Emerging food-borne zoonoses

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Summary

Diarrhoeal diseases, almost all of which are caused by food-borne or water-borne microbial pathogens, are leading causes of illness and death in less developed countries, killing an estimated 1.9 million people annually at the global level. Even in developed countries, it is estimated that up to one third of the population are affected by microbiological food-borne diseases each year. The majority of the pathogens causing this significant disease burden are now considered to be zoonotic. The occurrence of some of these zoonotic pathogens seems to have increased significantly over recent years.

The factors involved in such increases have not been well studied, but they are generally agreed to include changes in animal production systems and in the food production chain. Both types of changes can cause corresponding changes in patterns of exposure to the pathogens and the susceptibility pattern of the human population.

This paper will not attempt a more in-depth analysis of such factors. The authors briefly describe five of the most important emerging food-borne zoonotic pathogens: Salmonella spp., Campylobacter spp., enterohaemorrhagic Escherichia coli, Toxoplasma gondii and Cryptosporidium parvum. The paper does not include a full description of all important emerging food-borne pathogens but instead provides a description of the present situation, as regards these globally more important pathogens. In addition, the authors describe each pathogen according to the new framework of a Food and Agriculture Organization (FAO)/World Health Organization (WHO) microbiological risk assessment, which consists of hazard identification and characterisation, exposure assessment and risk characterisation. Moreover, the authors provide a brief account of attempts at risk mitigation, as well as suggestions for risk management for some of these pathogens, based on thorough international FAO/WHO risk assessments. The authors emphasise the importance of science-based programmes for the continued reduction of pathogens at relevant points of the ‘farm-to-fork’ food production chain, as this is the only sustainable basis for further reducing risks to human health in the area of preventable food-borne diseases.

Keywords


Introduction

There are an enormous number and variety of potential contamination sources along the food processing chain, and thus it is unrealistic to imagine that all food can be kept free from contamination throughout the production process. However, advances in the 20th Century, such as pasteurisation, refrigeration and more recent improvements in hazard analysis and control along the production chain, have contributed to improvements in
the microbiological safety of most foods. Nevertheless, food-borne disease remains a significant cause of morbidity and mortality both in the developed and above all in the developing world.

A recent national surveillance study in England and Wales revealed that one in five people develop infectious intestinal diseases each year, and that *Campylobacter* and *Salmonella* were the most common bacterial pathogens isolated (67). In the United States of America (USA), it has been estimated that food-borne diseases may cause up to 76 million illnesses, 325,000 hospitalisations and 1,800 deaths each year (44). Extrapolating these data to the rest of the world would mean that up to one third of the population in developed countries are affected by microbiological food-borne diseases each year, while the problem is likely to be even more widespread in developing countries (37). The poor are the most susceptible to ill health. Food and water-borne diarrhoeal diseases, for example, are the leading causes of illness and death in less developed countries, killing an estimated 1.9 million people annually, most of whom are children (73).

In the estimations from the USA (44), *Campylobacter*, non-typhoidal *Salmonella* and enterohaemorrhagic *Escherichia coli* (EHEC) account for the vast majority of bacterial food-borne disease cases requiring hospitalisation, whereas *Toxoplasma gondii* accounts for the great majority of severe cases of parasitic infections.

Traditionally, food safety efforts have focused on a number of toxicogenic bacterial pathogens, such as *Staphylococcus*, *Clostridium* and *Bacillus*. However, it seems that some important new problems have emerged. This short paper will briefly describe five of the most important emerging food-borne pathogens involved in animal production, as follows:
- *Salmonella* spp.
- *Campylobacter* spp.
- EHEC
- *T. gondii*
- *Cryptosporidium* spp.

It is not the intention of the authors to give a full description of all emerging food-borne pathogens but to try to outline the global situation in regard to some of the more important pathogens. It should be noted, however, that the contribution of human caliciviruses (‘Norwalk-like virus’ and ‘Sapporo-like virus’) to the infectious intestinal disease burden, has recently been evaluated in several countries. These caliciviruses are now estimated to be the cause of 13% to 17% of all the disease cases in the community that could be attributed to an infectious agent. Since a significant number of these infections are also due to food consumption, more work on food-borne viruses is clearly needed.

Although the authors use the formal headings of microbiological risk assessment, as defined in the Codex Alimentarius and applied by the recurrent Joint Food and Agriculture Organization/World Health Organization (FAO/WHO) Expert Meetings on Microbiological Risk Assessment, this paper does not intend to present either a qualitative or quantitative risk assessment for these five significant pathogens. The headings have been chosen to emphasise the importance of this new scientific discipline, as well as its inherent linkages to several existing disciplines, including epidemiology, clinical microbiology, applied microbiology and mathematical modelling. Moreover, the use of these headings indicates the potential for this new discipline to allow both better focused and better monitored disease control measures, eventually resulting in significant risk reductions.

As defined by WHO and the FAO, risk assessment (the scientific evaluation of known or potential adverse health effects) is an integral part of risk analysis, which also includes risk management (i.e. evaluating, selecting and implementing different courses of action) and risk communication (exchanging information among all interested parties). The four steps of risk assessment are presented in Figure 1 (74); they are, as follows:
- hazard identification
- exposure assessment
- hazard characterisation
- risk characterisation.

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**Fig. 1**
The four steps of a microbiological risk assessment (74)

Hazard identification involves identifying micro-organisms as hazards of concern in the relevant food.

Exposure assessment provides an estimate, with an associated level of uncertainty, of the occurrence and amount of the pathogen in a specified portion of food at the time of consumption.
Hazard characterisation provides a description of the adverse effects that may result from ingestion of a microorganism and presents a dose-response relationship.

Risk characterisation is the integration of the three previous steps to obtain a risk estimate, i.e. an estimate of the likelihood and severity of the adverse effects which will occur in a given population, with an associated level of uncertainty.

**Thermophilic Campylobacter species**

**Introduction**

It was already established in the 1970s that thermophilic Campylobacter was a common cause of bacterial gastroenteritis in humans (59). However, Campylobacter is now the leading cause of zoonotic enteric infections in most developed and developing countries (70). Most human Campylobacter infections are classified as sporadic single cases or as part of small family-related outbreaks. Identified outbreaks are not common. The reported incidence of Campylobacter infections has markedly increased in many developed countries within the last twenty years (Fig. 2).

**Hazard identification and characterisation**

Food-borne campylobacteriosis is associated mostly with *C. jejuni* (which is frequently isolated from chickens) and, to a lesser degree, with *C. coli* (often found in pigs). The principal reservoir of pathogenic Campylobacter spp. is the alimentary tract of wild and domesticated mammals and birds. It is evident that Campylobacter is commonly found in broilers, broiler breeder flocks, cattle, pigs, sheep, wild animals and birds, as well as in dogs (5). In water and other environments with sub-optimal growth conditions, Campylobacter may convert into a 'viable but non-culturale' state. The importance of this 'state' in the transmission of Campylobacter to animals and people is not clear.

The pathogenesis of Campylobacter is poorly understood. Several determinants of virulence have been described, but their relative roles and importance in the development of diarrhoea are not clear. Enteropathogenic Campylobacter can cause an acute enterocolitis, which is not easily distinguished from illness caused by other enteric pathogens. The incubation period may vary from one to eleven days, but typically lasts between one and three days. The main symptoms are as follows:

- malaise
- fever
- severe abdominal pain
- diarrhoea.

**Fig. 2**

The reported incidence of campylobacteriosis in ten European countries between 1985 and 1998 (63).
Campylobacter infections may be followed by rare but severe non-gastro-intestinal sequelae, e.g. reactive arthritis, Guillain-Barré syndrome and Miller Fisher syndrome. In general, very few deaths are related to Campylobacter infections and these deaths usually occur among infants, the elderly and people whose immune systems are suppressed (2).

In general, most human Campylobacter infections are self-limiting and do not need antimicrobial therapy. However, in severe cases, medication may be necessary. In the beginning of the 1990s, fluoroquinolone-resistant C. jejuni and C. coli emerged in human populations in Europe, as reported in the United Kingdom (UK), Austria, Finland and the Netherlands. This resistance has been linked to the approval of enrofloxacin for the treatment of diseases of broiler chickens. Investigations have shown that fluoroquinolone-sensitive C. jejuni strains were able to convert to resistant forms when fluoroquinolone was added to broiler chicken feed (35). Fluoroquinolone-resistant Campylobacter from chicken and other poultry is an emerging public health problem. Although lower levels of resistance are reported in many countries, the percentage of fluoroquinolone-resistant strains of Campylobacter has been shown to be high in Taiwan (57%), Thailand (84%) and Spain (88%) (40). It is important to realise that the countries reporting such prevalences are not necessarily the countries with the highest real prevalence, since many countries do not have any reporting mechanisms.

In developed countries, all age groups may become infected with Campylobacter. However, in most countries, the reporting rate of campylobacteriosis is higher in young children (from birth to four years old) and young adults. The high incidence rate in children may be a result of higher susceptibility, more frequent exposure (to companion animals, for example), or a higher notification rate in this age group compared to adults, reflecting the fact that parents are more likely to seek medical care for their children. In developing countries, illness is more common among infants and children and it is suggested that young adults and adults have acquired immunity as a result of repeated exposures (70).

**Exposure assessment**

As Campylobacter is common in the faeces of warm-blooded animals, inevitably the meat will be contaminated during slaughter. Thermophilic Campylobacters have an optimum growth temperature of 42°C and no growth below 32°C. Thus, it is reasonable to assume that Campylobacter spp. do not multiply during slaughtering, after processing or during transport and storage. In regard to cattle and pigs, Campylobacter concentrations have been shown to decline during the slaughtering processes. Broiler-chicken processing can reduce levels of Campylobacter on the skin by up to 100-fold (43). In various food items, Campylobacter survival has been recorded after several weeks of storage at 4°C, and after several months in frozen poultry.

In many countries, poultry meat in particular has been found to be contaminated with Campylobacter (up to 75% of all retail chicken samples tested positive). Lower prevalences of Campylobacter have been found in beef, pork, other meat products, raw milk, milk products and fish and fish products. More rarely, Campylobacter has also been found in vegetables.

Outbreaks and sporadic cases seem to have different epidemiological characteristics. For example, sporadic cases seem to peak in summer, whereas outbreaks (based on data from 57 outbreaks in the USA) seem to culminate in May and October (60).

The following major risk factors have usually been associated with outbreaks of campylobacteriosis:

- consumption of poultry
- drinking unpasteurised milk
- contact with untreated surface water
- contaminated public and private water supplies (50).

Cross-contamination of Campylobacter from raw chicken to prepared food has also been identified as a risk factor. A link was observed between infection and not washing the kitchen cutting board with soap (31).

The possible risk factors related to sporadic cases of human campylobacteriosis have been investigated in several case-control studies (38, 50). Most studies have identified handling raw poultry and eating poultry products as important risk factors, which account for a variable percentage of cases. Other food-related risk factors that have repeatedly been identified include consumption of other meat types, consumption of undercooked or barbecued meat, etc. Travel, contact with animals and recreational activities in natural surroundings are also known risk factors (46, 50). Several investigations have pointed out that having contact with companion animals, particularly young animals such as kittens and puppies, significantly increases the risk of Campylobacter infection (9, 50).

Water is an important part of the ecology of Campylobacter. Campylobacter has, in some regions, been isolated from surface water, rivers and lakes at prevalences of up to about 50% during cold winter months (12). The higher winter prevalence is explained by a higher survival rate at low temperatures and in shorter periods of daylight (10). Recent research in Finland indicates that swimming in natural sources of water is an independently associated risk for sporadic Campylobacter infection (58).
Poultry and other foods are thought to be the most likely potential sources of infection in many developed countries. However, in some developing countries, water-borne transmission, direct contact with animals and environmental sources are thought to be the major routes of human infection (70). In other countries, poultry living in the household or within close proximity of the household serve as sources for the organism. For instance, in Peru and Cameroon, chickens in the household have been shown to lead to household members being exposed to Campylobacter infection (39).

Risk characterisation

The incidence rates of Campylobacter infections vary widely, which may partly be explained by differences in surveillance systems. The true rate of infection is higher than the number of reported cases (from 7.6 up to 100 times as high) (67, 44).

The burden of human Campylobacter infections in developing countries is generally not known. It is likely that the rate of campylobacteriosis is high, especially among children below two years of age (11). Campylobacteriosis significantly contributes to malnutrition in infants, as the illness is particularly acute during the weaning period (70).

The FAO/WHO assessment of Campylobacter in broiler chickens presents a model that includes all stages of the chicken production chain, and can be used to generate baseline estimates of the risk of Campylobacter infection on a per-serving basis (25). The model evaluates the impact of various mitigation measures, such as chlorination in chill tanks and freezing, on the level of risk. Each stage of the supply chain is described as a separate module. Since each of these modules can be used separately or in combination, the overall model is potentially adaptable to the specific situation in each country. The baseline model is defined as a system with an overall flock Campylobacter prevalence of 80%, in which chickens are water chilled without free chlorine, and sold fresh (refrigerated but not frozen).

To characterise the risks of a specific system fully, the features of that system must be accurately captured and the data particular to that situation must be applied. It is possible to use alternative data but, in that case, such data must be carefully screened, to ensure that they are appropriate. Every system is likely to be different to some degree, just as every country is likely to have its own particular systems.

Constructing a model of a system (in this case, a chicken production/processing system) and examining it under various scenarios provides valuable insights into how the 'real-life' system would behave under those circumstances. The model can be used to reflect the particular realities that the scientists want to explore. In the FAO/WHO assessment, several scenarios were constructed. The results can be briefly summarised, as follows (25).

a) If the level of Campylobacter contamination is reasonably high, then reducing the frequency with which chickens are contaminated provides a greater return on investment than reducing the level of contamination. However, if the level of contamination is reasonably low, reducing the frequency of contamination is likely to be less effective than reducing the level of contamination further.

b) Reducing the 'between-flock' prevalence at the farm has an effect on risk reduction which is slightly greater than a one-to-one relationship. (In a one-to-one relationship, a percentage change in herd prevalence reduces the expected human risk by a similar percentage.) Thus, reducing the 'between-flock' prevalence by 50% is estimated to reduce the risk by slightly less than 50%. Reducing the within-flock prevalence, but maintaining the overall between-flock prevalence, produces a much lower reduction in risk. This is primarily due to the fact that the birds from these flocks are being processed in an environment in which they are surrounded by infected birds.

c) Reducing surface contamination after evisceration is estimated to have quite a significant effect. A 90% reduction in the surface contamination level after evisceration translates into a 63% reduction in the mean risk overall.

d) Since freezing is known to lower the level of Campylobacter contamination, it is estimated that frozen chickens have a lower risk than those that are sold and stored refrigerated. The difference is best illustrated through a scenario which demonstrates that an equivalent level of risk can be maintained for a more heavily contaminated product if that product is frozen. For instance, the mean risk for refrigerated chicken at a mean contamination level of 4.5 log colony-forming units (CFU) per gram is approximately the same as for frozen chicken at a mean contamination level of approximately 5.25 log CFU per gram.

Risk management

Campylobacter ecology at the poultry herd level is still not clearly understood. There are very few examples of implementing traditional risk management measures in poultry production to lower the risk of campylobacteriosis in the human population.

All elements of risk assessment, as presented above, contribute to providing risk managers with accurate information on the magnitude of the risk in question, as well as on potential mitigation strategies and their expected effectiveness.
Although a new risk management protocol is still being developed by the FAO/WHO Codex Alimentarius Commission, several countries have already attempted to reduce the burden of campylobacteriosis through a structured approach, which includes the scientific insights gained from risk assessments (7). The control measures applied include the following examples:

- in Iceland, the general application of freezing after slaughter
- in Denmark, testing flocks one week before slaughter, and only allowing the distribution of slaughtered birds from a flock as fresh meat after the flock has tested negative for the presence of Campylobacter
- in several countries, separating flocks that test positive for the presence of Campylobacter from flocks that test negative, enabling the distribution of 'Campylobacter-free' poultry meat
- in the USA, reducing Campylobacter contamination of consumer products through the use of water chlorination during poultry slaughter
- in Sweden, lowering the herd prevalence of Campylobacter in poultry production.

In discussing risk management efforts for most food-borne pathogens, the role played by those who finally prepare the food, whether in the home or in professional kitchens, should not be underestimated. For Campylobacter, as for the other pathogens described in this paper, educating the public about a few, relatively simple key precautions when preparing or preserving food would probably have a significant effect on lowering the risk of disease.

As a result, WHO has developed a simple educational campaign based around the 'Five keys to safer food' poster (71), as well as training material aimed at different audiences, such as children, ordinary consumers, street food vendors, etc. In brief, the 'Five keys to safer food' are as follows:

- keep clean
- separate raw and cooked
- cook food thoroughly
- keep food at safe temperatures
- use safe water and raw materials.

Several national food safety authorities are promoting similar messages but a more coherent global effort is needed to encompass more countries and, in particular, developing countries.

Cryptosporidium parvum

Introduction

Cryptosporidium species are intestinal protozoan parasites which are excreted in animal faeces as stable oocysts. Cryptosporidium has been detected in the faeces of a wide range of ruminant and non-ruminant animals, including farmed animals, wild animals, domestic companion animals and birds. It is known that young animals are frequent (faecal) carriers of oocysts of C. parvum. Thus, C. parvum has been identified as a potential hazard for food products or water reservoirs, which are likely to be contaminated with animal faeces or animal production run-off.

This parasite appears to be well adapted to survive and persist in faeces for extended periods, ranging from several weeks to many months. Because of this persistence, faeces are important potential vehicles of transmission within herds, farms, the water chain, the fresh food chain and the wider environment. It was only when the United States Centers for Disease Control and Prevention (CDC) reported Cryptosporidium as the causative agent of severe protracted diarrhoea in acquired immune deficiency syndrome (AIDS) patients that this parasite was recognised as an important human pathogen (15). In the 1990s, it was recognised as one of the leading known causes of water-borne disease outbreaks and an important cause of diarrhoea outbreaks in day-care centres.

Hazard identification and characterisation

Since the first human cases were documented in 1977, C. parvum can be termed an emerging pathogen. Outbreaks have typically been associated with the consumption of contaminated water or food (23, 45). Although there are few reports of food-related outbreaks, since such outbreaks seem difficult to document and are greatly under-reported, water-borne outbreaks have been documented often. Reports of water-borne outbreaks show a combination of causes, including contaminated source water, high turbidity and failures at the treatment plant (23). Other vehicles for transmission also exist, including recreational waters.

To date, the genus Cryptosporidium consists of at least ten recognised species. However, human infection is predominantly caused by C. parvum (23). The incubation period varies significantly but is usually around seven days (72). The disease is usually self-limiting, and symptoms normally last between two and twelve weeks. After infection, abdominal pain, nausea, fever and, in particular, diarrhoea can occur. Both the probability of infection and the probability of illness depend on the viability of the oocysts and the health of the consumer. Immunosuppressed individuals are at greater risk. For these people, especially the elderly and AIDS patients, diarrhoea can be severe, persistent and may even be life-threatening (41).

Current and Garcia (20) reviewed more than 100 studies on the prevalence of Cryptosporidium in people from over 40 countries. They noted that the prevalence rates in the more industrialised countries of North America and
Europe were between 1% and 3%, whereas, in underdeveloped countries, rates ranged from 5% in Asia to 10% in Africa, with some increases during the warmer and/or wetter months.

Models for dose-response relations have been described for *C. parvum* (29, 61). These models are based on data from human feeding trials. The probability of illness is determined by the probability of illness caused by a single oocyst (termed the R-value) multiplied by a mathematical function of the number of oocysts ingested. The function which best fitted the data was an exponential function, thereby leading to an exponential dose-response model. The R-value was estimated to be 0.0042 by Teunis et al. (61). Since infectivity is likely to increase among the AIDS population, Perz *et al.* (55) used a three-fold higher infectivity for people with AIDS.

This dose-response model, however, contains a great deal of uncertainty. Hoornstra and Hartog (32) calculated the mean probability of illness at different doses using the exponential dose-response model, resulting in a mean probability of infection when ingesting 100 viable oocysts of 35%, and a mean probability of infection when ingesting 10 viable oocysts of 5%. The predictions for a few oocysts are uncertain, but the extrapolation of the model will result in a certain probability of becoming ill from a single oocyst, because of the choice of a non-threshold model. The mean probability of infection when the dose is 1 oocyst is 0.4%. The mean probability of illness (cryptosporidiosis) after such infection is 61%, resulting in a probability of illness of 0.25%.

**Exposure assessment**

The organism *C. parvum* can be transmitted to humans through a number of routes, as follows:

- person-to-person infection
- human-to-water-to-human infection
- animal-to-human infection
- water-borne infection (from potable, surface and recreational water)
- food-borne infection.

In fact, *C. parvum* cannot replicate in food, but oocysts can potentially survive in contaminated foods and infect people, either directly, as a result of eating raw or undercooked food, or indirectly, as a result of cross-contamination. Mead *et al.* (44) assumed that 10% of cases in the USA were attributable to food-borne transmission, with the rest caused by the consumption of contaminated water or person-to-person transmission.

Foods of animal origin, including raw milk and/or dairy products, raw meats and fresh fruit and vegetables which have been produced in a contaminated environment, are most likely to pose a risk to humans.

Outbreaks of cryptosporidiosis due to *C. parvum* have recently been linked to dairy products (pasteurised bovine milk and raw goat milk), apple cider and meat products, including chicken salad, frozen tripe and raw sausages (23, 45). It is usually difficult to identify the source of infection due to the relatively long incubation period (2 to 11 days). In addition, the lack of suitable methods for the routine detection of this parasite in foods has also hampered epidemiological investigations (54).

Reduction in viability is an important factor. To evaluate the risk to the consumer, it is also necessary to calculate the numbers of *Cryptosporidium* at the point of consumption and to know the quantity of food consumed.

**Risk characterisation**

Even though Hoornstra and Hartog (32) reported that the mean probability of illness from the consumption of tap water was $1.5 \times 10^{-5}$ per year in the Netherlands, and Perz *et al.* (55) estimated that the median risk of infection from tap water for adults without AIDS was 9 infections per 10,000 persons per year in the USA, there are no quantitative risk assessments of *Cryptosporidium* in food in the literature.

The annual incidence rate was 21.2 cases per 100,000 persons in New Zealand in 2000 (51). Mead *et al.* (44) estimated approximately 300,000 cases of cryptosporidiosis per year in the USA (approximately 100 cases per 100,000 persons). Specific groups at risk of infection included the following:

a) children under five years of age

b) malnourished people

c) a range of immuno-compromised individuals, including:

- AIDS patients
- transplant recipients
- patients receiving chemotherapy for cancer
- institutionalised patients
- patients with immuno suppressive infectious diseases (23).

Various surveys have indicated that the oocyst prevalence in human faeces ranges from 1% to 2% in Europe, 0.6% to 4.3% in North America and 10% to 20% in the developing countries. However, serological evidence of past infections has shown positive rates of 25% to 35% in industrial countries and up to 65% in developing countries (1).
Risk management

To control the transmission of *C. parvum* through the food chain, a ‘farm-to-fork’ approach must be taken, with efforts focused at various points along the chain. On-farm controls should include managing livestock to reduce the transmission of infection between animals and the contamination of the farm environment. Of equal importance are the correct storage and/or treatment of animal wastes to limit the transmission of the parasite to the farm environment, where, as reported above, it may survive for considerable periods. Since *C. parvum* has been involved in major outbreaks, following the contamination of water reservoirs with animal production run-off, on-farm efforts to prevent transmission through animal wastes are seen as crucial. Active treatment processes of animal wastes that are currently used include composting, heat-drying and anaerobic digestion.

Millar et al. (45) indicated that the parasite is introduced to foodstuffs through the following:

- a) contaminated raw ingredients, e.g. unwashed lettuce destined for ‘ready-to-eat’ salads
- b) the addition of contaminated water as an important ingredient of the foodstuff during processing, e.g. in soft drinks production
- c) contamination while cleaning equipment with non-potable water or contaminated potable water during processing
- d) pest infestations, e.g. due to cockroaches, house flies, mice and rats
- e) introduction of the parasite into processed foodstuffs by infected food handlers.

The associated risk from each of these potential entry routes of oocysts into the foodstuff should be controlled through a ‘good hygienic practice’ and ‘hazard analysis critical control point’ system approach.

During food processing, common disease control measures may be successful in reducing or eliminating the parasite. These measures include the following:

- low pH
- freezing
- heating at 55°C for 30 seconds, 60°C for 15 seconds or 70°C for 5 seconds.

It should be noted that a combination of filtration and disinfection is required to control *C. parvum* in water, since chlorination alone has not been successful in eliminating water-borne *C. parvum* oocysts (45). At the domestic level, as with all food-borne pathogens, the risk of cryptosporidiosis can be reduced by adequate cooking of raw foods and good hygiene practices in the home, as in the ‘Five keys to safer food’ poster (71), to avoid the risk of cross-contamination.

Enterohaemorrhagic *Escherichia coli*

Introduction

*Escherichia coli* is a common inhabitant of the gut of humans and warm-blooded animals. Most strains of *E. coli* are harmless. Some strains, however, such as EHEC, can cause severe food-borne disease.

The significance of EHEC as a public health problem was first recognised in 1982, following an outbreak in the USA. The term EHEC was originally defined as those serotypes that cause a clinical illness similar to that caused by *E. coli* O157:H7 and contain similar virulence determinants, including a virulence plasmid that encodes enterohaemolysin (normally referred to as verocytotoxin [VT] – hence the term VTEC for positive strains). Thus, EHEC is now used as a term for VTEC strains that cause haemorrhagic colitis in humans.

In many countries, including the UK and the USA, *E. coli* O157:H7 is currently the most predominant food-borne EHEC. However, it is not the only EHEC strain associated with food-borne disease. A number of other O-serotypes of *E. coli*, such as O26, O103, O111, O118 and O145, cause significant morbidity in many countries (8). Although the overall incidence of disease caused by EHEC is low, the life-threatening complications of infection in young children and the elderly are cause for serious concern. New research in this area seems to indicate that EHEC is fairly prevalent in the bovine intestine, but uncertainties about the isolation, typing and virulence characterisation of this subgroup of the intestinally ubiquitous *E. coli* complicate the picture (69).

Hazard identification and characterisation

Most of the available information on EHEC is related to serotype O157:H7, which is easily differentiated biochemically from other *E. coli* strains because it ferments sorbitol slowly.

Ordinary *E. coli* strains colonise the gut of an infant within a few days of birth, typically from the mother by the faecal-oral route or from the environment. While results from the oral challenge of volunteers suggest that levels of 10⁴ to 10⁶ of other types of pathogenic *E. coli* are necessary for infection, epidemiological evidence
suggests that the risk of disease from EHEC can be high even at very low doses (<1,000 bacteria) (66). This would traditionally be expressed as ‘a very low infectious dose’, but this statement can also be misinterpreted as meaning that any person given a dose of 1,000 EHEC bacteria will become sick. Since this is not the case, scientists prefer to use a measure called the ‘dose-response’, meaning that a certain percentage of a particular group of people who receive a pre-determined amount of EHEC will get sick. This percentage is normally referred to as the ‘attack rate’. The Food Safety and Inspection Service in the USA recently carried out a risk assessment of \textit{E. coli} O157:H7 by comparing the dose-response of EHEC with that of \textit{Shigella dysenteriae}, for which better data exist. Using this model, the 50% attack rate for both bacteria is reached at approximately 750 micro-organisms. However, data from EHEC outbreaks demonstrate that doses below 50 micro-organisms can result in significant attack rates, especially among the young and the old (6).

As stated above, EHEC produces toxins. These are known as verotoxins or Shiga-like toxins because of their similarity to the toxins produced by \textit{Shigella dysenteriae}. The bacterium grows in a temperature range from approximately 7°C to 50°C, with an optimum temperature of 37°C. Some EHEC can grow in acidic foods, down to a pH of 4.4, as well as in foods with a minimum water activity of 0.95. The bacterium is destroyed by thorough cooking, until all parts of the food reach a temperature of 70°C.

Symptoms of the disease caused by EHEC include abdominal cramps and diarrhoea that may, in some cases, progress to bloody diarrhoea (haemorrhagic colitis). Fever and vomiting may also occur. The incubation period can range from 3 to 8 days, with a median of between 3 and 4 days. Most patients recover within 10 days. However, in a small proportion of patients (particularly young children and the elderly), the infection may lead to a life-threatening disease: haemolytic uraemic syndrome (HUS). This syndrome is characterised by acute renal failure, haemolytic anaemia and thrombocytopenia.

Exposure assessment

The reservoir for EHEC appears to be principally the gastro-intestinal systems of cattle and other ruminants.

In individual bovines, investigations seem to indicate a relatively low animal prevalence of EHEC (1% to 2%) (26). However, there seems to be a relatively high, but variable, level of bovine herd prevalence, ranging from 10% in some countries to nearly 100% in feedlot herds in the USA (6). However, new data from the USA seem to show that 28% of the animals presented for slaughter could be infected with \textit{E. coli} O157:H7, and due to additional contamination during the slaughter process, an average total of 43% of animals coming out of the slaughter line are contaminated with the pathogen (6).

Humans are infected with EHEC primarily through the consumption of contaminated foods, such as raw or undercooked ground meat products and raw milk. Faecal contamination of water and other foods, as well as cross-contamination during food preparation (originating from beef and other meat products, contaminated surfaces and kitchen utensils), can also cause infection. Examples of foods implicated in outbreaks of \textit{E. coli} O157:H7 include the following:

- undercooked hamburgers
- dried, cured salami
- unpasteurised fresh-pressed apple cider
- yogurt
- cheese
- milk.

An increasing number of outbreaks are associated with the consumption of fruit and vegetables (such as sprouts, lettuce, coleslaw and salad). Since several EHEC outbreaks have been caused by contaminated sprouts, it was agreed by the Codex Alimentarius Commission to include sprouts in the EHEC risk profile study (18).

Sprout contamination is typically due to contact with faeces from domestic or wild animals at some stage of the cultivation or handling, in particular the practice of using animal slurry as fertiliser on fields after germination. The largest O157 outbreak involved approximately 9,000 Japanese schoolchildren and was caused by contaminated radish sprouts (69). In general, EHEC has been isolated in the environment from soil and bodies of water, especially after the application of animal slurry, and found to survive for months in these environments (52). Water-borne transmission has also been reported, both from contaminated drinking water and from recreational waters.

Person-to-person contact is an important mode of transmission, and occurs through the oral-faecal route (69). An asymptomatic carrier state has been reported, where individuals show no clinical signs of disease but are capable of infecting others. The period during which EHEC is excreted is about one week or less in adults, but can be longer in children. Visiting farms and other venues where the general public might come into direct contact with farm animals has also been identified as an important risk factor for EHEC infection.

Risk characterisation

It is estimated that up to 10% of patients with EHEC infection may develop HUS, which has a case-fatality rate...
ranging from 2% to 7% (69). This syndrome is the most common cause of acute renal failure in young children. It can cause neurological complications (such as seizure, stroke and coma) in 25% of HUS patients and chronic renal sequelae, usually mild, in around 50% of survivors.

The incidence of EHEC infections varies by age group, with the highest incidence of reported cases occurring in children aged under 15 years. Data from developed countries demonstrate that, whereas the general incidence often varies between 0.1 and 2 cases per 100,000, in certain regions and time periods this incidence can increase significantly (69). Data from the USA suggest that more than two thirds of EHEC cases are the result of exposure to the pathogen through food (69). The USA data also suggest that EHEC infections are likely to be significantly under-reported, since the incidence arrived at through these estimations is more like 30 cases per 100,000 (44).

The percentage of EHEC infections which progress to HUS varies between sporadic cases (3% to 7%) and those associated with outbreaks (20% or more). In epidemiological terms, there is generally a background of sporadic cases, with occasional outbreaks. Some of these outbreaks have involved a high number of cases, such as the Japanese outbreak cited above. Data on the situation in developing countries are limited, as surveillance for this pathogen is not routinely conducted.

**Risk management**

Preventing infection requires disease control measures at all stages of the food chain, from agricultural production on the farm to the processing, manufacturing and preparation of foods in both commercial establishments and the domestic environment. The available data are not sufficient to recommend implementing specific disease control measures on the farm to reduce the incidence of EHEC in cattle. Nevertheless, research in this area should be encouraged as disease control measures at the farm level should be guided by strict hygiene rules.

Some countries (e.g. the USA) follow the policy that raw ground beef is considered adulterated if it is found to contain *E. coli* O157:H7. This has led to major recalls of very significant amounts of beef, typically of ground beef, and has most likely heightened producer awareness of the problem.

Preventive measures for *E. coli* O157:H7 infections are similar to those recommended for other food-borne diseases. However, some of these measures may need to be reinforced for EHEC, particularly in view of its importance to vulnerable groups, such as children and the elderly. Food handlers and the general public should follow good hygienic practices, such as those described in the WHO ‘Five keys to safer food’ poster (71).

Although, in general, an effective method of eliminating EHEC from food is to introduce a bactericidal treatment, such as heating (e.g. cooking or pasteurisation) or irradiation, a number of important foods are not easily treated in this way. One very important example is vegetable sprouts, which are produced for consumption without heat treatment. Reports of food-borne outbreaks of EHEC associated with raw sprouts have indicated that the pathogens found on the sprouts most likely originate from the seeds, which can be contaminated by liquid animal manure (slurry). During the germination stage, the low levels of pathogens present on the seeds may quickly reach levels high enough to cause disease. Therefore, specific care is needed, both in the agricultural practices used to produce seeds for sprouting and in sprout production practices.

Since a number of EHEC infections have been caused by contact with recreational water, it is also important to protect such bodies of water from animal wastes. This also applies to sources of drinking water.

**Salmonella species**

**Introduction**

Salmonellosis is one of the most frequently reported food-borne diseases worldwide. In Europe, *Salmonella* trends
have been well-documented over the past 20 years, but in other parts of the world it is rare to find comparable data for such a large group of countries for periods as long as this. According to the WHO surveillance programme for the control of food-borne diseases in Europe (in which fifty countries currently participate), incidences of salmonellosis in Europe have shown a general increase over the past twenty years (62).

Since 1985, there has been a significant increase in the incidence of salmonellosis in many countries. This increase reached a peak in 1992; even earlier in some countries (Fig. 3). Similar salmonellosis disease patterns seem to have evolved in most other regions of the world.

In retrospect, this pattern could be regarded as a global epidemic caused by *Salmonella enteritidis*, which originated primarily in the reservoir of infection building up in intensive poultry production systems. Originally, *S. enteritidis* appeared simultaneously around the world in the 1980s (56). During the 1990s, it was most probably spreading into the poultry production systems of developing countries (42).

In several countries, the incidence of salmonellosis has now decreased due to the implementation of control measures, including greater public awareness of the risk. In other countries, the incidence of salmonellosis continues to increase.

Another example of *Salmonella* spreading through the animal population, followed by a corresponding occurrence of human cases, is demonstrated by the multi-resistant *Salmonella* serovar Typhimurium Definitive Type (DT) 104. From the beginning of the 1990s, S. Typhimurium has spread rapidly through many European countries and North America (and probably elsewhere), mainly in cattle production systems but also, later, in pigs (16). An interesting comparison of the relative increases in DT104 isolates from the bovine and human populations in the north-west of the USA is presented in Figure 4 (21).

Although many different foods have been implicated in salmonellosis outbreaks, most cases are usually caused by the consumption of poultry and eggs. Reflecting this fact, the FAO and WHO have undertaken a risk assessment of *Salmonella* in eggs and broiler chickens, to aid in managing the risks associated with these products (24). Many international experts participated in this project, which will be further referred to below.

**Hazard identification and characterisation**

Over 2,000 serotypes of *Salmonella* have been identified. The most prevalent are *S. enteritidis*, S. Typhimurium and S. Heidelberg.
Exposure assessment

Foods of animal origin, especially poultry, poultry products and raw eggs, are often implicated in sporadic cases and outbreaks of human salmonellosis (13, 34). In addition, recent years have seen an increase in salmonellosis associated with contaminated fruits and vegetables. This contamination is typically due to contact with faeces from domestic or wild animals at some stage during cultivation or handling, in particular, the practice of using animal slurry as fertiliser on the fields after germination. In general, *Salmonella* spp. have been isolated from soil and plants, especially after the application of animal slurry, and found to be able to survive for months (depending on the initial level of contamination) in these environments (49).

The foodstuffs implicated in outbreaks from *Salmonella* spp. in the USA between 1993 and 1997 included, as follows (18):

- eggs (17 outbreaks)
- beef (13 outbreaks)
- ice cream (10 outbreaks)
- chicken (5 outbreaks)
- pork (16 outbreaks).

Using combined typing to link the strains that cause disease with those occurring in different foods, the estimated mean number of sources out of 1,713 cases of human salmonellosis in Denmark in 2003 were, as follows:

- 271 cases from table eggs
- 230 cases from imported poultry

Salmonellosis is characterised by the following symptoms:
- diarrhoea
- fever
- abdominal pain or cramps
- vomiting
- headache
- nausea.

The incubation period ranges from 8 to 72 hours. Symptoms can last up to a week. *Salmonella* infections vary from mild to severe, and are occasionally fatal. Fatalities are more often seen in susceptible populations, i.e. infants, the elderly and the immuno-compromised. A small proportion of infected people may develop Reiter’s syndrome, an arthritic disease characterised by joint pain, eye irritation and painful urination.

The appearance of *Salmonella* strains that are resistant to antimicrobials is added cause for concern. In addition to the spread of *S. Typhimurium DT104*, mentioned above, a more recent spread of *S. Newport* strains, which demonstrate antimicrobial resistance against some of the new ‘last-line’ antimicrobials (i.e. antimicrobials of last resort), is worrying. The general threat caused by the non-human use of antimicrobials, resulting in resistant zoonotic pathogens, has led to recent joint activities by the FAO, the World Organisation for Animal Health and the WHO (75).

The FAO/WHO risk assessment developed a new dose-response model based mainly on data from *Salmonella* outbreaks. This model is considered to be the most appropriate, internationally recognised model for predicting illness after the ingestion of a dose of *Salmonella*. It was based on observed (outbreak) data, with inherent uncertainties. These outbreak data are from a limited number of developed countries and may not apply to other regions (24). The dose-response model and the uncertainties contained in it, as well as the outbreak data used, are presented in Figure 5.
– 202 cases from Danish pork
– 13 from imported pork
– 4 from turkeys
– 36 from broiler chickens
– 17 from Danish beef
– 48 from imported beef
– 74 from outbreaks
– 526 from travel associated sources
– 271 from unknown sources (7).

Important contributing factors in the development of salmonellosis are, as follows:
– inadequate cooking
– cross-contamination
– slow cooling of food
– lack of refrigeration for several hours
– inadequate reheating before serving (24).

The FAO/WHO risk assessment (24) used the following parameters in the production module of the exposure assessment. This assessment predicted the percentage of contaminated eggs among the population of all eggs produced per unit of time:

\[ a) \text{ the flock prevalence (uncertain scalar distribution with parameter of 5\%, 25\% or 50\%) } \]

\[ b) \text{ the percentage of infected hens within infected flocks (log normal distribution; mean: 1.89\%, standard deviation: 6.96\%) } \]

\[ c) \text{ the fraction of eggs laid by infected hens that are contaminated with S. enteritidis (Beta distributed uncertain scalar, with parameter of alpha: 12, beta: 1109). } \]

The dose of S. enteritidis consumed in a meal which contains eggs depends on two factors, as follows:
– the amount of bacterial growth between the time the egg is laid and when it is prepared
– how the egg is prepared and cooked.

The growth of S. enteritidis in contaminated eggs is a function of storage time and temperature. The FAO/WHO risk assessment includes the possibilities of yolk-contaminated eggs and the growth of S. enteritidis in eggs before they are processed for egg products. These issues have not previously been addressed in exposure assessments of S. enteritidis in eggs. Yolk-contaminated eggs might allow more rapid growth of S. enteritidis, compared with eggs that are not yolk-contaminated (24).

The FAO/WHO exposure assessment for broilers estimated the distribution of the average number of CFU of Salmonella per serving for a contaminated chicken which is then undercooked (Fig. 6). Note that, in Figure 6, the interpretation of values of less than 1 CFU per serving is 1 CFU per multiple serving. For example, an average dose of 0.01 bacterial cells per serving can be represented as one serving out of 100 containing a single bacterial cell (24).

**Risk characterisation**

The international data, where available, indicate an estimated human incidence of salmonellosis in 1997 of 14 to 120 cases per 100,000 (64). However, it is likely that even these estimations reflect serious under-reporting. The CDC estimate a total of 1.4 million human cases (corresponding to an incidence of approximately 500 per 100,000) and 582 deaths in the USA annually (44).

In the FAO/WHO risk assessment, the probability of illness (risk) was derived by combining the estimated number of organisms ingested with information on the dose-response relationship. The resulting risk characterisation enabled the subsequent modification of the model parameters, so that the efficacy of risk mitigation strategies that target those parameters could be evaluated.

For broiler chickens, it was found that the relationship between a reduction in the prevalence of Salmonella-contaminated chickens and the corresponding reduction in the risk of human illness is one to one. That is, a percentage change in animal prevalence will reduce the expected human
If management strategies are implemented that affect the level of contamination, i.e. the numbers of *Salmonella* on chickens, the impact on the risk of illness is estimated to be greater than one to one. If the *Salmonella* cell numbers on broiler chickens exiting the chill tank at the end of processing are reduced by 40% on the non-log scale, this reduces the expected risk of illness per serving by approximately 65%.

It was also found that a small reduction in the frequency of undercooking and the magnitude of the undercooking event results in a marked decrease in the expected risk of illness per serving. However, it is important to note that altering cooking practices does not address the risk of illness through cross-contamination. The strategy of changing the cooking practices of the consumer must be tempered by the fact that cross-contamination may, in fact, be the predominant source of the risk of illness, and the nature of cross-contamination in the home is still a highly uncertain phenomenon (24).

The risk of human illness from *S. enteritidis* in eggs increases as flock prevalence increases. However, uncertainty about the predicted risk also increases as flock prevalence increases. Reducing flock prevalence results in a directly proportional reduction in human health risk (e.g. reducing flock prevalence from 50% to 25% results in a halving of the mean probability of illness per serving). Reducing prevalence within infected flocks also results in a directly proportional reduction in human health risk. For example, the risk of illness per serving from eggs produced by a flock with a 1% within-flock prevalence is one-tenth that of a flock with a 10% within-flock prevalence. Adjusting both egg storage time and temperature profiles for eggs from production to consumption was associated with significant effects on the predicted risk of human illness (24).

**Risk management**

Appropriate risk management strategies for controlling salmonellosis can be effectively implemented by the appropriate authority(ies) of each country and should be discussed in the national context. Each country can select those risk management strategies along the entire food production chain which are most appropriate to its particular situation. What is feasible and highly effective for one country might be quite unrealistic and/or ineffective for another. The FAO/WHO *Salmonella* risk assessment provides information that may be useful in determining the impact of various intervention strategies on disease risk from contaminated eggs and poultry (24). Similar risk assessments covering other food groups will reinforce the scientific basis for action.

Different countries have implemented different programmes to lower the risk of salmonellosis. In Sweden, a compulsory programme was established to control *Salmonella* in poultry. It involved the test screening and quarantine of grandparent stock and pre-slaughter test screening of broilers. Disease control measures on parent stock, hatcheries and layers continue to be voluntary in Sweden, but mandatory testing of layers during production and before slaughter has been required since 1994 (47).

In Denmark, from 1988 to 2000, authorities initiated a series of action plans to control human salmonellosis primarily through initiatives on the farm. Following the peaks of human salmonellosis caused by serotypes related to pigs (1988), chicken (1993) and eggs (1997), such action plans did succeed in reducing *Salmonella* prevalence at the farm level and the resulting human disease burden (30). It is interesting to note that measuring the success of these measures was only possible through centrally managed typing regimes (primarily phage typing) of strains from the whole food chain and from human isolates, enabling a ‘pathogen-account’ system attributing the fraction of human disease to foods (see further description below).

Potential intervention programmes for egg production which have been theoretically evaluated in the FAO/WHO risk assessment include ‘test and divert’ systems, vaccination of flocks and refrigeration of eggs. Programmes to test and divert animals which tested positive for the presence of *Salmonella* three times (at the beginning of egg production, four months later and just before flock depopulation), administered to the entire population of egg production flocks for four years, would reduce the risk of human illness from shell eggs by more than 90%. Testing once a year for four years would reduce the risk by over 70%. Vaccination programmes are assumed to be capable of reducing the frequency of occurrence of contaminated eggs by approximately 75%. The effects of time and temperature restrictions were evaluated assuming a flock prevalence of 25%. Restricting shelf-life to fewer than 14 days reduced the predicted risk of illness per serving by a negligible amount (~ 1%). However, keeping retail storage temperatures at no more than 7.7°C reduced the risk of illness per serving by about 60%. If shelf-life were reduced to 7 days, risk per serving would also be reduced by about 60% (24).

When following the success of management programmes, it is important to be able to separate salmonellosis cases according to food source. One interesting new approach involves attempting to link the fraction of human salmonellosis cases to food commodities. This approach compares the relative fractions of the total number of laboratory-confirmed human cases caused by different *Salmonella* serotypes and phage types against the corresponding *Salmonella* types isolated in the various food sources. It is also called a ‘pathogen account’ system (4).

Investigations into the epidemiology of *Salmonella* in the USA used only serotyping but were still able to tentatively
link the outcomes of pathogen-reduction programmes for meat, poultry and eggs to the serotype distributions in humans (53). Other investigations found important differences between the serotype distributions of *Salmonella* isolates taken from food animals after slaughter and the serotype distributions of isolates taken from people in the USA (57).

Although this study could be said to raise questions about raw animal products being the primary source for human salmonellosis, the implications of differences in ecological behaviour and/or in the virulence of different serotypes could also be questioned. In addition, the importance of the relative fractions of salmonellosis caused by a non-meat or non-food source was not dealt with directly. In general the use of new typing methodology to improve linkage capacity is an important new scientific area. Using such methods and linking data, by means of a more formal risk assessment methodology, may enable the broadening of the ‘relative isolate fraction’ approach to other food-borne pathogens as well, resulting in more efficient linkages between foods and patients.

**Toxoplasma gondii**

**Introduction**

Toxoplasmosis is a highly prevalent disease worldwide with many serious, long-term implications. It is caused by the obligate intracellular parasite *Toxoplasma gondii*. National seropositivity figures range from 16% in the general population in the USA (36) to between 40% and 80% in women of child-bearing age in countries in Europe and Africa (3, 22).

The WHO first recognised the importance of toxoplasmosis at an expert meeting in 1968. Almost twenty years later, another meeting was held to discuss the public health implications of the disease (68). Yet today toxoplasmosis remains prevalent worldwide, without a current policy or active programme to manage this disease.

The human disease burden of toxoplasmosis, particularly to foetuses and immuno-compromised people, is probably significant.

**Hazard identification and characterisation**

The obligate intracellular parasite *T. gondii* mainly affects the central nervous system. The definitive host is the cat family Felidae, in whose intestine the parasite undergoes the sexual part of its life cycle. The parasite is transmitted to a wide range of intermediate hosts in birds and mammals, including humans.

Congenital toxoplasmosis, which occurs when a woman becomes infected shortly before or during pregnancy, leads to such foetal symptoms as mental retardation, blindness, cerebral palsy, stillbirth and spontaneous abortion (64). In the immuno-compromised, symptoms include retinoinchoroiditis, heart problems, psychiatric problems and encephalitis. Even normally healthy individuals may be negatively affected. There may also be a link between toxoplasmosis and brain function, with infected men exhibiting lower IQs and less novelty-seeking behaviour than do their healthy counterparts (64).

The public health implications are highly significant. Between 2 and 4 of every 1,000 live births worldwide are affected by congenital toxoplasmosis. Furthermore, acquired toxoplasmosis is one of the principal opportunistic diseases affecting people with the human immunodeficiency virus (HIV) (65).

**Exposure assessment**

Humans can become infected by several different modes of transmission. The primary means is probably through uncooked or undercooked meat, although direct ingestion of cat faecal material through contaminated drinking water or soil can be alternative routes of infection. In developed countries, the food route is thought to be the most important and in fact, in the USA, some 50% of *Toxoplasma* infections are estimated to be food-borne (44). In Europe, a multi-centre case-control study encompassing Naples, Lausanne, Copenhagen, Oslo, Brussels and Milan estimated that 30% to 63% of infections were due to undercooked meat consumption (19). Comparable data from developing countries are generally not available, but the significant increase in meat consumption in these countries seems to indicate that this route of infection will increase in importance in these regions.

The infection route starts when a cat is first infected and begins shedding oocysts in its faeces. These sporulate within one to five days, and remain infective for up to one year in moist conditions, and an indefinite period of time in uncooked meat. The oocysts can be ingested by pigs or sheep, ultimately resulting in cysts in the muscle of these animals. These cysts will then be present in the meat but can be inactivated by either high or low temperatures. Likewise, the sporozoites can be directly ingested by humans who come into contact with contaminated soil.

Human infection thus follows one of several different routes, as follows:

- maternal-foetal transmission after infection of the mother
- consumption of uncooked or undercooked meat or contaminated water
- ingestion of soil
- contact with infected domestic or feral cats.
Risk characterisation

Although it is estimated that perhaps 25% of the general population carry the *Toxoplasma* parasite, few have symptoms because the immune system usually keeps the parasite from causing illness.

Those at the highest risk from acquired toxoplasmosis are those with a compromised immune system, such as HIV-positive people, organ transplant recipients, or those undergoing chemotherapy. The organisation UNAIDS gives figures for the prevalence of toxoplasmosis among those who are HIV-positive:

- 21% in the Côte d’Ivoire
- 14% to 34% in Brazil
- 17% in Mexico
- 2% in Thailand
- 11% in Zaire (65).

Moreover, AIDS patients with toxoplasmosis can show an altered mental status in as many as 60% of cases (64). In the era before highly active anti-retroviral therapy (HAART), acute toxoplasmosis was a symptom of AIDS in up to 30% of cases. The advent of HAART has lowered the numbers of affected HIV-positive individuals, but this remains an area of great concern (28).

Several estimates of the level of risk of congenital toxoplasmosis are available. In the USA, there are estimated to be between 1 in 10,000 and 10 in 10,000 cases of congenital toxoplasmosis in the country each year, leading to between 400 and 4,000 new cases (33). On the other hand, a study in France indicated that 54% of pregnant women were seropositive, leading to 6.6 babies per 1,000 births being born with toxoplasmosis each year (3). A study in Franceville, Gabon, reported that the risk for congenital toxoplasmosis was 3.4 per 1,000 (48). If primary infection of the mother occurs during pregnancy, there is a 40% chance of foetal infection. The transmission rate and severity of infection are related to the gestational age of the foetus at the time of infection. The brain and retina are often affected, and there can be a wide range of clinical disease.

Risk estimates for healthy individuals are not readily available, but seropositivity figures in the general population, mentioned in the introduction to this section above and ranging from 16% to 80%, seem to indicate a relatively high risk of infection. The geographical spread of incidence estimates of several of the different clinical manifestations of toxoplasmosis has been documented. Developing countries often have significantly higher incidence levels. In one specific study from the UK, the general estimated lifetime risk of ocular symptoms in British-born individuals was 18 per 100,000. However, in the same study, people born in West Africa had a 100-fold higher incidence of symptoms than people born in Britain (27).

Risk management

The economic burden of toxoplasmosis is likely to be very high. One study in the USA estimated a cost of US$7.7 billion per year, for congenital infections alone (14). One of the most pressing issues when confronting this disease is the lack of well-designed, controlled studies which analyse the cost effectiveness of the various public health measures implemented to control the disease. The most effective measure is to prevent women acquiring the disease during pregnancy by avoiding risk factors for *T. gondii* infection. Health education may decrease the incidence of toxoplasmosis during pregnancy by 60%.

Screening programmes for pregnant women and neonates do exist in some countries. Since 1976, France has implemented a screening programme involving premarital, pre-natal and post-natal screening, in conjunction with educating pregnant women about the disease (33). When a pregnant woman seroconverts, drug therapy with spiramycin is recommended and, if the foetus exhibits infection, pyrimethamine and sulfadiazine or sulfadoxine may be considered (33).

Educating girls, women of child-bearing age, pregnant women and the immuno-compromised about the dangers of toxoplasmosis, as well as veterinarians and health-care workers, is one of the most cost-effective means of controlling the disease. Basic hygiene precautions, such as thoroughly cooking meat, avoiding cat faeces and washing hands, surfaces, and cooking implements, are simple ways to avoid infection. Promoting the ‘Five keys to safer food’ WHO initiative (71) will also go a long way towards preventing infection. These WHO recommendations are simple rules which promote safer food handling and preparation practices, as follows:

- keep hands, surfaces and utensils clean
- separate raw and cooked foods
- cook food thoroughly
- keep food at safe temperatures
- use safe water and raw materials.

In addition to these more general rules, some specific recommendations should also be mentioned, as follows:

- do not allow cats on surfaces where food is prepared
- wash hands after handling cats and avoid kissing them
- cover outdoor sandboxes, as cats may use them as litter trays.
Toxi-infections alimentaires émergentes à caractère zoonotique

J. Schlundt, H. Toyofuku, J. Jansen & S.A. Herbst

Résumé
Les infections diarrhéiques, provoquées pour la plupart par des micro-organismes pathogènes présents dans les aliments ou dans l’eau, constituent une cause majeure de morbidité et de mortalité dans les pays les moins développés. On estime qu’elles tuent chaque année près de 1,9 million de personnes. Dans les pays développés, jusqu’à un tiers de la population serait touché chaque année par une toxi-infection alimentaire microbiologique. La majorité des micro-organismes pathogènes responsables de ce problème sanitaire d’envergure sont désormais considérés comme des agents pathogènes à caractère zoonotique. L’apparition de certains de ces agents au cours des dernières années semble en nette augmentation.

Si les facteurs responsables de cette augmentation n’ont pas été étudiés de façon approfondie, un lien est généralement établi avec l’évolution des systèmes de production animale et de la chaîne de production alimentaire, notamment, qui modifie le type d’exposition et de sensibilité au sein de la population humaine.

L’objet de cet article n’est pas d’offrir une analyse plus approfondie de ces facteurs, mais de décrire succinctement cinq agents pathogènes émergents à caractère zoonotique parmi les plus importants : Salmonella spp., Campylobacter spp., Escherichia coli entérohémorragique, Toxoplasma gondii et Cryptosporidium parvum. Il ne vise pas une description exhaustive des principaux agents pathogènes émergents à caractère zoonotique, mais propose une synthèse de la situation au regard de ces agents pathogènes, les plus importants sur le plan mondial. Chaque agent pathogène est décrit conformément au nouveau système d’évaluation des risques microbiologiques établi conjointement par l’Organisation des Nations unies pour l’alimentation et l’agriculture (FAO) et l’Organisation Mondiale de la Santé (OMS), qui prévoit l’identification et la caractérisation des dangers, l’évaluation de l’exposition et la caractérisation du risque. Les auteurs présentent en outre un bref compte rendu des actions entreprises en vue de réduire les risques, ainsi que des suggestions en matière de gestion des risques pour certains des agents pathogènes s’appuyant sur des évaluations approfondies réalisées par la FAO et l’OMS à l’échelle internationale. Ils soulignent qu’en matière de prévention des toxi-infections alimentaires, la diminution durable du risque pour la santé humaine passe obligatoirement par des programmes scientifiquement fondés de réduction des agents pathogènes à certaines étapes pertinentes de la chaîne de production alimentaire dans son ensemble, « de l’étable à la table ».

Mots-clés
Las enfermedades diarreicas, provocadas casi todas por patógenos microbianos transmitidos por los alimentos o el agua, constituyen una de las principales causas de morbilidad y mortalidad en los países menos desarrollados y son responsables, según las estimaciones, de la muerte de 1,9 millones de personas al año. Incluso en los países desarrollados se calcula que hasta un tercio de la población se ve afectada cada año por enfermedades microbianas de transmisión alimentaria. Hoy se considera que los patógenos causantes de esta importante morbilidad son en su mayoría patógenos zoonóticos. De unos años a esta parte, algunos de ellos parecen presentarse con una frecuencia sensiblemente mayor.

Aunque no se han estudiado a fondo los factores relacionados con ese aumento, en general se piensa que los más importantes son la evolución de los sistemas de producción animal y los cambios en la cadena de producción alimentaria, factores ambos que alteran los patrones de exposición y sensibilidad de las poblaciones humanas.

Los autores no pretenden analizar en detalle esos factores, sino describir brevemente cinco de los principales patógenos zoonóticos emergentes que se transmiten por vía alimentaria: Salmonella spp., Campylobacter spp., Escherichia coli enterohemorrágica, Toxoplasma gondii y Cryptosporidium parvum. No se trata pues de describir exhaustivamente todos los patógenos de ese tipo sino de exponer a grandes líneas la situación actual con respecto a los mencionados patógenos, que figuran entre los más importantes a escala mundial. Los autores describen cada microorganismo con arreglo a un modelo de determinación del riesgo microbiológico de la Organización de las Naciones Unidas para la Agricultura y la Alimentación (FAO) y la Organización Mundial de la Salud (OMS), que integra la identificación y caracterización de los peligros, la evaluación de la exposición y la caracterización de los riesgos. Además, los autores dan cuenta brevemente de una serie de iniciativas para tratar de reducir el riesgo, y formulan propuestas para gestionar los riesgos derivados de algunos de los patógenos a partir del modelo FAO-OMS. Por otro lado recalcan que, en lo que concierne a las enfermedades prevenibles transmitidas por vía alimentaria, la reducción duradera de los riesgos que presentan para la salud humana pasa necesariamente por programas dotados de sólidos fundamentos científicos que sirvan para reducir la presencia de patógenos en los puntos pertinentes de la cadena de producción “del campo a la mesa”.

Palabras clave
References


