminated with larval cysticerci is consumed raw or poorly cooked. Whereas infections with adult tapeworms are not generally severe, infection with some larval stages can result in life-threatening conditions. When eggs of the pork tapeworm, *Ta. solium*, are ingested they develop into larval cysticerci which migrate to various sites in the body including the brain, in which case the condition is called neurocysticercosis (60–90% of *Ta. solium* taeniasis cases), and can be fatal. These infections are most often found in rural, developing countries with poor hygiene where pigs are allowed to roam freely and eat human feces. This allows the tapeworm cycle of infection to be completed and taeniasis becomes endemic in these regions. For instance, because pork is a staple meat in many provinces in China, cysticercosis is highly endemic, with >1 million cases annually; those affected are mainly ethnic minority groups preferring to eat raw pork; it is also associated with an increase in tourism and the promotion of ‘ethnic’ dishes to attract customers. In some regions of Mexico, prevalence is 3.6% of the general population. This compares with approximately 1000 cases in the USA where most cases are immigrants from Latin America. Approximately 50,000 people die each year from cysticercosis. However, there is limited information on the epidemiological profile of foodborne cestodes and trematodes (which are discussed in the next section), and infections from these are likely to be underestimated.

**Trematodes**

Approximately 6000 trematode species ('flukes') have been described, but only a few are important human parasites. Infection of humans occurs through the consumption of contaminated freshwater fish, frogs, shellfish, snails, tadpoles, snakes, water plants (e.g., watercress), and other aquatic products eaten raw or insufficiently cooked. Raw, pickled, or undercooked fish and other aquatic products are prepared in various ways. Such dishes have been extant for hundreds of years with high cultural, ethnic, and nutritional significance, making it difficult for many people to change to safer food habits. Examples of typical traditional preparations include raw crab meat spiced with soy sauce in the Republic of Korea (South Korea), raw grass carp dishes in China, and fresh uncooked small- or medium-sized fish, moderately or extensively fermented in Thailand and Laos. In the 1990s, an estimated 750 million people were at risk of infections with foodborne trematodes (>10% of the world’s population), which comprise liver flukes (*Clonorchis sinensis*, *Fasciola hepatica*, *Opisthorchis felineus*, and *Opisthorchis viverrini*), lung flukes (*Paragonimus* spp.), and intestinal flukes (e.g., *Echinostoma* spp., *Fasciolopsis buski*, and the heterophyids). More recent estimates are staggering despite medical advances; the at-risk populations for clonorchiasis, paragonimiasis, fascioliasis, and opisthorchiasis are 601, 293, 91, and 80 million, respectively. The global estimate for the number of people infected with *Clonorchis sinensis* is 35 million; mostly in China, for *Paragonimus* spp. > 20 million; for *O. viverrini* 10 million infections with 8 million in Thailand and 2 million in the Laos; for *O. felineus* 1.2 million; and for *Fasciola* species 2.4–17 million; whereas for several species of liver fluke 40–50 million. The annual mortality rate is probably much higher than the estimated 10,000. *Clonorchis sinensis* is endemic in China, South Korea, Taiwan, and Vietnam. *Opisthorchis viverrini* is prevalent in Cambodia, Laos, Thailand, and Vietnam, respectively.
O. felineus is endemic in the former Soviet Union, Kazakhstan, and the Ukraine. Fasciola hepatica is endemic on all continents but is of most concern in mountainous regions in South America, Cuba, Iran, Egypt, and Western Europe, with infections from F. gigantica being restricted to Africa and Asia. Paragonimus infections occur mainly in tropical and subtropical areas of East and South Asia and sub-Saharan Africa. Intestinal fluke infections from Echinostoma spp. occur in China, India, Indonesia, Japan, Malaysia, Russia, South Korea, the Philippines, and Thailand. Fasciolopis buski is endemic to Bangladesh, China, India, Indonesia, Laos, Malaysia, Taiwan, Thailand, and Vietnam. Heterophyes heterophyes infections are reported from Egypt, Greece, Iran, Italy, Japan, South Korea, Sudan, Tunisia, and Turkey. The most commonly found intestinal fluke infection in China, South Korea, and Taiwan is Metagonimus yokogawai.

Clonorchiasis in China is endemic in several regions, and it is estimated that 15 million Chinese are infected with Clonorchis sinensis, with numbers tripling over the past decade. Individuals can become infected through consumption of raw freshwater fish (more men than women), handling freshwater fish and not washing, and water contaminated with infected feces, for example, where privies are built adjacent to fish ponds. This increase is partly attributable to rural residents who move to urban centers where raw fish consumption is more fashionable. The disease may decline in time when people are more inclined to eat more of raw, large aquaculture-raised fish (which do not harbor metacercaria) as well as a disinclination to eat fish from rivers polluted with industrial waste. In South Korea, eating raw vegetables and aquatic species are a valued part of many culinary traditions, and the soil may be contaminated through night soil (human fecal waste used as fertilizer) or polluted water. In Japan, foodborne trematode infections are most common in rural areas, where traditional food habits are more preserved and raw freshwater fishes and game meat are also incorporated into the diet. Owing to general unawareness of foodborne parasitic zoonoses, the cysts caused by many trematode infections (such as paragonimiasis and fascioliasis) are often mistaken for cancers, and examination for these false carcinomas results in large economic losses. There is currently no legislative system applied to these types of infections, and both increased surveillance for and awareness of these is needed. In Laos, it is estimated that 1 744 000 individuals are infected with opisthorchiasis throughout the country, with practically the whole population at risk (4 360 000). A campaign in 2008 delivered praziquantel in one Laotian province, targeting children and adults to control both opisthorchiasis and schistosomiasis. However, reinfection can occur rapidly following treatment when there are no supportive control measures in place, such as stressing the importance of thoroughly cooking all aquatic products and boiling water before consumption. Agricultural reform may increase risk factors, as demonstrated when fascioliasis emerged after irrigation systems had been built in Egypt and Peru. However, environmental change can reduce the risk of infections. In Henan province, China, low prevalences of paragonimiasis were found for two villages, where gold mining had contaminated streams and killed crabs, the second intermediate host of paragonimiasis. Pesticide use in rice paddies and contaminated streams and killed crabs, the second intermediate aquatic hosts in China and South Korea. Unfortunately, although these reduce infections, the risk of chronic disease through inappropriate chemical action may increase. Foodborne trematodiases are being more frequently diagnosed in developed countries due to increasing travel patterns and consumption of exotic foods, and it is probable that climate change will favor conditions for human fascioliasis in newer regions, as air temperature and rainfall are crucial for the fluke to flourish. One area of concern is the rapid development of aquaculture. In well-controlled operations, the risk of infection is lower than in wild-caught fish. However, in Vietnam, metacercariae have been found in cultured fish or waste-fed ponds, with up to 50% of the fish infected with foodborne trematodes. The implementation of HACCP systems should be encouraged, as has been done for farming carp, where these have prevented O. viverrini from entering the ponds by monitoring the water supply, fish feed, and pond conditions, and control measures taken to address each deviation. Also, if cold storage is available for aquatic products, any metacercariae present will be killed.

Transmissible Spongiform Encephalopathies (TSEs) Including Bovine Spongiform Encephalopathy (BSE)

TSEs are a group of progressive conditions that affect the brain and CNS of certain animals, including humans. TSEs are unique diseases in that their etiology may be genetic, sporadic, or infectious via ingestion of infected foodstuffs and via iatrogenic (therapeutic action) means. TSEs cannot be transmitted through the air or through touching or most other forms of casual contact, but through contact with infected tissue, body fluids, or contaminated medical instruments, eating infected tissue, transfusion, or transplantation. Normal sterilization procedures such as boiling or irradiating materials fail to render the agents noninfective. For BSE, the best studied of the TSEs, misfolded or contorted prion proteins carry the disease between animals and cause a deterioration of the brain, characterized by the appearance of vacuoles, or clear holes in brain neurons, that give the infected brain its spongiform appearance. BSE in cattle was initially recognized in the UK in 1986, but the cause was not immediately identified, and BSE was subsequently reported in other European countries, with more than 1000 reported cases in 1992, and by 2000, the total number of infected cattle had increased to 180 000. Smaller numbers of cases were reported from Japan, Canada, and the USA, but all were traced back to affected animals in Europe. BSE – a lethal CNS disease specifically targeting cattle produces changes in temperament, such as nervousness or aggression; abnormal postures; lack of coordination and difficulty in rising (‘downers’); decreased milk production; or loss of body condition despite continued appetite. The incubation period ranges from 2 to 8 years. Following the onset of clinical signs, the animal’s condition deteriorates until death ensues or the animal is euthanized. This usually occurs within 2 weeks to 6 months. Most cases in the UK affected dairy cows between 3 and 6 years of age. The primary means of transmission of BSE to cattle was by eating feed contaminated with rendered material, for example, spinal cord, from BSE-infected cattle; this practice is banned today, as there is no guarantee that the feed will be
Creutzfeldt–Jakob disease (CJD; a dementia), Gerstmann–Sträussler–Scheinker syndrome (a dementia), familial insomnia (hallucinations and dementia), Alpers’ syndrome (intractable seizures in infants), and kuru (ritual eating of brains from animals taken from areas where CWD has been identified). There is also a transmissible mink encephalopathy. There are probably more TSEs to be diagnosed in animals.

In sheep and goats, scrapie has been documented for many years (infected animals scrape themselves because of itchy skin): this is a fatal, degenerative disease that affects the nervous systems. European Food Safety Authority (EFSA) has stated that sheep and goat milk and derived products are unlikely to present any risk of TSE contamination if the milk comes from healthy animals. EFSA also considers the risk from eating sheep and goat meat to be low, due to the BSE measures currently in place. However, out of fear of BSE-related illnesses, many European countries banned some traditional sheep or goat products made without removing the spinal cord, such as smalahove (Norwegian origin delicacy of smoked sheep’s head at Christmas) and smokie (West African origin of blowtorching the fleece off the unskinned carcass of an old sheep or goat).

Chronic wasting disease (CWD) of deer, elk, and moose in western and mid-western USA and Canada, which may be mainly transmitted through saliva, appears to be spreading eastwards. Although there is no proven link between affected cervids and human TSE, hunters should avoid eating tissues (e.g., brain, spinal cord, eyes, spleen, tonsils, and lymph nodes) from animals taken from areas where CWD has been identified. There is also a transmissible mink encephalopathy. There are probably more TSEs to be diagnosed in animals.

Chronic Effects of Foodborne Disease Infections

Most foodborne illnesses result in acute symptoms including diarrhea, vomiting, abdominal pain, cramps, and sometimes fever and jaundice, and are self-limiting. The majority of these cases recover within a few days to a few weeks with no lasting effects. However, for some pathogens and some infected individuals illnesses can last much longer and may have life-shortening outcomes. If the disease becomes systemic and affects the bloodstream and one or more organs, without rapid and appropriate medical intervention, the patient may either die or have a long recovery period.

Life-Threatening and Chronic Infections

Those foodborne diseases deemed to be most severe include brucellosis, listeriosis, typhoid fever, and botulism, although none are common in the developed world. However, the health consequences of these when they occur can be serious and life threatening. Many parasitic diseases also fit this picture of a long-lasting chronic condition with severe effects. Those most vulnerable are the very young, very old, those already ill, and pregnant women, all of whom may have underlying health conditions or lower immunity than healthy adults. For example, in pregnant women listeriosis can lead to abortion, stillbirth, or malformation of the fetus, and the overall fatality rate is approximately 30%. Also, those who are malnourished due to inadequate food production are at greater risk of severe infections. These conditions may follow
adverse climatic conditions, such as flooding, drought, or extended high ambient temperatures, and the only available nutritious food may be spoiled or in short supply. Also, repeated episodes of foodborne diseases over a period of time can lead to malnutrition, with serious impact on the growth and the immune system of infants and children. An infant whose resistance is suppressed becomes more vulnerable to other diseases (including respiratory tract infections) and is subsequently caught in a vicious cycle of malnutrition and other diseases (including respiratory tract infections) and is subsequently caught in a vicious cycle of malnutrition and systemic infections can be very serious indeed if they cannot be treated quickly by antimicrobial drugs. Although some \textit{L. monocyctogenes} infections are mild (gastrointestinal form or with a flu-like syndrome) and can occur without seeking medical aid, they can lead to life-threatening complications, such as septicemia, meningitis, encephalitis, osteomyelitis, and endocarditis. Early in pregnancy, a \textit{Listeria} infection may lead to miscarriage, even if the mother is only mildly ill. Later in pregnancy, such an infection may lead to stillbirth, premature birth, or a potentially fatal infection in the baby after birth. Infants who survive a \textit{Listeria} infection may experience long-term neurological damage and delayed development. Adults aged over 60 years can also be seriously affected by listeriosis, and death rates may be as high as 10–20% for this age group.

Enterohemorrhagic \textit{E. coli} (EHEC) strains, which include \textit{E. coli} O157:H7, produce verotoxin/Shiga toxin that can cause diarrhea, ranging from mild and nonbloody to stools that are virtually all blood but contain no fecal leukocytes. Complications of EHEC infection can include HUS and TTP. These are sometimes called sequelae but are really a continuation of the virulence factors of the \textit{E. coli}, for example, it takes time for kidney damage to be apparent compared with HC and bloody diarrhea. HUS is characterized by the acute onset of microangiopathic hemolytic anemia (loss of blood through small blood vessels), renal injury, and low platelet count. TTP also is characterized by these features but can include CNS involvement and fever and may have a more gradual onset. Most cases of HUS occur after acute diarrhea, often bloody. Antibiotic treatment of \textit{E. coli} O157:H7 colitis may, in fact, stimulate further verotoxin/Shiga toxin production. This will increase the risk of HUS, which is a potential life-threatening condition and can induce hypertension, proteinuria, and chronic renal failure in 5% of affected patients. After exposure to VTEC/STEC, 38–61% of individuals develop HC and 3–9% (in sporadic infections) to 20% (in outbreaks) progress to overt HUS. The overall incidence of HUS is estimated to be 2.1 cases per 100 000 persons per year, with a peak incidence in children who are younger than 5 years (6.1 per 100 000 per year), and the lowest rate in adults who are 50–59 years of age (0.5 per 100 000 per year). A Canadian prospective study showed an annual incidence of 1.11 cases of diarrhea-associated HUS per 100 000 children under the age of 16 years. The provinces of Ontario, Quebec, and Alberta, respectively, accounted for 40%, 31%, and 18% of the cases. The mortality rate was 4%, and 34% for children who underwent dialysis for a median of 12 days (range 2–60 days). Individuals in countries where rare or undercooked beef is commonly consumed are at greater risk for HUS following \textit{E. coli} infections. There are approximately 400 new cases of HUS per year in Argentina, the country with the highest incidence in the world. In the acute phase, mortality in children is 2–4%.

HUS has been associated with \textit{E. coli} O157:H7 outbreaks in nursing homes, child care centers, and schools. Major vehicles of infection include ground beef, unpasteurized milk and juice, sprouts, leafy greens, and salami. Waterborne transmission occurs through swimming in contaminated lakes or pools, or through contaminated water. Because low numbers of organisms can cause infection, EHEC is easily transmitted from person-to-person and has been difficult to control in child care centers. From 1982 to 2002 in the USA, the HUS case rate was significantly higher among ground beef-associated outbreaks compared with all other foodborne outbreaks (5.5 vs. 2.5). In the large multistate hamburger outbreak of 1992–93, there were 589 cases of which 41 (7%) developed HUS. In more recent outbreak scenarios, HUS occurs in a higher proportion of cases. One example is the \textit{E. coli} O157:H7 spinach outbreak of 2006 where 205 cases were reported in many states and Canada; 103 were hospitalized, 31 developed HUS (30.1%), and 3 died. The percentage of case patients in whom HUS developed (29%) was high compared to previous \textit{E. coli} O157:H7 outbreaks (15–20%). This finding is consistent with studies that associate \textit{E. coli} expressing Shiga toxin 2 with a higher incidence of HUS. HUS is reported not only from outbreaks caused by O157 serotype but also other VTEC/STEC serotype events as well. In an outbreak in Belgium in 2007, O145 and \textit{E. coli} O26 infections occurred among consumers of ice cream produced at a farm. Five children, ranging in age from 2 to 11 years, developed HUS, and seven other coexposed persons contracted severe diarrhea. In three of the five HUS cases, VTEC O145 infections were laboratory confirmed, one in association with VTEC O26. Identical isolates of \textit{E. coli} O145 and O26 were detected in fecal samples of patients and in ice cream leftovers from one of the birthday parties, and on the farm. The ice cream was made from pasteurized milk and was most likely contaminated by a food worker. A secondary consequence of HUS and kidney dialysis is the risk of diabetes, either shortly after HUS is diagnosed or many years later. The toxin can damage the insulin-producing cells in the pancreas and cause an insulin deficiency.

Infections caused by \textit{V. vulnificus} may present as fulminate septicemia, decreased blood pressure (septic shock), often complicated with necrotizing cutaneous lesions. Wound infections can also cause septicemia. The case-fatality rate for patients with preexisting septicemia is more than 50%. Antibiotic therapy is usually successful in limited systemic infections, but the high mortality associated with this septicemia suggests susceptible individuals should be forewarned about eating raw shellfish.

Anthrax most commonly occurs in animals such as pigs, cattle, horses, and goats, but it can also infect people through skin contact with the spores (cutaneous anthrax), by inhaling the spores (pulmonary anthrax), or by eating meat that contains the spores (intestinal anthrax). Symptoms of intestinal anthrax appear in approximately 1–7 days, with severe abdominal pain, nausea, vomiting, severe diarrhea, and bleeding from the gastrointestinal tract (stomach and intestines). If the symptoms are not treated quickly, septicemia may follow with...
possibly meningitis and pneumonia. Intestinal anthrax has a fatality rate of 25–60%. Certain regions of the world (South and Central America, Southern and Eastern Europe, Asia, Africa, the Caribbean, and the Middle East) report more anthrax in animals than others. For instance, an outbreak of anthrax killed at least 1500 wild game animals in nature preserves in southeastern Zimbabwe in 2009, and at least 83 hippopotamuses in a popular Ugandan game park in 2010. Natural anthrax is endemic in these and many other countries, including Canada and the USA, where the spores can live for decades in dry soil and be ingested by animals ruminating for remnants of vegetation in the driest months. Most human foodborne anthrax cases come from persons scavenging carcasses of animals that have died of anthrax such as described above with the meat consumed either raw or after minimal cooking. In 2010, an outbreak of anthrax in Bangladesh among hundreds of cows infected more than 500 people. The disease spread due to the slaughtering of infected cows from which the meat was sold and consumed. Vaccination of cattle was being carried out to contain the outbreak.

Brucellosis is an important zoonosis and a significant cause of reproductive losses in animals. In domestic livestock, it is caused by *Brucella abortus* (cattle), *Brucella melitensis* or *Brucella ovis* (sheep and goats), and *Brucella suis* (pigs). In humans, brucellosis causes undulant fever and other serious, debilitating, and sometimes chronic infections. Most cases are caused by occupational exposure to infected animals or the ingestion of unpasteurized dairy products. In the USA, *B. suis* has been eliminated from commercial pigs and *B. abortus* has nearly been eradicated from domesticated ruminants. In many patients, the symptoms last for 2–4 weeks and are followed by spontaneous recovery. Others develop an intermittent fever and other persistent symptoms that typically wax and wane at 2- to 14-day intervals. Most people with this undulant form recover completely in 3–12 months. A few patients become chronically ill.

For persons infected with *T. solium*, the pork tapeworm, after eating infected pork that has been undercooked, nearly 250 000 ova are passed daily from human feces to the environment. Cysticercosis is a systemic infection that results from ingesting the eggs of *Taenia*. The eggs are usually found in fecally contaminated water or food. Other possible routes are through autoinfection as a result of the entry of eggs into the stomach by retroperistalsis (reverse peristalsis) or as a result of accidental ingestion of eggs from the host’s own fecally contaminated hands. Humans, in this case, are intermediate hosts. Ova are digested in the stomach and release oncospheres that penetrate the intestinal wall to reach the bloodstream. These oncospheres develop into cysticerci in any organ but are common in brain, subcutaneous tissue, or the eye. The cysticerci become mature and viable approximately 2 months after egg ingestion, and can persist for more than 10 years without any apparent symptoms. Although most brain infections remain asymptomatic, much later, intense inflammation is provoked around the degenerating and calcified cyst, and can result in persistent headaches, seizures, and altered mental status (neurocysticercosis). Cysticercosis is highly endemic in Central and South America, and some parts of Africa and Asia. In Latin America, an estimated 75 million persons live in endemic areas and 400 000 people have symptomatic disease. In the USA, the disease is found in immigrants from Mexico, Central and South America.

Cholangiohepatitis, or recurrent pyogenic cholangitis (RPC), is characterized by a recurrent syndrome of bacterial cholangitis that occurs in association with intrahepatic pigment stones and intrahepatic biliary obstruction. Infection in the biliary system from the parasitic nematode, *Ascaris lumbricoides*, or from trematodes, such as liver flukes *Clonorchis sinensis* and *O. viverrini*, often results in significant epithelial damage. These flukes reside in the peripheral small bile ducts of the liver and produce chronic inflammation of the bile duct, bile duct dilatation, mechanical obstruction, and bile duct wall thickening. Coliforms may then result in portal bacteremia by bacterial translocation as a result of this epithelial damage. Repeated portal bacteremia may cause biliary stasis, obstruction, and stone formation from deficient glucuronidation as a consequence of extreme malnutrition, which consequently creates conditions for RPC. These flukes also are potentially carcinogenic to humans. Human infection of *F. hepatica* allow the flukes to migrate in the liver (hepatic phase) and reside in the bile ducts (biliary phase). In areas of endemic infection, more cholangiocarcinosis cases are now diagnosed incidentally during radiological examinations and there is increasing evidence for links between cholangiochola and cholangiocarcinoma, especially where there is chronic infection.

### Autoimmune Sequelae to Gastrointestinal Infections

Infections from enteric bacteria may give rise to a number of chronic joint diseases which include reactive arthritis, Reiter’s syndrome, and ankylosing spondylitis. It is the elevated antibody levels to these organisms generated during the infection that lead to these sequelae. Case reports and outbreak investigations have demonstrated an association between reactive arthritis and infection with *Salmonella*, *Shigella*, *Campylobacter*, and *Yersinia* species with a frequency of reactive arthritis ranging from 1% to 21%. Symptoms commonly begin approximately 7–30 days after an intestinal illness. The knees and ankles are often affected but also other joints. The duration of symptoms varies considerably but, in most individuals, they subside in less than 6 months. However, some individuals may take in excess of 1 year to recover fully, and a significant portion of affected persons suffer persistent or relapsing illnesses. In addition to environmental factors, a human leukocyte antigen (HLA)-B27 genotype is a predisposing factor in over two-thirds of patients with reactive arthritis. Unfortunately, the use of antibiotics in these patients has not been shown to be effective. Initial treatment consists of high doses of potent nonsteroidal anti-inflammatory drugs.

Some large outbreaks have been followed up to identify cases with prolonged arthritis. In 1984, in Ontario, Canada, an outbreak of *S. Typhimurium* occurred among police officers who were serving as security guards on routes during a papal visit; they had eaten meat sandwiches provided by a caterer when they were on duty. Of the 1608 police officers involved, 432 experienced acute gastroenteritis. Within 3 months following the outbreak, 27 (6.4%) of these officers had developed acute arthritis.
Infections are relatively common, especially in reactive arthritis patients. In Scandinavia, where Yersinia O:3 infection is frequently associated with reactive arthritis, 1339 patients with reactive arthritis. In 2002–03, Danish researchers surveyed 100,000). A similar study in the UK from 1991 to 2001 indicated that the incidence of GBS in a cohort of patients presenting with Campylobacter enteritis (11.7 per 100,000) was 77 times greater than that in the general population (<0.2 per 100,000).

GBS is the most common cause of acute flaccid paralysis. GBS is an autoimmune disorder of the peripheral nervous system characterized by weakness and numbness in the extremities, evolving over a period of several days or weeks; it can eventually paralyze the entire body. Typically, an ascending paralysis occurs, with weakness in the legs spreading to the upper limbs and the face along with complete loss of deep tendon reflexes. Muscles may become so weak that patients need to be put on a ventilator, to allow proper breathing, or be fed through a tube into the stomach because swallowing is not possible. Symptoms are often worst during the first 2 or 3 weeks. With prompt treatment by plasmapheresis or intravenous immunoglobulins and supportive care, the majority of patients regain full functional capacity, but even without any treatment most people eventually recover completely. However, in some cases, the effects are seemingly permanent and can cause premature death; 3–10% of patients die and 20% are still unable to walk after 6 months, and many have pain and fatigue that can persist for months or years. GBS is typically preceded by a Campylobacter infection. Basically, the body’s immune system reacts to the infection attacks the myelin sheaths protecting the nerves, and this interferes with the way that nerves send signals between the brain and body. Campylobacter is associated with several pathological types of GBS, including the demyelinating (acute inflammatory demyelinating polyneuropathy) and axonal (acute motor axonal neuropathy) forms. Different strains of Campylobacter as well as host factors likely play an important role in determining who develops GBS as well as the nerve targets for the host immune attack of peripheral nerves. GBS is unlike other disorders such as multiple sclerosis and Lou Gehrig’s disease, and does not generally cause nerve damage to the brain or spinal cord. Although GBS cases have occurred following Campylobacter outbreaks from raw milk and water, most diagnosed Campylobacter cases are sporadic with no known vehicle association. So, the burden of GBS relating to campylobacteriosis has relied on a few extensive epidemiological investigations.

For instance, a Swedish study with patient data from 1987 to 1995 showed that the risk of developing GBS during the 2 months following a symptomatic episode of Ca. jejuni infection (30.4 per 100,000) was approximately 100 times higher than the risk in the general population (0.3 per 100,000). A similar study in the UK from 1991 to 2001 indicated that the incidence of GBS in a cohort of patients presenting with Campylobacter enteritis (11.7 per 100,000) was 77 times greater than that in the general population (<0.2 per 100,000).

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