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Food Safety: Emerging Pathogens

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Glossary

Biofilms Multicellular communities (i.e., cell aggregates) that provide bacteria with the ability to grow adhered to biotic and abiotic surfaces.
Clones (or clonal groups) Genetically related (identical or similar) isolates of an organism derived from a single common ancestor.
Genotype The genetic information dictating a particular trait.
Phage type A set of bacterial strains susceptible to the same bacteriophages.
Phenotype Visible, expressed trait influenced both by the genetic information and the environment.
Quorum sensing Cell-to-cell communication in the context of which gene expression is regulated in response to changes in cell population density.
Serotype Group of isolates distinguished from others by the type of expressed surface antigens.
Shiga toxins Family of related toxins that inhibit protein synthesis, originally described to be produced by *Shigella dysenteriae*.
Strain An isolate (or group of isolates) that can be distinguished from other isolates of the same species by phenotypic or genotypic characteristics.
Zoonosis Any disease that can be transmitted from animals to humans.

Introduction

The appearance (or emergence) of new or unexpected pathogens in foods have been identified as one of the most important trends likely to affect food safety in the next 50 years (Tauxe *et al.*, 2010). Different, and often confusing, definitions have been proposed for 'emerging pathogens.' For instance, an emerging pathogen has been defined as a pathogen that is linked to a novel and serious to public health disease (Smith and Fratamico, 1995). In other cases, the term 'emerging' has been used to describe the appearance of microbial strains that have developed enhanced resistance to stresses and have adapted to new environments (Mor-Mur and Yuste, 2010). It has been proposed that, with particular reference to foodborne pathogens, the terms 'new,' 'evolving,' 'emerging,' and 'reemerging' should be differentiated and considered separately. In this sense, 'new foodborne pathogens' are serious hazards for public health and important causal agents of outbreaks that have not been previously described, whereas 'evolving foodborne pathogens' are those that become more potent (i.e., increased involvement in foodborne outbreaks) or more associated with other food products as well as those that were already known but not recognized as agents of human illness (Mor-Mur and Yuste, 2010). Finally, 'emerging foodborne pathogens' are foodborne pathogens that have newly arisen, meaning that they may have been recognized as pathogens but only recently are associated with foodborne transmission, whereas the 'reemerging' had been known for some time, but had fallen to low levels and are now showing increasing trends (Mor-Mur and Yuste, 2010; Sofos, 2008).

The emergence (or reemergence) of foodborne pathogens is a complex process and depends on the interaction of multiple factors including the following: (1) changes in agricultural practices (e.g., increased use of antibiotics in animal production); (2) microbial adaptation and evolution (e.g., enhanced virulence); (3) technological changes in the food industry (e.g., production, processing, packaging, and handling); (4) changes in human behavior and particularly in people's eating habits (e.g., increased consumption of raw/undercooked or minimally processed foods); (5) changes in demographics (e.g., migration, urbanization, aging of the population, and increasing number of people with conditions that result in immunosuppression); (6) health care and public health infrastructure; (7) environmental parameters (e.g., climate changes); (8) the global trade in foods leading to greater interdependence on the food safety systems of different countries; and (9) development of improved and more sensitive methodologies for the isolation, detection, and identification of foodborne pathogens (Miller *et al.*, 1998; Smith and Fratamico, 1995; Schofield, 1992; Tauxe *et al.*, 2010). In general, virtually any change affecting directly or indirectly the food chain is expected to create a selection pressure that will ultimately result in the emergence of foodborne pathogens (Miller *et al.*, 1998; Smith and Fratamico, 1995).

This article describes the characteristics, epidemiology, prevalence in foods, transmission routes to humans, and means of control of pathogens that are foodborne or have the potential to be foodborne, with a particular emphasis being placed on bacterial pathogens.

Bacteria

Aeromonas spp.

Aeromonas spp. are Gram-negative, rod-shaped, facultatively anaerobic, catalase-positive, chemoorganotrophic, nonspore-forming bacteria, and most of them are motile by polar flagella (Igbinosa *et al.*, 2012; Isonhood and Drake, 2002). This genus is characterized by considerable phenotypic variation, with gas

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production being variable and often temperature dependent, whereas marked differences can also be observed in cellular morphology (Forsythe and Varnam, 2009). Aeromonads are easily differentiated from the bacterial species of the family Enterobacteriaceae, with which they share many biochemical characteristics, based on their positive oxidase reaction (Igbinosa *et al.*, 2012). The taxonomy and nomenclature of the genus *Aeromonas* have been complex and have undergone several changes over time (Igbinosa *et al.*, 2012; Janda and Abbott, 2010). Although formerly positioned in the family Vibrionaceae, the genus *Aeromonas* is now officially classified within the family Aeromonadaceae (Igbinosa *et al.*, 2012; Merino *et al.*, 1995). Furthermore, four *Aeromonas* species were originally identified: *Aeromonas hydrophila*, *Aeromonas sobria*, *Aeromonas caviae*, and *Aeromonas salmonicida*. However, distinct biochemical and genetic (i.e., deoxyribonucleic acid (DNA) hybridization) groups, referred to as phenospecies and genospecies, respectively, were later identified, resulting in significant reconsiderations on species designation and classification (Isonhood and Drake, 2002; Janda and Abbott, 2010). Currently, the genus comprises more than 15 species, with the latest research data indicating the existence of 13 phenospecies and 19 genospecies (Janda and Abbott, 2010).

Extensive variability in the optimum growth temperature of *Aeromonas* spp., with the latter growing optimally at temperature ranges between 22 and 35 °C (Igbinosa *et al.*, 2012; Isonhood and Drake, 2002). Indeed, optimum growth temperature has been one of the phenotypic markers (along with motility, production of indole, and elaboration of a melanin-like pigment on tyrosine agar) traditionally used for the differentiation of species of the genus into two major groups based on physiological properties and host specificity: (1) motile aeromonads, which grow optimally at 35–37 °C, are predicted to cause human infections and are represented by *Ae. hydrophila* and (2) nonmotile aeromonads, which grow optimally at 22–28 °C, are associated primarily with fish infections and are represented by *Ae. salmonicida* (Igbinosa *et al.*, 2012; Joseph and Carnahan, 2000). A few species can exhibit growth at a wide temperature range; for instance, *Ae. hydrophila* is capable of growing at temperatures ranging from 1 to 42 °C (Isonhood and Drake, 2002). The ability of many strains of the genus to grow at refrigeration temperatures (4–5 °C) has been well-established and long acknowledged as one of the most important factors contributing to the public health significance of aeromonads in foods (Buchanan and Palumbo, 1985; Kirov, 1993; Knøchel, 1990). Most *Aeromonas* spp. tolerate high pH well and can resist pH values ranging from 4.5 to 9.0, with the optimum pH being in the range 5.5–9.0 (Igbinosa *et al.*, 2012; Isonhood and Drake, 2002; Merino *et al.*, 1995). In general, aeromonads do not tolerate NaCl concentrations higher than 5% (Knøchel, 1990), whereas the lowest water activity (a_w) value allowing growth varies with the humectant (Merino *et al.*, 1995).

Aeromonas spp. are ubiquitous organisms, having the potential to be isolated from a wide range of environmental niches including aquatic habitats (surface water, groundwater, chlorinated, and nonchlorinated drinking water), natural soils, fish, foods, domesticated pets, invertebrate species, birds, and insects (Igbinosa *et al.*, 2012; Janda and Abbott, 2010). They are also found in raw sewage, sewage effluents, and sewage-contaminated waters and activated sludge (Dumontet *et al.*, 2001; Igbinosa *et al.*, 2012). Aeromonads have long been considered as opportunistic pathogens of both aquatic (warm- and cold-water fish) and terrestrial animals (Harikrishnan and Balasundaram, 2005; Isonhood and Drake, 2002; Queiroga *et al.*, 2012). Nonetheless, the motile mesophilic aeromonads, and particularly *Ae. hydrophila*, have been recently identified as emerging human pathogens, gaining continuously increasing public health recognition as potential causative agents of both gastrointestinal and extraintestinal infections, primarily in immunocompromized individuals (Cabral, 2010; Igbinosa *et al.*, 2012; Isonhood and Drake, 2002; Janda and Abbott, 2010; Merino *et al.*, 1995; Senderovich *et al.*, 2012).

Although not supported by the findings of volunteer human feeding studies, epidemiological data (i.e., presence of the organisms in the stools of individuals with diarrhea, in the absence of other known enteric pathogens) have frequently suggested aeromonads as putative enteropathogens (Forsythe and Varnam, 2009). Most human pathogenic strains are now recognized as being grouped into three genospecies: *Ae. hydrophila* hybridization group (HG) 1, *Ae. caviae* HG 4, and *Aeromonas veronii* biovar sobria HG 8 (Forsythe and Varnam, 2009). Gastroenteritis associated with aeromonads may vary in severity from mild, self-limiting diarrhea to dysentery or cholera-like illness with the latter being potentially life threatening (Forsythe and Varnam, 2009; Igbinosa *et al.*, 2012). Although *Aeromonas* spp. have been recognized as emerging human pathogens, their exact role as enteric pathogens has not been definitely established; their mechanisms of pathogenicity remain vague and their infectious dose is unknown (Forsythe and Varnam, 2009; Igbinosa *et al.*, 2012; Isonhood and Drake, 2002). Several putative virulence factors of *Aeromonas* spp. that can be associated with gastroenteritis have been identified including hemolysins, invasins, adhesins, endotoxin (or lipopolysaccharide), proteases, fimbriae, pili, capsular polysaccharides, S-layers, siderophores, and various extracellular enzymes (Igbinosa *et al.*, 2012; Isonhood and Drake, 2002; Merino *et al.*, 1995). Extraintestinal human infections associated with aeromonads include septicemia, meningitis, cellulitis, myonecrosis, peritonitis, hepatitis, pancreatic abscesses, respiratory, urogenital and eye infections, endocarditis, osteomyelitis, and septic arthritis (Forsythe and Varnam, 2009; Janda and Abbott, 2010; Roberts *et al.*, 2006; Talon *et al.*, 1998). At greatest risk for *Aeromonas* infections are people with predisposing conditions (e.g., deficient immune system, leukemia, and liver disease) as well as young (6 months to 2 years old) children (Forsythe and Varnam, 2009; Gracey, 1994).

The most common routes of infection suggested for *Aeromonas* spp. are the ingestion of contaminated water (drinking or natural mineral water) or food, and contact of the organisms with a break in the skin (e.g., when swimming in contaminated water) (Cabral, 2010; Igbinosa *et al.*, 2012). Despite the fact that the relative importance of water and food in *Aeromonas* infections has been the subject of considerable discussion, these two sources are most likely interrelated; given the strong association of aeromonads with water, it has been suggested that risk of human exposure is greatest through consumption of contaminated water, or food processed with contaminated water (Forsythe and Varnam, 2009). With particular reference to foods of animal origin, also significant in

the transmission of aeromonads to humans is expected to be the contribution of aeromonad-contaminated animals (symptomatic or not), with animal feces appearing to be the major source of contamination of foods (Igbinosa *et al.*, 2012). Furthermore, given their wide environmental distribution in conjunction with their ability to form biofilms, which may provide increased resistance to conventional bactericidal treatments, *Aeromonas* spp. may establish niches in food-processing equipment, with the latter potentially serving as a source of cross-contamination of foods in the absence of sufficient cleaning and sanitation (Cotton and Marshall, 1998; Isonhood and Drake, 2002).

Aeromonas spp. have been isolated from a wide range of foods of both plant and animal origin including fresh vegetables, fish, shellfish, meat, poultry, and dairy products (**Table 1**). Despite their frequent presence in foods, *Aeromonas* isolates may be nontoxigenic questioning their foodborne pathogen potential (Kirov, 1993). Indeed, *Aeromonas* spp. occur commonly on minimally processed produce items as well as on fresh fish, meat, and poultry as part of their normal spoilage microflora (Forsythe and Varnam, 2009; Jacxsens *et al.*, 1999; Samelis, 2006). Although potentially pathogenic, genospecies of *Aeromonas* have been occasionally isolated from food samples (Neyts *et al.*, 2000; Forsythe and Varnam, 2009), the link between food contamination and human disease can be definitely established only via confirmed epidemiological data. Nevertheless, such data are limited as only few foodborne outbreaks associated with *Aeromonas* spp. have been documented with the majority of them involving seafood (Altwegg *et al.*, 1991; Ghenghesh *et al.*, 2008; Isonhood and Drake, 2002; Kirov, 1993). A recent foodborne outbreak of *Ae. hydrophila* was reported in a college in China; more than 200 students were reported to be sick with acute diarrhea and, as supported by the findings of the conducted epidemiological investigation, the most probable source of the organism was salad ingredients washed in contaminated tank water (Qian *et al.*, 2012).

In continuation to its initial recognition as an agent of human illness, the genus *Aeromonas* is being considered as a pathogen of emerging importance due to a number of special features, including its ubiquitous presence in water and food, the abundance of virulence factors, and the psychrotrophic nature of many of its isolates (Smith and Fratamico, 1995; Vivekanandhan *et al.*, 2005). Another issue of major importance for the public health significance of *Aeromonas* spp. is the increasing documentation of isolates exhibiting resistance to several antimicrobial agents (Queiroga *et al.*, 2012). Hence, in the context of basic control procedures common for all foodborne pathogens (i.e., prevention of contamination, reduction of contamination, and prevention of microbial growth), the aforementioned issues need to be particularly addressed with regard to aeromonads. Water used for washing of food, and particularly of food products intended to be consumed raw such as fresh or minimally processed produce items, should be chlorinated or otherwise disinfected and care should be taken to ensure that water distribution systems are not colonized by *Aeromonas* spp. (Forsythe and Varnam, 2009). Although managing their growth in biofilms can be very difficult, the entry of aeromonads into water distribution systems can be significantly reduced through effective treatment and maintenance procedures, such as maintaining temperatures below 14 °C, providing free-chlorine levels above 0.1–0.2 mg l^{-1}, and limiting the levels of organic carbon compounds in the water (Igbinosa *et al.*, 2012). Regarding the control of the organisms in aquaculture systems, and, thus, in fish and seafood, proper disposal of diseased animals, maintaining high standards of water quality, temperature control, and disinfection of equipment are expected to be useful and effective approaches (Igbinosa *et al.*, 2012). Moreover, disease prevention by means of vaccination and immunostimulation of fish in aquaculture has been shown to be successful against several bacterial pathogens, including *Aeromonas* spp. However, alternative control approaches in aquaculture such as the application of probiotics and herbals may also be promising, while allowing at the same time for reduced cost of disease management compared with the use of antibiotics, chemicals, and vaccinations, as well as for reduced incidence of multidrug-resistant (MDR) *Aeromonas* strains (Harikrishnan and Balasundaram, 2005). Given that aeromonads are not particularly heat or acid resistant, they do not exhibit unusual resistance to conventional food-processing procedures (Isonhood and Drake, 2002), whereas nonthermal processing technologies such as irradiation are also expected to be effective against these organisms on various types of foods (Nagar and Bandekar, 2011). Therefore, as also supported by the findings of epidemiological investigations, particular emphasis needs to be placed on the prevention of contamination/recontamination of foods with the organisms via the implementation of appropriate sanitary measures such as proper food handling practices and efficient sewage disposal systems (Igbinosa *et al.*, 2012; Qian *et al.*, 2012). Furthermore, the implementation of improved diagnostic and detection procedures appears to be essential for proper surveillance of water, food, and sanitation facilities, as well as of human infections which may be considerably underestimated particularly in developing countries (Igbinosa, *et al.*, 2012; Qian *et al.*, 2012). The development of novel or the improvement of existing molecular-based techniques is expected to be very useful toward this direction, allowing for an enhanced detection of aeromonads and, thus, for clarification of their true role as pathogens (Ghatak *et al.*, 2012; Tichoniuk *et al.*, 2010). Finally, given that the pathogenesis of *Aeromonas* spp. is multifactorial, with a large number of virulence genes being identified and quorum-sensing signal molecules being potentially associated with the expression of virulence determinants, research on these fields of study will provide a better understanding of the mechanism(s) underlying the emergence of these organisms as human pathogens and help to develop effective diagnostics and novel therapeutics (Chan *et al.*, 2011; Yu *et al.*, 2005).

Arcobacter spp.

Arcobacter species are Gram-negative, spiral, curved to S-shaped, fastidious, and nonspore forming microorganisms belonging to the family Campylobacteraceae (Vandamme and De Ley, 1991). They are motile by a single unsheathed polar flagellum, exhibiting darting or corkscrew movement (Blackburn and McClure, 2009). The organisms were first isolated from aborted bovine and normal porcine fetuses, sows with

Table 1 Prevalence of certain emerging bacterial pathogens in some foods

Bacterial agent	*Food*	*Country*	*Prevalence (%)*[a]	*Reference*
Aeromonas spp.	Chicken	Turkey	86.9	Yucel and Çitak (2003)
	Fish	India	33.6	Vivekanandhan *et al.* (2005)
		India	26.6; salted and dried finfish	Udgata *et al.* (2009)
		Nigeria	67.0; fresh fish	Igbinosa *et al.* (2006)
			70.0; smoked fish	
	Meat	Nigeria	54.0	Igbinosa *et al.* (2006)
		Turkey	67.7; minced meat	Yucel and Çitak (2003)
	Meat products	Nigeria	80.0	Igbinosa *et al.* (2006)
	Milk	Nigeria	85.0; raw milk	Igbinosa *et al.* (2006)
		Turkey	47.7; raw milk	Yucel and Çitak (2003)
			16.1; pasteurized milk	
	Poultry	Nigeria	80.0	Igbinosa *et al.* (2006)
	Prawns	India	17.6	Vivekanandhan *et al.* (2005)
	Shrimp	Nigeria	60.0	Igbinosa *et al.* (2006)
	Vegetables	Nigeria	35.0	Igbinosa *et al.* (2006)
Arcobacter spp.	Beef	Belgium	31.3	Collado *et al.* (2009)
		Malaysia	38.0	Shah *et al.* (2011)
	Chicken	Belgium	64.3	Collado *et al.* (2009)
		Korea	21.1	Lee *et al.* (2010)
		Malaysia	39.0	Amare *et al.* (2011)
	Clams	Belgium	100.0	Collado *et al.* (2009)
	Duck meat	Belgium	40.0	Collado *et al.* (2009)
	Ground beef	Belgium	9.0	De Smet *et al.* (2010)
		Turkey	37.0	Aydin *et al.* (2007)
	Milk	Belgium	3.2	Pianta *et al.* (2007)
		Northern Ireland	46.0	Scullion *et al.* (2006)
	Mussels	Belgium	41.1	Collado *et al.* (2009)
	Pork	Belgium	21.0	Van Driessche and Houf (2007a)
			53.0	Collado *et al.* (2009)
	Rabbit meat	Belgium	10.0	Collado *et al.* (2009)
	Turkey meat	Belgium	33.3	Collado *et al.* (2009)
Clostridium difficile	Chicken	Canada	12.8	Weese *et al.* (2010)
		The Netherlands	2.7	De Boer *et al.* (2011)
	Fish	Canada	9.1	Metcalf *et al.* (2011)
	Ground beef	Canada	20.8	Rodriguez-Palacios *et al.* (2007)
		Canada	6.7	Rodriguez-Palacios *et al.* (2009)
		US	50.0	Songer *et al.* (2009)
	Ground meats	Austria	3.0	Jöbstl *et al.* (2010)
	Ground pork	US	42.9	Songer *et al.* (2009)
	Ground turkey	US	44.4	Songer *et al.* (2009)
	Ground veal	Canada	14.3	Rodriguez-Palacios *et al.* (2007)
	Lamb	The Netherlands	6.3	De Boer *et al.* (2011)
	Pork	Canada	1.8	Metcalf *et al.* (2010)
		US	9.5	Harvey *et al.* (2011)
	Pork sausage	US	23.1	Songer *et al.* (2009)
	Salads	Scotland	7.5	Bakri *et al.* (2009)
	Scallops	Canada	33.3	Metcalf *et al.* (2011)
	Shrimp	Canada	15.4	Metcalf *et al.* (2011)
			33.3 (frozen)	
			10.0 (cooked)	
	Summer sausage	US	14.3	Songer *et al.* (2009)
	Veal chops	Canada	4.6	Rodriguez-Palacios *et al.* (2009)
	Vegetables	Canada	4.5	Metcalf *et al.* (2010)
Cronobacter spp.	Cereals/cereal products	Czech Republic	15.1	Hochel *et al.* (2012)
		The Netherlands	4.9	Kandhai *et al.* (2010)
	Cereal-based follow-up formula	South Korea	6.0	Kim *et al.* (2011)
	Cheese products	UK	3.2	Iversen and Forsythe (2004)
	Dried infant foods	UK	10.2	Iversen and Forsythe (2004)
	Eggs	Czech Republic	10.0	Hochel *et al.* (2012)
	Grains	South Korea	18.0	Chon *et al.* (2012)
	Herbs and spices	Czech Republic	13.5	Hochel *et al.* (2012)

(*Continued*)

Table 1 Continued

Bacterial agent	*Food*	*Country*	*Prevalence (%)*[a]	*Reference*
		The Netherlands	3.6	Kandhai *et al.* (2010)
		South Korea	19.2	Chon *et al.* (2012)
		UK	32.8	Iversen and Forsythe (2004)
	Legumes	Czech Republic	27.5	Hochel *et al.* (2012)
	Marine products	South Korea	7.5	Chon *et al.* (2012)
	Minced meats	The Netherlands	3.2	Kandhai *et al.* (2010)
	Powdered infant formula	South Korea	5.3	Kim *et al.* (2011)
		The Netherlands	2.3	Kandhai *et al.* (2010)
	Powdered infant formula milk	UK	2.4	Iversen and Forsythe (2004)
	Powdered milk	Czech Republic	10.0	Hochel *et al.* (2012)
		The Netherlands	4.0	Kandhai *et al.* (2010)
		UK	4.2	Iversen and Forsythe (2004)
	Seeds	Czech Republic	41.2	Hochel *et al.* (2012)
	Vegetables	The Netherlands	4.3	Kandhai *et al.* (2010)
		South Korea	30.0	Chon *et al.* (2012)

[a]Percentage (%) of positive samples.

reproductive problems, and asymptomatic pigs (Ellis *et al.*, 1977, 1978; Neill *et al.*, 1978, 1979). The genus *Arcobacter* was proposed by Vandamme *et al.* (1991) to describe those organisms formerly designated 'aerotolerant campylobacters,' was classified along with the genera *Camplylobacter* and *Helicobacter* within the ribosomal ribonucleic acid (rRNA) Superfamily VI, and currently includes 12 recognized species: *Arcobacter butzleri*, *Arcobacter cryaerophilus*, *Arcobacter skirrowii*, *Arcobacter nitrofigilis*, *Arcobacter cibarus*, *Arcobacter halophilus*, *Arcobacter molluscorum*, *Arcobacter defluvii*, *Arcobacter marinus*, *Arcobacter trophiarum*, *Arcobacter mytili*, and *Arcobacter thereius* (Shah *et al.*, 2011). *Arcobacter* spp. can grow at temperatures ranging from 15 to 42 °C, at pH values between 5.5 and 9.5, and under both aerobic and anaerobic conditions with their optimal, however, growth occurring under microaerophilic conditions (i.e., 3–10% oxygen) (Blackburn and McClure, 2009; Vandamme *et al.*, 1991). The ability of arcobacters to grow at 15 °C under aerobic conditions is the basis for their differentiation from campylobacters with which they have similar morphological, metabolic, and several other phenotypic and genotypic features (Blackburn and McClure, 2009; Shah *et al.*, 2011).

Livestock animals, and primarily poultry and swine, constitute significant reservoirs of *Arcobacter* spp. (Phillips, 2001; Snelling *et al.*, 2006; Van Driessche *et al.*, 2004). Arcobacters have commonly been isolated from feces and rectal swabs of clinically healthy cattle, sheep, and horses at prevalences ranging from 3.6% to 41.7% (Shah *et al.*, 2011). According to the findings of De Smet *et al.* (2011), healthy small ruminants (i.e., primarily sheep and to a smaller extent goats) are important carriers of these organisms. In general, the presence of *Arcobacter* spp. in the feces of healthy livestock at slaughter poses an important risk of carcass, meat, and possibly milk (in the case of ruminants) contamination (De Smet *et al.*, 2011; Van Driessche *et al.*, 2003). Nevertheless, and despite their frequent isolation from poultry carcasses, organisms of the genus *Arcobacter* have rarely been isolated from the intestinal content of poultry, rendering the fecal origin of carcass contamination questionable and suggesting that contamination may occur at the postslaughter level (Houf and Van Driessche, 2007; Phillips, 2001; Van Driessche and Houf, 2007b; Van Driessche *et al.*, 2003). As demonstrated by the results of a study assessing the distribution of arcobacters in chickens, the organisms were isolated from neck skin samples but not from the intestinal tract or from the feathers; however, the way of sample collection and the time period for sample processing were identified as crucial parameters for the interpretation of such findings (Houf and Van Driessche, 2007). In addition to livestock, *Arcobacter* spp. have been isolated from wild and nondomesticated animals (e.g., raccoons, rhinoceroses, and gazelles) as well as from pets such as dogs and cats (Fera *et al.*, 2009; Houf *et al.*, 2008; Shah *et al.*, 2011). In spite of their natural occurrence in healthy animals, arcobacters have also been associated with animal infections, with their main clinical manifestations including abortion, mastitis, and enteritis (Phillips, 2001; Wesley, 1997). *Arcobacter* spp. have also been found in different water sources (e.g., sea, lake, river, canal, and groundwater), with the latter assumed to play a significant role in the transmission of the organisms to both animals and humans (Phillips, 2001; Snelling *et al.*, 2006; Shah *et al.*, 2011). More specifically, *Ar. butzleri* has been isolated from canal water, from well-water sources, as well as from water samples in water treatment plants from all stages of processing (Phillips, 2001). However, given the organism's sensitivity to chlorine, its presence in water is probably the result of either inadequate chlorination or posttreatment contamination (Phillips, 2001; Wesley, 1997). Furthermore, *Ar. butzleri* has been found in various types of sewage sludge (Phillips, 2001).

For a long time, the importance of *Arcobacter* spp. as human pathogens was uncertain, and still, very little is known about the epidemiology, pathogenesis, and real clinical significance of these organisms. The lack of a standard protocol for primary isolation and of routine screening procedures, as well as the similarity of the symptoms of *Arcobacter* infections with campylobacteriosis (i.e., *Campylobacter jejuni* infection) have hindered the assessment of infection rates (resulting in underestimation of infections) and the establishment of a definitive association between human illness and the pathogenicity of these organisms (Phillips, 2001; Shah *et al.*, 2011). On the basis of the findings of studies undertaken in Belgium and

France, *Ar. butzleri* was the fourth most common *Campylobacter*-like organism isolated from human stools, whereas *Arcobacter* presence has also been recorded in other countries such as Thailand and South Africa (Shah *et al.*, 2011). Indeed, predominantly *Ar. butzleri* and to a smaller extent *Ar. cryaerophilus* and *Ar. skirrowii* are the species with the strongest association with human disease (Shah *et al.*, 2011; Vanderberg *et al.*, 2004; Wesley, 1997). *Arcobacter* infections are mainly manifested in the form of enteritis with its main clinical symptoms being persistent and watery diarrhea with abdominal pain (at a higher frequency compared with campylobacteriosis), nausea, vomiting, and fever (Snelling *et al.*, 2006; Vanderberg *et al.*, 2004). In addition to its association with enteritis, it has been suggested that *Ar. butzleri* has the potential to invade other parts of the body and cause considerable complications. Indeed, the organism has been isolated from patients with liver cirrhosis and acute gangrenous appendicitis, from septicemic patients, as well as from the blood of uremic patients with hematogenous pneumonia (Shah *et al.*, 2011). Certain factors such as health status, age, and hypertension may predispose a person to *Arcobacter* infection (Shah *et al.*, 2011). The currently available knowledge regarding the dose response and pathogenicity of *Arcobacter* spp. is still very limited. Although some potential virulence factors have been identified, very little is known about the genes involved in the pathogenesis of these organisms (Houf and Stephan, 2007; Shah *et al.*, 2011; Snelling *et al.*, 2006). With regard to potential infection routes, these mainly include drinking of contaminated water (particularly in developing countries with inadequate water supplies), and consumption and handling of contaminated food (primarily raw or undercooked meat) (Ho *et al.*, 2006; Taylor *et al.*, 1991; Wesley, 1996, 1997). Nevertheless, contact with pets and person-to-person transmission have also been suggested as potential risk factors for human infections (Fera *et al.*, 2009; Houf *et al.*, 2008; Vandamme *et al.*, 1992).

Although the lack of a standard isolation method may result in considerable underestimation of their true occurrence in foods, *Arcobacter* spp. have been frequently isolated from products of animal origin with the highest prevalence being reported in poultry meat, followed by pork and beef (Blackburn and McClure, 2009; Cervenka, 2007; Shah *et al.*, 2011). The prevalence of the organisms in chicken can be as high as 100%, whereas the detection rates in beef, pork, mutton, and milk have been shown to range from approximately 1% to 50% (Cervenka, 2007; Shah *et al.*, 2011). However, arcobacters have not been found in eggs, and only rarely their incidence has been reported in seafood such as clams and mussels (Cervenka, 2007; Collado *et al.*, 2009). It was the isolation of arcobacters (and particularly *Ar. butzleri*, *Ar. cryaerophilus*, and *Ar. skirrowii*) over the last decade from various foods of animal origin that resulted in their classification as emerging foodborne pathogens by the International Commission on Microbiological Specifications for Foods (ICMSF, 2002), despite their very poor association with human foodborne diseases. Although an outbreak of recurrent abdominal cramps in a nursery and primary school in Italy in 1983 was associated with *Ar. butzleri*, with the successive timing of the cases suggesting a person-to-person transmission, no specific food vehicle was identified (Vandamme *et al.*, 1992). Only recently were arcobacters associated with an outbreak of foodborne illness; *Ar. butzleri* was identified as the most likely cause of a foodborne outbreak among attendees of a wedding reception in Wisconsin, US, with the collected epidemiological data demonstrating that rigorous investigation of outbreaks of undetermined etiology is valuable for enhancing one's understanding of emerging agents of foodborne diseases (Lappi *et al.*, 2013).

Arcobacters can easily be inactivated by heating food to an internal temperature of 70 °C as well as by chlorination (Shah *et al.*, 2011). Hence, the risk of transmission to humans of *Arcobacter* spp. via properly cooked foods and chlorinated water should be regarded as negligible (Wesley, 1996, 1997). With particular reference to poultry products, given that arcobacters are probably not normal inhabitants of the poultry intestine (Houf and Van Driessche, 2007; Phillips, 2001), control measures should be focused on preventing contamination of, and proliferation in, the broiler environment as well as at the postslaughter level (Blackburn and McClure, 2009). Although *Ar. butzleri* is believed to be more resistant to irradiation than *Ca. jejuni* (Phillips, 2001; Shah *et al.*, 2011; Wesley, 1997), irradiation with approximately 0.3 kGy for 10 s is sufficient for their inactivation (Shah *et al.*, 2011); thus, irradiation doses currently allowed for pork in the US (i.e., 0.3–1.0 kGy) should provide an effective means of reducing, if not completely eliminating, this organism from pork (Phillips, 2001). Furthermore, organic acid solutions, including those used widely in meat decontamination (e.g., acetic and citric acids at concentrations higher than 0.2%), have demonstrated considerable effectiveness and, therefore, application potential for controlling arcobacters on meat and poultry surfaces (Cervenka, 2007; Shah *et al.*, 2011; Skrivanova *et al.*, 2011; Snelling *et al.*, 2006). Being organisms of recent interest, there are no standard widely accepted methodologies for the detection, isolation, and typing of *Arcobacter* spp. (Blackburn and McClure, 2009). Although several methods using both aerobic and microaerophilic conditions and based on media for *Campylobacter* have been proposed, the currently available methods need to be improved in terms of specificity and sensitivity, with the development and application of molecular techniques gaining increasing interest (Blackburn and McClure, 2009; Douidah *et al.*, 2010; Shah *et al.*, 2011). Robust and reliable molecular typing methods along with basic research on the virulence characteristics of arcobacters are expected to contribute significantly to the accurate identification of this emerging foodborne pathogen as well as to a better understanding of its epidemiology and distribution both in the environment and in foods (Douidah *et al.*, 2010; Shah *et al.*, 2011; Snelling *et al.*, 2006). *Arcobacter* spp. exhibit susceptibility to aminoglycosides (e.g., kanamycin and streptomycin) and thus, the latter antibiotics may be regarded as suitable for the treatment of *Arcobacter* infections when adequate control of their use in veterinary and human medicine is in place (Snelling *et al.*, 2006). Nevertheless, due to the fact that there is evidence of acquired resistance of *Arcobacter* spp. to antimicrobials generally prescribed as first-line drugs for the treatment of campylobacteriosis (e.g., erythromycin, tetracycline, chloramphenicol, and ciprofloxacin), future research trends include studies on the number and diversity of antibiotic-resistant *Arcobacter* strains, as well as assessment of the transfer potential of antibiotic resistance genes among

Arcobacter spp. and between *Arcobacter* and *Campylobacter* (Blackburn and McClure, 2009; Snelling *et al.*, 2006).

Clostridium difficile

Clostridium difficile is a Gram-positive, spore forming, and anaerobic bacillus that has been relatively recently identified as a human pathogen (Dawson *et al.*, 2009; Gibbs, 2009). The first confirmed case of *Cl. difficile* infection (CDI) was reported in 1977 (Larson *et al.*, 1978), when the use of clindamycin was introduced and resulted in a rapid increase in the number of pseudomembranous colitis cases (Dawson *et al.*, 2009). *Cl. difficile* grows optimally at 35–40 °C, ferments amino acids in order to create adenosine triphosphate as an energy source, and can also utilize sugars (Gibbs, 2009). Extensive research on the genome of this bacterium has been carried out aiming at elucidating its mechanisms of infection and pathogenicity. Pathogenic strains of the organism produce two distinct toxins, both of which are high-molecular weight proteins capable of binding to specific receptors on the intestinal mucosal cells: (1) toxin A, an enterotoxin and (2) toxin B, a cytotoxin (Gibbs, 2009). Colonization of the gut by *Cl. difficile* and toxin production results in an acute inflammatory response and severe damage to the intestinal epithelium, particularly following treatment with broad-spectrum antibiotics (Dawson *et al.*, 2009; Rupnik, 2007). The organism, which may be naturally present in the gastrointestinal tract of healthy adults and infants, is usually kept under control by the normal intestinal microflora (Gibbs, 2009; Warren and Guerrant, 2011). When, however, certain antibiotics disrupt the protective gut microflora, indigenous or ingested spores of *Cl. difficile* germinate, multiply rapidly, colonize the gastrointestinal tract, and produce toxins (Dawson *et al.*, 2009; Gibbs, 2009; Warren and Guerrant, 2011). Although any broad-spectrum antibiotic can be associated with CDI, the latter has been primarily linked to clindamycin, cephalosporins, penicillins, and fluoroquinolones (Warren and Guerrant, 2011).

Since its initial recognition, the incidence and severity of CDI has considerably increased and *Cl. difficile* currently constitutes one of the most frequent causative agents of nosocomial diarrhea worldwide (Dawson *et al.*, 2009; O'Donoghue and Kyne, 2010; Rupnik, 2007). Symptoms of CDI may vary from mild diarrhea to life-threatening pseudomembranous colitis, and in addition to patients on antimicrobial treatment, the population at risk for the infection includes patients on other therapies that may also alter the balance of the gut microbiota (e.g., antacid/proton pump inhibitors and nonsteroidal antiinflammatory drugs), as well as the immunocompromized and the elderly (Dawson *et al.*, 2009). As a result of the worldwide increase in the incidence of CDI in the past decade, several molecular typing approaches have been developed in order to enhance the understanding of the epidemiology of *Cl. difficile*, including pulsed-field gel electrophoresis, restriction endonuclease analysis, toxinotyping (i.e., using sequencing data of toxins A and B), multilocus sequence typing, and polymerase chain reaction (PCR)-ribotyping (Dawson *et al.*, 2009). Recent changes in the epidemiology of *Cl. difficile* that have contributed to its characterization as a 'continually evolving pathogen' include (1) the emergence of new groups of highly virulent strains (e.g., strains with PCR-ribotype 027) causing outbreaks of increased disease severity, high relapse rate, and significant mortality in North America, Japan, and Europe (Dawson *et al.*, 2009; Gould and Limbago, 2010; Kuijper *et al.*, 2007; O'Donoghue and Kyne, 2010) and (2) the onset of community-acquired cases involving low-risk population groups (i.e., young individuals, not subjected to antibiotic therapy or previous hospitalization) (Gould and Limbago, 2010; Rupnik, 2007).

The increasing rate of community-associated cases of CDI has raised questions with regard to the routes of transmission of *Cl. difficile* to humans, with foodborne acquisition through consumption or handling of contaminated food products being hypothesized as a possible source of such infections (Gould and Limbago, 2010). In addition to constituting an important pathogenic organism for humans, *Cl. difficile* has also been recognized as an emerging animal pathogen (Rupnik, 2007; Songer and Anderson, 2006), and although a definitive link between the organism's carriage by animals and human disease has not been established, it has been suggested that food animals are likely to play an important role in the transmission of this pathogen to humans through food (Gould and Limbago, 2010). Indeed, there are several reports suggesting that food animals (both healthy and symptomatic) can be reservoirs for *Cl. difficile* (Dawson *et al.*, 2009; Simango and Mwakurudza, 2008; Thitaram *et al.*, 2011), whereas a marked overlap between isolates from animals and humans (including highly virulent outbreak subtypes) has also been documented (Keel *et al.*, 2007; Rupnik, 2007; Zidaric *et al.*, 2008). The organism can also be recovered from a wide variety of environmental sources including soil, seawater, and freshwater (Gould and Limbago, 2010). Hence, food products may become contaminated with *Cl. difficile* via multiple routes. With particular reference to meat and meat products, the organism could be either introduced during processing or be initially present in the muscle tissue (Rupnik, 2007). The presence of *Cl. difficile* spores in the feces of swine or beef cattle, for instance, may result in contamination of pork and beef products during slaughter (Thitaram *et al.*, 2011). In addition to meat and meat products, *Cl. difficile* has been isolated from a diverse set of foods such as chicken, produce, fish, and seafood (**Table 1**).

Given that broad-spectrum antibiotics exacerbate CDI, treatment of the disease is complicated with the administration of very few antibiotics, such as metronidazole and vancomycin, appearing to be effective (Kuijper *et al.*, 2007). As antibiotic resistance constitutes one of the most important virulence factors for *Cl. difficile*, attempts to prevent infections should focus on controlling the overall use of antibiotics, and particularly high-risk antibiotics such as cephalosporins, clindamycin, and fluoroquinolones (Dawson *et al.*, 2009; Kuijper *et al.*, 2007). However, it has been suggested that with the development of more potent antibiotics for other resistant bacterial pathogens, the problem of CDI is expected to continue (Warren and Guerrant, 2011). Therefore, despite the fact that new treatment options (both antibiotic and nonantibiotic alternatives) are becoming available (Kuijper *et al.*, 2007; O'Donoghue and Kyne, 2010), the antibiotic susceptibilities of *Cl. difficile* isolates need to be assessed and known; although such knowledge might not be relevant to the infection's treatment per se, it is expected to

facilitate the identification of the predisposing and prevailing antibiotic pressure to which this pathogen is subjected (Warren and Guerrant, 2011). In addition to classical virulence determinants such as toxin production and antibiotic resistance, the evaluation of other factors (e.g., increased gut colonization, increased resistance to bile salts, and increased motility/chemotaxis) should be very useful in explaining the emergence of epidemic *Cl. difficile* strains (Dawson *et al.*, 2009). Moreover, given that *Cl. difficile* spores can survive on surfaces for long periods of time and are resistant to many disinfectants, research on spore germination would provide useful information for controlling the spreading and persistence of this organism (Dawson *et al.*, 2009). With particular reference to community-associated cases of CDI, in order to understand the dynamics of and risk factors for the development of human disease, including the true occurrence and importance of foodborne transmission, more research covering the following areas is required (Gould and Limbago, 2010; Rodriguez-Palacios, *et al.*, 2010; Thitaram *et al.*, 2011): (1) determination of the infectious dose of *Cl. difficile*, which is currently unknown, and comparison of it with the microbial load typically present on contaminated foods at the time of consumption; (2) surveillance for human and animal infections utilizing standard subtyping systems capable of discerning common sources of these infections; (3) detailed strain typing and epidemiological investigations aiming at establishing the relationship between food animals and human isolates and, thus, determining the true potential for acquisition of foodborne disease; (4) development of consensus best practice methods for food testing; and (5) improving our understanding of the effects of heating (and assessment of the need for revising current cooking recommendations) and surface decontamination on *Cl. difficile* spores.

Cronobacter spp.

Cronobacter spp. are Gram-negative, facultatively anaerobic, and motile with peritrichous flagella rods, which are members of the family Enterobacteriaceae (Iversen *et al.*, 2008). The genus *Cronobacter*, formerly known as *Enterobacter sakazakii* (Farmer *et al.*, 1980), consists of five species, plus a possible sixth species: *Cronobacter sakazakii*, *Cronobacter malonaticus*, *Cronobacter turicensis*, *Cronobacter muytjensii*, *Cronobacter dublinensis*, and Genomospecies 1 (Forsythe and Varnam, 2009; Iversen *et al.*, 2008). *Cronobacter* species differentiation is primarily based on DNA sequence analysis, supported by biochemical differentiation (Forsythe and Varnam, 2009). *Cronobacter* spp. are considered emerging opportunistic pathogens and the etiological agents of life-threatening infections among infants (Bowen and Braden, 2006; CDC, 2009; Drudy *et al.*, 2006), with the first reported outbreak referring to neonatal meningitis in England in 1958 that resulted in the deaths of two infants (Urmenyi and Franklin, 1961). Few virulence factors have been identified so far (Pagotto *et al.*, 2003; Townsend *et al.*, 2008), though there is evidence of considerable virulence variability among different subtypes of these organisms (Townsend *et al.*, 2008). Owing to their isolation from neonatal infections, the species *Cr. sakazakii*, *Cr. malonaticus*, and *Cr. turicensis* are of particular interest (Forsythe and Varnam, 2009). Despite its low incidence, *Cronobacter* infection, whose main clinical manifestations include meningitis, septicemia, and necrotizing enterocolitis, is associated with significant morbidity (i.e., irreversible neurological sequelae resulting in quadriplegia, developmental impedance, and impaired sight and hearing) and with mortality rates as high as 80% (Drudy *et al.*, 2006). Powdered infant formula (PIF) products have been epidemiologically linked to several cases of *Cronobacter* infection (Bowen and Braden, 2006; CDC, 2009; Himelright *et al.*, 2002; Van Acker *et al.*, 2001), and premature/low-birth-weight infants and those aged less than 28 days are more at risk than older infants due to their underdeveloped immune status and lack of competing intestinal flora (Drudy *et al.*, 2006; Forsythe and Varnam, 2009). Infants of more than 3 months appear to be at considerably less risk for fatal infections (O'Brien *et al.*, 2009), though the few available reports of *Cronobacter* infections in adults usually refer to individuals with underlying diseases (e.g., malignancies) (Drudy *et al.*, 2006). *Cronobacter* spp. are naturally resistant to all macrolides, lincomycin, clindamycin, streptogramins, rifampicin, fusidic acid, and fosfomycin, and the infections associated with these organisms have been traditionally treated with a combination of ampicillin with gentamicin or chloramphenicol (Drudy *et al.*, 2006).

Specific natural reservoirs of *Cronobacter* spp. have not been established yet, with the distribution of these organisms appearing to be ubiquitous. On the basis of their presence in dry herbs and spices, it has been hypothesized that the natural habitat of *Cronobacter* spp. may be plant materials (Chon *et al.*, 2012; Drudy *et al.*, 2006; Hochel *et al.*, 2012; Iversen and Forsythe, 2004). Nonetheless, organisms of the genus *Cronobacter* have also been isolated from animal sources, a wide range of clinical sources (e.g., cerebrospinal fluid, blood, bone marrow, urine, intestinal and respiratory tracts, wounds, and feces), hospital settings, food-processing, and household environments, as well as from multiple food sources (Drudy *et al.*, 2006). *Cronobacter* spp., and primarily *Cr. sakazakii*, have been detected in various food products mainly of plant origin and dried food ingredients (**Table 1**). However, strong association has been observed only with PIF in which the pathogen can be introduced either intrinsically (i.e., at some stage during the manufacturing process) or extrinsically (i.e., during preparation of PIF at hospital neonatal units or at home) (Drudy *et al.*, 2006). As supported by molecular typing data, production facilities may serve as points of continuous entry and dissemination of *Cronobacter* spp. into milk powder products (Lehner *et al.*, 2010). Indeed, studies assessing the occurrence and distribution of the pathogen within milk powder-processing plants have revealed a number of potential reservoirs as well as of practices/events that may compromise the safety of the final product. Potential sources of *Cronobacter* spp. in manufacturing environments are the supply air, spray-drying towers, roller dryers, textile filters for exhaust air, vacuum cleaners, and the filling line of the processing units (Hein *et al.*, 2009; Jacobs *et al.*, 2011; Reich *et al.*, 2010). Long-term persistence of certain *Cronobacter* subtypes in milk powder processing units has been observed in some cases (Craven *et al.*, 2010; Hein *et al.*, 2009). On the basis of the findings of Craven *et al.* (2010), the most prevalent and persistent *Cronobacter* clones were isolated from external roofs above spray dryers, in air treatment areas and where high foot traffic occurs. Practices/events that may result in

contamination of the final milk powder include reintroduction of filtered powder into the product flow, passing of contaminated milk concentrated through the process unheated, and failure of established hygiene measures (e.g., cleaning-in-place events and heat treatments) to completely eliminate *Cronobacter* spp. from all areas of the processing line (Hein *et al.*, 2009; Jacobs *et al.*, 2011). In addition to food-processing facilities, *Cronobacter* spp. have been isolated from various sites in domestic environments (Kilonzo-Nthenge *et al.*, 2012; Molloy *et al.*, 2009). Hence, given this and that the organisms can also be present in the feces or on skin of healthy individuals (Kandhai *et al.*, 2010), in the absence of good hygiene and food handling practices, PIF contamination may also occur during preparation at home.

To reduce the risk of PIF contamination with *Cronobacter* spp., control measures should be in place throughout the food chain. Manufacturers should implement strategies aiming at controlling the initial populations of these organisms and reducing the risks of PIF contamination both during production and at the postprocessing level. PIF products should be formulated in accordance with the Codex Alimentarius Commission Standards (CCFH, 2008), although manufacturers are being encouraged to develop a greater range of commercially sterile alternative formula products specifically targeting high-risk groups (i.e., premature/low-birth-weight infants) (Drudy *et al.*, 2006). With regard to the development and application of control interventions at the manufacturing level, research data suggest that gamma irradiation may be effective against *Cronobacter* spp. in PIF (Osaili *et al.*, 2007), whereas organic acids such as propionic acid and acetic acid may possibly be used as preservatives to inhibit the survival and growth of these organisms in liquid foods (Back *et al.*, 2009). In addition to PIF manufacturers, caregivers in hospital neonatal units as well as food handlers at home are also responsible for the safety of this product, and should be continuously alerted that PIF is not a sterile product, and that, therefore, the use of hygienic measures during preparation is essential (Drudy *et al.*, 2006). Furthermore, given that infant formula can support prolific bacterial growth, appropriate temperature control of reconstituted product is of vital importance for its safety (Forsythe and Varnam, 2009). The World Health Organization (WHO) and the United Nations Children's Fund recommend that, where possible, infants should be exclusively breastfed for the first 6 months of life (WHO/UNICEF, 2003). Furthermore, the WHO in collaboration with the Food and Agriculture Organization (FAO) of the United Nations have issued recommendations regarding the safe preparation, storage, and handling of PIF in both care settings and the home. According to these recommendations, PIF should be reconstituted at 70 °C and either used within 2 h after preparation or stored in the refrigerator ($\leq$5 °C) for up to 24 h; feed that has not been consumed within 2 h should be discarded (FAO/WHO, 2007). Finally, as certain procedural and environmental factors within neonatal intensive care units may have an important effect on infant formula contamination (Steele and Short, 2008), increasing the awareness of the potential threats posed by *Cronobacter* spp. among medical personnel and caregivers via continuous education is of vital importance for protecting high-risk infants (Drudy *et al.*, 2006). With reference to future trends, given that since its initial recognition the incidence of *Cronobacter* in PIF on the market has appeared to decrease, though improvement of hygiene measures has been associated with reduction of reported outbreaks, it has been opined that of particular interest are also powdered nutrition formulas frequently consumed as dietary supplements by the elderly and other immunocompromized individuals, population groups which are also susceptible to *Cronobacter* infections (Forsythe and Varnam, 2009).

Escherichia coli

There are a number of different enteropathogenic groups of *E. coli* that have been shown to cause various types of gastrointestinal infections, with enterohemorrhagic *E. coli* (EHEC) being recognized as an etiological agent of serious illness and mortality in outbreaks of foodborne illness involving a large variety of foods and proceeding to hemolytic uremic syndrome (HUS) (Viazis and Diez-Gonzalez, 2011). A common characteristic of all EHEC strains is their ability to produce shiga toxins, and as such, they are commonly referred to as shiga toxin-producing *E. coli* (STEC) (Viazis and Diez-Gonzalez, 2011). STEC cause sporadic or epidemic foodborne or waterborne illness, whose clinical spectrum involves diarrhea, hemorrhagic colitis, and the potentially fatal HUS (Karmali, 2005). The most common serotype implicated worldwide as the major cause of hemorrhagic colitis and HUS is *E. coli* O157:H7, whose detection and diagnosis is based on its inability to ferment the carbohydrate sorbitol (Bielaszewska and Karch, 2000; Karmali, 2005; Viazis and Diez-Gonzalez, 2011). Since the initial association of *E. coli* O157:H7 with epidemic foodborne disease in 1982, and the consequent definition of a new foodborne zoonosis (Riley *et al.*, 1983), more than 200 different O serogroups of *E. coli* have been shown to produce shiga toxins, and more than 100 of these STEC have been linked to human disease (Johnson *et al.*, 2006). STEC strains capable of fermenting sorbitol have also been isolated from patients and associated with an increasing number of outbreaks and sporadic cases of diarrhea and HUS (Bielaszewska and Karch, 2000). Although their linkage to human disease is not as well understood as that of *E. coli* O157:H7 and their true occurrence is most likely underestimated, the rising public health significance of sorbitol-fermenting STEC has been acknowledged (Bielaszewska and Karch, 2000; Gerber *et al.*, 2002).

Non-O157 shiga toxin-producing Escherichia coli

Non-O157 STEC are a heterogeneous group of organisms consisting of more than 100 serogroups (Bielaszewska and Karch, 2000). The clinical diagnosis of non-O157 STEC can be very challenging because, similar to *E. coli* O157:H7, they are capable of inducing a range of illnesses, including diarrhea, hemorrhagic colitis, and HUS, either as sporadic cases or in the form of outbreaks (Johnson *et al.*, 2006). However, despite the similarities of their clinical manifestations, genomic studies suggest that O157 and non-O157 STEC have different evolutionary histories (Smith and Fratamico, 2012). Although cattle are regarded as the major reservoir for clinically significant non-O157 STEC, other animals such as sheep, goats, deer, and swine may also be carriers of these organisms (Monaghan *et al.*, 2012; Smith and Fratamico, 2012). Outbreaks caused by

this group of STEC have been associated with ingestion of contaminated food and water, as well as with person-to-person contact (Kaspar *et al.*, 2010; Smith and Fratamico, 2012). Non-O157 STEC strains have been linked to major foodborne outbreaks in the USA, Europe, Australia, Japan, and other countries, being often responsible for a considerable portion of the total reported STEC foodborne infections (Kaspar *et al.*, 2010; Scallan *et al.*, 2011; Smith and Fratamico, 2012). Foods that have been associated with illness caused by these organisms include raw and pasteurized cow's milk, ice cream, cheeses, fermented sausages, ground beef, cider, and vegetables (Mathusa *et al.*, 2010; Smith and Fratamico, 2012).

Serogroups that have emerged as significant etiological agents of human disease (both diarrhea and HUS) in several parts of the world include O26, O45, O103, O111, O121, and O145 (Bielaszewska and Karch, 2000; Bielaszewska *et al.*, 2007; Brooks *et al.*, 2005; Brown *et al.*, 2012; CDC, 2013; Gerber *et al.*, 2002; Patton *et al.*, 1996; Vally *et al.*, 2012). Indeed, approximately three-fourths of the disease-inducing non-O157 STEC isolates reported to the Centers for Disease Control and Prevention of the United States Department of Health and Human Services belonged to the above-mentioned six O serogroups (Brooks *et al.*, 2005). Hence, as a result of their increasing public health impact, these six serogroups were declared by the United States Department of Agriculture's Food Safety and Inspection Service as adulterants if present in raw nonintact beef products (USDA-FSIS, 2011). Molecular subtyping data indicate that strains of serogroups O26, O103, and O111 belong to their own clonal lineage and exhibit unique virulence profiles (Bielaszewska and Karch, 2000). Additional non-O157 STEC serogroups that have been reported to emerge in Europe are O100 and O127 (Orth *et al.*, 2006). It has been suggested that certain non-O157 STEC strains (e.g., strains producing shiga toxin 2 (Stx2)) may be more likely to precipitate HUS than others (e.g., strains producing shiga toxin 1 (Stx1) alone) (Brooks *et al.*, 2005; Johnson *et al.*, 2006). Nevertheless, due to the generally limited information that is currently available with regard to the virulence and stress responses of non-O157 STEC, it is difficult to draw solid conclusions on their pathogenicity or their behavior when exposed to stress in the environment, in food, and during food processing (Smith and Fratamico, 2012). It has been opined that the incidence, distribution, and pathological spectrum of these emerging agents is expected to be elucidated only through improved surveillance, with the latter requiring a number of individual conditions to be met including increased clinical suspicion, improved laboratory isolation through the development and use of rapid, sensitive, accurate, and inexpensive techniques, as well as continued serotyping of isolates in public health laboratories (Brooks *et al.*, 2005; Johnson *et al.*, 2006).

Sorbitol-fermenting shiga toxin-producing Escherichia coli *O157:H*$^-$

Sorbitol-fermenting STEC O157:H$^-$ (H$^-$ indicates nonmotility) strains have been identified as agents of severe human disease, such as HUS, with the organisms being isolated throughout Europe including the Czech Republic, Hungary, Finland, and the UK, as well as in Australia (Orth *et al.*, 2009). The first isolation of these organisms was reported during a HUS outbreak investigation in Bavaria, Germany, in 1988; nonmotile *E. coli* strains, harboring the *stx2* gene, and fermenting sorbitol within 24 h of incubation were isolated from the stools of two out of six affected children (Karch *et al.*, 1990). This group of organisms appears to represent a new clone within the *E. coli* O157 serogroup, having its own typical combination of virulence factors (Bielaszewska and Karch, 2000). Indeed, it has been observed that illness associated with sorbitol-fermenting STEC O157:H$^-$ is rarely confined to diarrhea and usually progresses to life-threatening HUS, and that patients infected with these organisms tend to develop HUS more frequently than patients infected with other EHEC strains, indicating a potential hypervirulence of this group of STEC (Nielsen *et al.*, 2011; Orth *et al.*, 2009). Although some epidemiological data indicate potential existence of differential reservoirs of and vehicles of infections caused by STEC O157:H$^-$ in comparison to STEC O157:H7 (Karch and Bielaszewska, 2001), the reservoir and transmission routes of the former organisms are still largely unknown (Orth *et al.*, 2009). Evidence that bovine animals may constitute a reservoir of sorbitol-fermenting STEC O157:H$^-$ and, thus, a source of human disease, has been provided in some cases (Bielaszewska *et al.*, 2000; Orth *et al.*, 2006). Moreover, it has been hypothesized that these pathogens might be adapted to the human intestine and that humans may constitute their primary reservoir; however, the role of person-to-person transmission, which is assumed to be the major route of spreading of STEC O157:H$^-$ infections, still needs to be established (Karch and Bielaszewska, 2001). To properly assess the epidemiological significance of STEC O157:H$^-$ and to better understand its epidemiology, microbiological detection of this pathogen and HUS surveillance need to be improved (Karch and Bielaszewska, 2001; Nielsen *et al.*, 2011). Given that detection of all HUS-causing strains, including the sorbitol-fermenting STEC O157:H$^-$, cannot be assured on the basis of phenotypic characteristics, screening for shiga toxins (e.g., by enzyme-linked immunosorbent assay (ELISA), or shiga toxin genes (e.g., by PCR) is strongly recommended (Orth *et al.*, 2009).

Sorbitol-fermenting shiga toxin-negative Escherichia coli *O157:H*$^-$

Serogroup O157:H$^-$ strains capable of fermenting sorbitol, but not producing shiga toxins, have also been identified as agents of diarrhea and HUS in different countries such as Austria, Germany, and India (Allerberger *et al.*, 2000; Chakraborty *et al.*, 2003; Schmidt *et al.*, 1999). Although their origin, pathogenic mechanisms, and role in human disease warrant clarification, the following hypotheses have been postulated regarding sorbitol-fermenting Stx-negative *E. coli* O157:H$^-$ (Karch and Bielaszewska, 2001): (1) they might have emerged from sorbitol-fermenting STEC O157:H$^-$ organisms by losing their *stx* genes during infection, isolation, or subculture; (2) they might be progenitors of sorbitol-fermenting STEC O157:H$^-$, with the latter arising by transduction with *stx*-converting bacteriophages; and (3) they might be inherently Stx-negative and cause the underlying diseases through the potential possession of additional, as yet unidentified, virulence factor(s).

Mycobacterium paratuberculosis

Mycobacterium avium subspecies *paratuberculosis* (MAP) was first described by Johne and Frothingham in 1895 in the context of an investigation of the cause of chronic diarrhea in cattle, and it has been classified as a member of the family Mycobacteriaceae with the latter consisting of Gram-positive, strictly aerobic, nonmotile, and acid-fast rod-shaped bacteria with fastidious growth requirements (Griffiths, 2009; Legrand *et al.*, 2000). MAP is the etiological agent of paratuberculosis, a chronic granulomatous enteritis in ruminants, also known as Johne's disease, which results in diarrhea, weight loss, and ultimately death (Griffiths, 2009; Skovgaard, 2007). Johne's disease is widespread in dairy cattle and its prevalence is increasing in food-producing animals globally, causing significant financial losses (Greenstein and Collins, 2004; Skovgaard, 2007). Owing to the remarkable clinical, epidemiological, and pathological similarity of Johne's disease to Crohn's disease in humans, a chronic inflammatory disease most commonly affecting the terminal ileum, MAP has also been proposed as the etiological agent of Crohn's disease (Greenstein and Collins, 2004; Griffiths, 2009; Skovgaard, 2007). It has been suggested that multiple interacting factors (genetic predisposition, infectious agents like MAP, alteration of intestinal microflora, and immune-mediated tissue damage) are involved in the development of this disease, with the relative importance of each one of them not being determined (Griffiths, 2009; Pistone *et al.*, 2012). Despite the fact that since the first proposal of a possible link of MAP with Crohn's disease (Chiodini *et al.*, 1984) a considerable amount of data indicating such association has been generated (Feller *et al.*, 2007), a definitive causal relationship has yet to be established (Pistone *et al.*, 2012). Nevertheless, the precautionary principle approach has been advocated until the role of the organism has been definitively determined (Griffiths, 2009), and potential vehicles of transmission of the organism from animals to humans include milk and dairy products as well as raw meat contaminated via feces during slaughtering (Kim and Griffiths, 2011; Skovgaard, 2007). As MAP has been isolated from both raw and pasteurized milk, the efficacy of routine pasteurization against the organism has been questioned (Greenstein and Collins, 2004; Griffiths, 2009; Skovgaard, 2007). However, based on the findings of certain studies, among the numerous studies assessing the ability of MAP to survive pasteurization, it has been concluded that the organism is unable to survive high-temperature short-time (HTST) pasteurization (Griffiths, 2009). Other issues of concern with regard to food safety that warrant further investigation are the ability of MAP to survive the low-temperature thermization processes used in manufacturing of many cheeses, as well as its prevalence and evolution during cheese ripening (Skovgaard, 2007). Additional possible means of transmission of MAP to humans include contaminated water and animal contact (Greenstein and Collins, 2004). The rising concerns regarding the putative zoonotic and foodborne transmission potential of MAP, in conjunction with the difficulties associated with the culture of this organism, render the development of rapid and sensitive methods for its detection and characterization essential (Griffiths, 2009; Kim and Griffiths, 2011; Skovgaard, 2007). Information regarding the relationship between MAP and Crohn's disease, the prevalence and survival of this emerging pathogen in foods and in the environment, as well as potential control approaches has been reviewed in detail by Griffiths (2009).

Salmonella enterica

Salmonellosis, the infection caused by the bacterium *Sa. enterica*, is an illness known for more than 100 years (Bailey *et al.*, 2010). Among the so-called 'host-restricted' *Sa. enterica* serotypes, the ones that are associated with animal hosts (e.g., Gallinarum and Abortusovis) usually elicit very mild symptomatology in humans, whereas, the 'human-restricted' *Sa. enterica* serotypes Typhi, Paratyphi A, and Paratyphi B (which are not usually pathogenic to animals) commonly cause severe systemic disease such as typhoid or enteric fever in humans (Velge *et al.*, 2005). Widespread *Sa. enterica* serotypes, such as Enteritidis and Typhimurium, generally cause gastrointestinal infection to humans, known as nontyphoidal salmonellosis which, however, may also be associated with serious clinical outcomes (e.g., bacteraemia, endovascular infections, and focal infections), particularly in susceptible individuals (Hohmann, 2001; Velge *et al.*, 2005). In addition to host-related factors (e.g., health status, immunosuppresion, age, and genetic defects), the exact clinical outcome of nontyphoidal salmonellosis depends on the virulence traits of the *Sa. enterica* strains responsible for the infection, as certain serotypes of the pathogen are more likely than others to cause systemic infections not only in susceptible hosts, but also in people with no identifiable predisposing conditions (Fierer and Guiney, 2001).

Nontyphoidal *Salmonella* has been a leading cause of foodborne illness in many countries (Adak *et al.*, 2005; Scallan *et al.*, 2011). The emergence of human foodborne infections caused by *Salmonella* Enteritidis and by multiple-antibiotic-resistant strains of *Salmonella* Typhimurium constituted two major changes in the epidemiology of nontyphoidal salmonellosis in the European Union and the USA in the second half of the twentieth century (Velge *et al.*, 2005). Although *Sa. enterica* serotypes Enteritidis and Typhimurium are the serotypes most commonly associated with human infections (CDC, 2011a; EFSA-ECDC, 2012), the emergence and potential connection to human disease of rare serotypes of the organism during the last decades has attracted the attention of the scientific community. For instance, the prevalence, among human clinical cases, of *Sa. enterica* serotype 4,5,12:i:, a serotype antigenically similar and genetically closely related to *Salmonella* Typhimurium, has increased considerably in many countries in the last decade (Soyer *et al.*, 2009). This emerging *Sa. enterica* serotype, which represents multiple distinct clones, has been responsible for a number of human salmonellosis outbreaks (e.g., in Spain, Luxemburg, and the USA) and has been isolated from different foods and animals over the last decades (Soyer *et al.*, 2009). *Salmonella enterica* serotype Cerro has been identified as a potentially emerging pathogen of cattle; given the fact that other *Sa. enterica* serotypes important to bovine health have emerged to become leading causes of human foodborne disease, close monitoring of *Salmonella* Cerro is warranted (Cummings *et al.*, 2010). According to the

findings of a study assessing the association of *Sa. enterica* with foodborne and waterborne diseases in Korea during 1998–2007, although the three most prevalent serotypes were Typhi, Enteritidis, and Typhimurium, there were also remarkable outbreaks caused by rare serotypes such as Othmarschen, London, and Paratyphi A (Kim, 2010). *Salmonella enterica* serotype Napoli is another emerging serotype in Italy, France, and Switzerland; characterization of strains of this serotype isolated in Italy from human cases, foods of animal origin, and the environment showed an array of virulence genes similar to those of other serotypes of public health significance, demonstrating its ability to cause infection in humans (Graziani *et al.*, 2011). Lastly, *Sa. enterica* serotype Weltevreden, which has long been associated with meat products in Southeast Asia, is an emerging serotype associated with meat and particularly with plant products in Western countries (Brankatschk *et al.*, 2012; Emberland *et al.*, 2007). Nonetheless, to enhance one's understanding of the ecology and risk factors for human infection of the aforementioned emerging serotypes, further studies are required.

Streptococcus suis

Streptococcus suis is an encapsulated Gram-positive, facultatively anaerobic coccus which is emerging as an important threat to human health (Segura, 2009; Wertheim *et al.*, 2009). The organism's main reservoir is swine, with its natural habitat being the upper respiratory, genital, and alimentary tracts of pigs (Ma *et al.*, 2008; Segura, 2009). *Streptococcus suis* was first described by veterinarians in 1954 as the etiological agent of outbreaks of meningitis, septicemia, and purulent arthritis among piglets (Field *et al.*, 1954). The first human cases of *St. suis* infection were reported in 1968 in Denmark (Perch *et al.*, 1968), and since then numerous cases have been reported worldwide including the UK, France, Germany, The Netherlands, Sweden, New Zealand, Thailand, Singapore, Taiwan, and Hong Kong (Ma *et al.*, 2008). On the basis of composition of the polysaccharide capsule, 35 serotypes of the organism have been identified, with serotype 2, however, being associated with the majority of cases of human infections (Lun *et al.*, 2007; Wertheim *et al.*, 2009; Segura, 2009). Despite the fact that most reports refer to sporadic cases of infection, an outbreak of acute disease in humans in Sichuan Province, China in 2005, involving 215 cases and 38 deaths, highlighted the importance of *St. suis* as an emerging zoonotic pathogen (Yu *et al.*, 2006). The epidemiology of *St. suis* infections in humans remains largely undefined (Segura, 2009). Nonetheless, and in concordance with the pathogen's natural distribution in the environment, human cases are most frequently reported from countries where pig-rearing is common (particularly in Southeast Asia), and the majority of them are associated with cutaneous contact with infected pigs or with handling or consumption of uncooked or undercooked pork (Segura, 2009; Wangsomboonsiri *et al.*, 2008; Wertheim *et al.*, 2009). *Streptococcus suis* causes systemic infection in humans affecting several organ systems, with its most common clinical manifestation being meningitis, while patients are also likely to develop skin problems (e.g., petechiae, purpura, and ecchymoses) (Wertheim *et al.*, 2009). Less common manifestations of the infection include endocarditis, acute pyogenic arthritis, endophthalmitis and uveitis, peritonitis, rhabdomyolysis, and spondylodiscitis, whereas a striking feature that may be reported by up to one-half of patients is subjective hearing loss (Segura, 2009; Wertheim, *et al.*, 2009). Simple control measures, embraced by both workers with occupational exposures and the general public, should be adequate to prevent the majority of cases of *St. suis* infection. Such measures include cautious handling of pigs or raw pork (e.g., wearing gloves during swine slaughtering or processing of pork meat, hand washing after handling of raw pork meat, and avoiding cross-contamination between raw and cooked pork) and thorough cooking of pork meat (Ma *et al.*, 2008; Segura, 2009; Wertheim *et al.*, 2009). Future research shedding light on the virulence factors, the selective pressures resulting in virulence enhancement, as well as the geographical localization of emerging highly virulent types of *St. suis*, is expected to improve significantly one's understanding of the complex evolution of this pathogen (Segura, 2009).

Antimicrobial-Resistant Strains

The appearance of antimicrobial-resistant bacteria has been promoted by the extensive use or misuse of antimicrobial agents, not only in the treatment of infected humans and animals but also as growth-enhancing or health-promoting agents in livestock, seafood, and plant production (Helmuth, 2000; Hur *et al.*, 2012). The overuse of antimicrobials in animal husbandry may result in a long-lasting, strong selective pressure on bacteria prevalent in intensive production units, leading to the emergence of antimicrobial-resistant strains in food animals, which are then transmitted to humans either directly or through the food supply (Angulo *et al.*, 2000; Fey *et al.*, 2000; Helmuth, 2000). A growing concern over the past 30 years is the worldwide emergence and increasing prevalence of MDR phenotypes among bacterial strains, and particularly among *Sa. enterica* serotypes, exhibiting resistance to several clinically important antimicrobial agents traditionally used to treat bacterial infections in human and veterinary medicine (Hur *et al.*, 2012). The first reports on resistant *Salmonella* date back to the early 1960s and refer mainly to monoresistant strains of the organism (Helmuth, 2000). Widespread resistance of *Sa. enterica* serotypes Typhi and Paratyphi A to chloramphenicol, ampicillin, and cotrimoxazole was documented at the end of the 1980s and early 1990s with MDR isolates of these serotypes causing significant outbreaks, particularly in Asia (Parry *et al.*, 2002). Nonetheless, the declining resistance levels as observed and reported in the following years suggested that drugs such as chloramphenicol may be used again as first-line therapy for enteric fever (Parry, 2003). Low-level resistance to ciprofloxacin in the aforementioned serotypes has been reported in areas of Central, South and Southeast Asia, frequently resulting in fluoroquinolone treatment failures (Aarestrup *et al.*, 2003; Parry *et al.*, 2002). With reference to nontyphoidal *Salmonella*, MDR strains have been found to be of various serotypes including Agona, Anatum, Choleraesuis, Derby, Dublin, Heidelberg, Kentucky, Newport, Pullorum, Schwarzengrund, Seftenberg, Typhimurium, and Uganda (Hur *et al.*, 2012; Wasyl and Hoszowski, 2012). Antimicrobial

resistance has generally been less of a problem in *Sa. enterica* serotype Enteritidis (Hur *et al.*, 2012; Parry, 2003). An event of major public health significance has been the emergence and clonal spread of certain MDR genotypes, with the most characteristic example being the global epidemic spread of *Sa. enterica* serotype Typhimurium definitive type 104 (DT104) (Butaye *et al.*, 2006). This phage type commonly exhibits the following pentaresistance pattern (i.e., resistance to five antimicrobial agents): ampicillin, chloramphenicol, streptomycin, sulfamethoxazole, and tetracycline (R-type ACSSuT) (Helmuth, 2000; Hur *et al.*, 2012; Parry, 2003). Additional resistance to trimethoprim and low-level resistance to ciprofloxacin have also been occasionally observed among *Salmonella* Typhimurium DT104 isolates (Parry, 2003; Velge *et al.*, 2005).

The ability to produce β-lactamases (i.e., enzymes that hydrolyze β-lactams) constitutes one of the most important resistance mechanisms of Gram-negative rods, and the emerging resistance related to the production of extended-spectrum β-lactamases (ESBLs) has been identified as a significant problem with regard to handling of bacterial infections (Gniadkowski, 2001; Paterson, 2006). ESBLs are mainly found in strains of *E. coli* and *Klebsiella pneumoniae*, but are also reported in other members of the family Enterobacteriaceae as well as in *Pseudomonas aeruginosa*, and infections with ESBL-producing bacterial strains are mainly encountered, sporadically or in outbreaks, in critical care units in hospitals (Shah *et al.*, 2004). ESBLs are typically plasmid, rather than chromosomally mediated enzymes, and the majority of them have evolved from class A β-lactamases, namely TEM-1, TEM-2, and SHV-1, which are frequently expressed in Gram-negative bacteria and confer resistance to ampicillin, amoxicillin, and other penicillins, as well as to early generation cephalosporins (Paterson, 2006; Shah *et al.*, 2004). TEM- or SHV-type ESBLs are typically not active against cephamycins or carbapenems, and can generally be inhibited by β-lactamase inhibitors (e.g., clavulanate, sulbactam, or tazobactam) (Paterson, 2006). Persistent exposure of bacterial strains to a multitude of β-lactams has induced dynamic and continuous production and mutation of β-lactamases of certain strains, expanding their activity even against newly developed β-lactam antibiotics (Shah *et al.*, 2004). For instance, mutations of the genes encoding TEM-1, TEM-2, or SHV-1 gave rise to new β-lactamases, capable of hydrolyzing third-generation cephalosporins and aztreonam (Paterson, 2006). Additional families of ESBLs that may also be expressed by Enterobacteriaceae include the CTX-M-type (cefotaximases) and OXA-type (primarily in *P. aeruginosa*) enzymes as well as novel unrelated β-lactamases (Jin and Ling, 2006; Paterson, 2006; Shah *et al.*, 2004). In addition to ESBLs, other β-lactamases capable of hydrolyzing extended-spectrum cephalosporins are: (1) class C cephalosporinases (AmpC) which, unlike ESBLs, are also active against cephamycins and resistant against β-lactamase inhibitors and (2) carbapenemases that have a broader range of activity and are also active against carbapenems (Gniadkowski, 2001; Paterson, 2006).

There is ample evidence of person-to-person transmission with regard to β-lactamase producers (Paterson, 2006). Furthermore, although β-lactamase-producing bacterial strains are mainly prevalent in hospital settings, there is evidence suggesting that they also are emerging and spreading in the community, with community-acquired cases of infections including both urinary tract and gastrointestinal infections (Paterson, 2006). β-lactamase-producing bacteria that have been associated with drug-resistant gastroenteritis include *Sa. enterica*, *Shigella* spp., *Vibrio cholerae*, and STEC (Paterson, 2006). With reference to *Sa. enterica*, both ESBLs and AmpC β-lactamases have been identified, and the resistance of this pathogen to third-generation cephalosporins is of great concern as ceftriaxone constitutes the antimicrobial of choice for treating invasive salmonellosis caused by MDR strains in children (i.e., where the use of quinolones is not indicated) (Butaye *et al.*, 2006; Paterson, 2006; Velge *et al.*, 2005). The emergence and worldwide increase of ESBL (particularly of the CTX-M group) producers among *Sa. enterica* serotypes, including the serotypes Enteritidis, Typhimurium, and Virchow, has been acknowledged as an important public health issue during the last years (Bonalli *et al.*, 2011; Jin and Ling, 2006; Riano *et al.*, 2009). Although more research is required in order to track the evolution of ESBLs in *Sa. enterica* isolates from different environments, some findings do support the potential for clonal spread of CTX-M-type ESBLs among animals and humans (Riano *et al.*, 2009). Furthermore, *Sa. enterica* serotype Newport isolates resistant to multiple antimicrobials, including extended-spectrum cephalosporins via the production of AmpC, known as serotype Newport MDR-AmpC isolates, have recently emerged. The common recovery of MDR-AmpC isolates of *Salmonella* Newport from food animals and humans suggests their potential transmission to humans through the food chain (Zhao *et al.*, 2003). Indeed, as supported by epidemiological data, infections with such isolates appear to be acquired through the food supply, most likely from bovine and perhaps poultry sources, and particularly among individuals already taking antimicrobial agents (CDC, 2002; Varma *et al.*, 2006). The emergence and worldwide dissemination of multiresistant *E. coli* producing ESBLs, particularly of the CTX-M-type, as an important cause of both nosocomial and community-onset infections have also been acknowledged (Oteo *et al.*, 2010; Pitout *et al.*, 2004). Similar to the case of *Sa. enterica*, the rising trend of antimicrobial resistance and the high prevalence of β-lactamase determinants in *E. coli* strains of animal origin (Li *et al.*, 2010) dictate the development and implementation of effective control interventions.

Surveillance programs, at both national and international levels, have been proven to be valuable tools for ensuring prudent use of antimicrobials in livestock and veterinary medicine as well as for monitoring the appearance of antimicrobial resistance (Hur *et al.*, 2012; Parry, 2003). In addition to appropriate antibiotic management, improved vaccines and diagnostics as well as adherence to infection control measures (e.g., use of gloves and adequate hand hygiene in health care settings) may also be needed in order to address this public health and food safety issue (Hur *et al.*, 2012; Oteo *et al.*, 2010; Paterson, 2006). Moreover, continued research on the genetic determinants of antimicrobial resistance (e.g., plasmid sequencing projects) and on the ecology, epidemiology, and evolution of MDR strains is expected to provide a better understanding of the emergence and distribution of antimicrobial resistance, and, therefore, to allow for the design of improved control measures (Alcaine *et al.*, 2007; Louden *et al.*, 2012).

Other Bacteria

Although its foodborne pathogen status has been well-established, constituting the leading cause of sporadic bacterial gastroenteritis, *Ca. jejuni* has also been recognized as one of the most prevalent and serious emerging bacterial pathogens of meat, poultry, and derived products (Mor-Mur and Yuste, 2010). Its low infectious dose (i.e., less than 100 cells can cause disease) and its ability to survive refrigeration and freezing delineate the relevance of this organism to food safety and public health, with raw or undercooked poultry being the primary source of campylobacteriosis (Mor-Mur and Yuste, 2010). Complications of campylobacteriosis include reactive arthritis, pancreatitis, meningitis, endocarditis, and the Guillain–Barré syndrome, a disorder of the peripheral nervous system, with the latter being primarily associated with serotypes O:19, O:4, and O:1 of the pathogen (Mor-Mur and Yuste, 2010). Owing to difficulties associated with culturing of *Ca. jejuni*, it has been opined that the incidence of campylobacteriosis in the past was most likely underestimated, with foodborne outbreaks being erroneously identified as caused by other organisms such as *Salmonella* spp. (Mor-Mur and Yuste, 2010).

Bacterial species belonging to the genus *Helicobacter* have also been identified as potentially emerging foodborne pathogens. One such example is *Helicobacter pylori*, which, since its initial recognition in 1982, has been implicated as the etiological agent of gastritis and as a major contributing factor in the development of peptic gastroduodenal ulcers (Gracey, 1994; Meng and Doyle, 1998). Although humans were originally thought as the only natural host of *H. pylori*, the organism has also been isolated from nonhuman primates, suggesting that it may be a zoonotic pathogen with its transmission occurring from animals to humans (Meng and Doyle, 1998). Additional modes of *H. pylori* transmission that have been proposed include fecal–oral transfer and person-to-person spread (Gracey, 1994), though there is also evidence supporting the hypothesis of waterborne transmission (Meng and Doyle, 1998). Nevertheless, the significance of this organism as a foodborne pathogen has not been firmly established yet. *Helicobacter pullorum* is another species of potential importance as an emerging foodborne pathogen; it has been associated with diarrhea, gastroenteritis, and liver diseases in humans, to which it may be transmitted via consumption of undercooked poultry products (Skovgaard, 2007).

In addition to the aforementioned bacterial pathogens, attention should be paid to well-established foodborne pathogens, which, however, appear to reemerge exhibiting additional characteristics/abilities (e.g., higher virulence or lower infectious dose) or being associated with other, frequently unexpected, food vehicles. Examples of such foodborne pathogens are *Sa. enterica* and *Listeria monocytogenes*. Recent salmonellosis outbreaks have been associated with unexpected food products such as microwavable, ready-to-cook foods, breaded (sometime prebrowned) chicken nuggets, and chicken entrees, as well as peanut butter, demonstrating that the landscape of foodborne infections is in flux (Tauxe *et al.*, 2010). Furthermore, both *Sa. enterica* and *L. monocytogenes*, as well as other established foodborne pathogens, have been increasingly associated with illness outbreaks linked to consumption of fresh produce (CDC, 2011b, 2012; Tauxe *et al.*, 2010). For instance, an unusually large listeriosis outbreak associated with cantaloupe was reported in 2011 in the US (CDC, 2011b); this was the first epidemiologic association of *L. monocytogenes* with melon, whereas a novel serotype 1/2a outbreak strain and two novel epidemic clones of the pathogen were identified during the outbreak investigation (Lomonaco *et al.*, 2013).

Viruses

Various groups of viruses are well recognized as important agents of waterborne and foodborne illness. Among these, the gastroenteritis-causing noroviruses (NoVs) and hepatitis A virus have been identified as the most important foodborne pathogens on the basis of their highly infectious nature, and the large numbers of outbreaks and people affected (Duizer and Koopmans, 2009; Koopmans and Duizer, 2004). Despite the well-established foodborne pathogen status of certain viral agents, such as the above two groups, foodborne viruses can be regarded as an emerging problem as a whole, due to the decrease in immunity of populations in countries with high standards of hygiene observed in recent years (Koopmans and Duizer, 2004). In general, a definitive association of direct or indirect animal contact with foodborne infection in humans has not been established, and most documented foodborne viral outbreaks can be traced to foods that have been manually handled by infected food handlers (symptomatic or not) and not heated or minimally processed afterwards (Koopmans and Duizer, 2004). Although most frequently observed at the end of the food chain, viral contamination of food can occur anywhere in the process from farm to fork, and a wide variety of food items have been associated with epidemic disease including deli meats, sandwiches, bakery products, berries, dishes containing fresh (or fresh frozen) fruits and vegetables, and, most importantly, shellfish (Koopmans and Duizer, 2004). Given that viruses, unlike bacteria, are strict intracellular pathogens (i.e., cannot replicate in harvested or processed food), and, thus, viral contamination of food is not expected to increase during processing, transport, or storage, the emphasis with regard to their control should be placed on prevention of contamination by proper implementation of good hygiene practices, good manufacturing practices, and Hazard Analysis and Critical Control Points programs (Koopmans and Duizer, 2004). With reference to recommended areas for future research, these include development of simple, efficient, and reproducible detection methods of viruses in foods, assessment of viral survival on different food commodities, and determination of the duration and levels of shedding of viral pathogens in people with and without symptoms (Koopmans and Duizer, 2004).

As viruses causing gastroenteritis have been well recognized as among the most common causes of foodborne illness worldwide, with NoVs ranking number one in many industrialized countries (Duizer and Koopmans, 2009), they will not be further discussed in this article. The information provided in the following sections refers to viral pathogens that are or have the potential to be foodborne, and that would better fit the term 'emerging.'

Hepatitis Viruses

The enterically transmitted hepatitis viruses are transmitted by the fecal–oral route, either directly from person to person or indirectly when water or food contaminated with fecal material is ingested, and replicate and cause disease in the liver (Mattison *et al.*, 2009). The hepatitis A virus (HAV) is a member of the Picornaviridae family, in the genus Hepatovirus, and consists of nonenveloped, icosahedral capsids of approximately 30 nm in diameter enclosing a 7.5 kb single-stranded, polyadenylated RNA genome (Mattison *et al.*, 2009). The incidence and severity of HAV infection may vary considerably both among and within countries. In many developing regions of the world where hygienic standards (i.e., clean water, sewage systems, and proper hygiene practices) may be below acceptable standards, HAV infection is endemic, with the majority of people being infected in early childhood and virtually all adults appearing to be immune; in these areas, HAV transmission occurs primarily from person to person, and outbreaks are not that common as most infections occur among children, who generally remain asymptomatic (Koopmans and Duizer, 2004; Mattison *et al.*, 2009). In contrast, in the developed countries, where HAV endemicity is low, the majority of adults are susceptible to HAV infection, and HAV constitutes a serious and increasing public health concern (Koopmans and Duizer, 2004; Mattison *et al.*, 2009). Viral hepatitis, which is generally an acute infection but its resolution provides life-long immune protection against future infections, is characterized by fever, jaundice, light-colored stools, dark-colored urine, abdominal pain, and occasional diarrhea (Mattison *et al.*, 2009). As virus shedding may start 10–14 days before the onset of symptoms, HAV spreading can be extensive (Koopmans and Duizer, 2004). Although the accurate identification of foodborne sources of HAV infection is frequently difficult due to the long incubation period between infection and symptomatic disease, foodborne outbreaks of HAV have been associated with many different food types, and primarily with shellfish (due to fecal contamination of shellfish growing waters) and fresh or frozen produce (Mattison *et al.*, 2009).

Hepatitis E virus (HEV) is a member of the genus Hepevirus in the Hepeviridae family, with its particle being a nonenveloped icosahedron of approximately 30 nm in diameter and its genome a 7.5 kb single-stranded RNA molecule (Mattison *et al.*, 2009). Although HEV is known for its ability to cause acute clinical hepatitis, primarily as waterborne outbreaks and sporadic infections, in young adults throughout the developing world, there are recent reports on zoonotic foodborne autochthonous HEV infections in developed countries (Khuroo and Khuroo, 2008; Nicand *et al.*, 2009). In this sense, hepatitis E is regarded as an emerging concern in western countries (i.e., Western Europe, North America, Japan, and Australia), and considerable advances have been successful in understanding its epidemiology (Mattison *et al.*, 2009; Khuroo and Khuroo, 2008). Emerging HEV infections in the developed world have been more frequently associated with people of advanced age (i.e., more than 50 years) (Mattison *et al.*, 2009; Nicand *et al.*, 2009). In addition to its well-established waterborne transmission, HEV may be transmitted to humans via the following routes: (1) consumption of raw or undercooked meat of naturally infected wild (e.g., boar and deer) and domesticated (e.g., pigs) animals; (2) occupational exposure to infected animals; (3) parenteral transmission; and (4) vertical transmission from mother to child (Khuroo and Khuroo, 2008; Nicand *et al.*, 2009). The incubation period of HEV varies from 15 to 45 days, and the typical clinical symptoms of the infection are similar to those associated with HAV infection and include jaundice, dark urine, enlarged tender liver, elevated liver enzymes, abdominal pain, and tenderness accompanied by nausea, vomiting, and fever (Khuroo and Khuroo, 2008). Nevertheless, the disease may vary in severity and is often complicated by protracted coagulopathy and cholestasis, whereas chronic HEV infection, chronic hepatitis, and cirrhosis have been frequently reported in organ transplant recipients (Khuroo and Khuroo, 2008). High rates of fulminant hepatitis and fatality have been documented for classical HEV infections in pregnant women, particularly in the third trimester (Mattison *et al.*, 2009).

Other Viruses

In addition to the discussion above in Section Hepatitis Viruses, there are also some emerging viruses that, although not usually transmitted via the fecal–oral route, may infect via the gastrointestinal tract and have the potential to emerge as food safety concerns. These viruses are avian influenza viruses, the coronavirus, and the tick-borne encephalitis virus (Mattison *et al.*, 2009).

The avian influenza virus A genus is classified in the family Orthomyxoviridae and is characterized by pleomorhpic, enveloped virions with a segmented single-stranded RNA genome. The viruses of this genus are classified into subtypes based on the two envelope glycoproteins, the hemagglutinase (H type) and the neuraminidase (N type), and there are 16 known H types and 9 known N types (Mattison *et al.*, 2009). All of the known subtypes of influenza A viruses have been found in birds, and viruses containing combinations of the H1, H2, H3, N1, and N2 types are considered to be established in the human population, whereas viruses of the H5, H7, and H9 subtypes have been associated with sporadic human infections (Mattison *et al.*, 2009). Humans acquire avian influenza viruses primarily through direct contact of the mucous membranes with infectious secretions and excreta from infected birds or contaminated poultry products (Doyle and Erickson, 2006). An avian influenza virus subtype that has recently attracted the attention of public health authorities is the highly pathogenic H5N1 virus, which has been detected in poultry from more than 50 countries and it has infected humans in Vietnam, Thailand, Indonesia, Cambodia, China, Turkey, Iraq, Azerbaijan, Egypt, and Djibouti (De Jong and Hien, 2006; Mattison *et al.*, 2009). This virus replicates to extremely high levels in the upper respiratory tract, causing an intense inflammatory response, and is associated with a particularly high case fatality rate, frequently more than 60% (De Jong *et al.*, 2006). Despite its limited ability to spread from person to person, there is concern that H5N1 virus could acquire the ability to spread effectively in humans and result in a worldwide pandemic (Mattison *et al.*, 2009). Although almost all H5N1 infections of humans have been linked to close contact with infected poultry (De Jong and Hien, 2006), given that the virus has been isolated from various parts of infected

poultry such as the blood, bones and meat, the consumption of raw or undercooked poultry products as a potential source of infection cannot be renounced (Mattison *et al.*, 2009).

Although coronaviruses typically cause mild respiratory disease, a particularly virulent strain, known as the 'sudden acute respiratory syndrome coronavirus,' emerged in 2003, causing more than 8000 cases of systemic infections as well as respiratory illness, and being associated with an approximately 10% case fatality rate (Mattison *et al.*, 2009; Wang and Chang, 2004). The fact that this virus was isolated from the digestive tract, as well as from feces and sewage, raises the possibility that it may have had the potential to be transmitted through the fecal–oral route and food products (Mattison *et al.*, 2009). The tick-borne encephalitis virus is considered endemic to Europe, and is usually transmitted to humans by tick bites; nonetheless, some cases have been linked to the consumption of raw milk from infected cattle or goats (Mattison *et al.*, 2009). Finally, additional emerging viruses of interest are the gastroenteritis-causing parvoviruses, toroviruses, and picobirnaviruses (Mattison *et al.*, 2009).

Parasites

Waterborne and foodborne parasitic infections have received considerable attention in the last decade. Despite the fact that many of these infections are well recognized for many years, they are considered emerging as a result of either true higher incidence or higher detection (Dorny *et al.*, 2009). Factors that can be associated with increased human exposure to foodborne parasites and, thus, with the emergence or reemergence of many foodborne parasitic diseases as this has been documented recently on a worldwide basis, include changing eating habits (e.g., increased demand for exotic and raw food), population growth and particularly increase of population of highly susceptible people, increased international travel, globalization of food supply, changes in food production systems, climate changes, and improved diagnostic tools (Broglia and Kapel, 2011; Dorny *et al.*, 2009). A characteristic example is that of the increasing development of certain farming practices such as aquaculture, driven by the rising global demand for protein of animal origin, in developing countries where the existing health monitoring practices may not be sufficient or adequately implemented (Broglia and Kapel, 2011). It has been acknowledged that there is an urgent need for better monitoring and control of foodborne parasites, and some of the suggested means for achieving this are the use of new risk assessment tools, the development and utilization of new monitoring technologies (both serological and molecular), as well as health education (Dorny *et al.*, 2009). In general, given that foodborne parasitic infections are actually reflecting a complex system of interconnected biological, economic, social, and cultural variables, it has been proposed that their control should be based on a holistic approach, with the latter, however, requiring a large amount of high-quality data as well as systematic collaboration across sectors and disciplines (Broglia and Kapel, 2011). The information provided below refers to emerging parasites that have been associated with human infections and are or have the potential to be foodborne.

Cyclospora cayetanensis

Cyclospora cayetanensis is a single-cell coccidian protozoan, which has been implicated as the etiological agent in cases of watery diarrhea, nausea, vomiting, fatigue, and anorexia in humans and other primates (Dorny *et al.*, 2009; Rose and Slifko, 1999). Despite the fact that the infection is generally treatable with none or mild symptomatology in the immunocompetent, it may be associated with a profuse and prolonged (lasting for several months) diarrhea in immunocompromized individuals (Rose and Slifko, 1999). Outbreaks of *Cy. cayetanensis* infection have been increasingly observed since the 1990s, particularly in North America and Asia (Dorny *et al.*, 2009). Although traditionally regarded as a waterborne parasite, outbreaks of *Cy. cayetanensis* infections have also been linked to various fresh fruits, vegetables, and herbs such as blackberries, raspberries, strawberries, lettuce, and basil (Dorny *et al.*, 2009; Rose and Slifko, 1999; Smith and Evans, 2009).

Cryptosporidium and Giardia

The parasites of the genera *Cryptosporidium* and *Giardia* are well-recognized causes of protozoan waterborne diseases known as cryptosporidiosis and giardiasis, respectively. Owing to their extensive genetic diversity, the taxonomy of these parasites has frequently been a matter of debate (Dorny *et al.*, 2009). In spite of the fact that these parasites have been primarily associated with waterborne outbreaks and with infections contracted via animal handling or contact with children, they may also contaminate food commodities (e.g., soft fruits and vegetables, and shellfish) and cause infections in humans by these routes (Dorny *et al.*, 2009).

Cryptosporidium is a coccidian parasite that infects a wide range of vertebrate hosts including mammals, rodents, birds, reptiles, and fish (Smith and Evans, 2009). This parasite has emerged as an important pathogen of humans in the last 25 years, with eight described and five undescribed species potentially infecting immunocompetent and immunocompromized humans. Among these species, *Cryptosporidium hominis* and *Cryptosporidium parvum* are the most commonly detected, though the latter is the best studied species and has been identified as a major zoonotic pathogen and a significant contributor to environmental contamination with oocysts (Smith and Evans, 2009). Cryptosporidiosis is a cholera-like disease, its main symptoms are large volumes of fluid loss, fever, and abdominal pain, and its mortality rate can be high (i.e., 50%–60%) in the immunocompromized population (Rose and Slifko, 1999). Foods that have been (or suspected to be) linked to foodborne outbreaks of cryptosporidiosis include apple cider, chicken salad, and cow's milk (Smith and Evans, 2009).

The genus *Giardia* includes species that are host specific (infecting rodents, amphibians, birds, great blue herons, the prairie vole and mammals), as well as species that have zoonotic potential. The parasites that infect humans belong to the species *Giardia duodenalis*, formerly also known as *Giardia intestinalis*, and *Giardia lamblia* (Smith and Evans, 2009). The most common symptoms of giardiasis are diarrhea followed by flatulence and cramps (Rose and Slifko, 1999). Examples of foodstuffs that have been associated with documented outbreaks of giardiasis are Christmas pudding, fruit salad,

home-canned salmon, ice, noodle salad, raw sliced vegetables, sandwiches, and tripe soup, and food handlers have been identified in most of the cases as the most likely source of food contamination (Smith and Evans, 2009).

Toxoplasma gondii

Toxoplasma gondii is a coccidian protozoan parasite of man and animals with a worldwide distribution, constituting one of the most significant parasitic pathogens in Europe and the USA (Mead *et al.*, 1999; Vaillant *et al.*, 2005). Although it has a disease burden similar to that of salmonellosis and camplylobacteriosis, toxoplasmosis (i.e., the disease caused by *T. gondii*) is still regarded as a considerably underreported disease (Dorny *et al.*, 2009). On the basis of multilocus restriction fragment length polymorphism techniques, there are three genotypes within *T. gondii* (genotypes I, II, and III) which correlate with different patterns of human disease, with, however, the majority of human toxoplasmosis cases being associated with genotype II (Smith and Evans, 2009). Toxoplasmosis in immunocompetent hosts is either asymptomatic or associated with nonspecific clinical symptoms such as pyrexia, lymphadenopathy, malaise, and myalgia (Smith and Evans, 2009). Nonetheless, the disease can be severe and is generally considered as a serious health problem in pregnant women, who can pass the infection to the fetus, as well as in immunocompromized people (e.g., acquired immunodeficiency syndrome patients, organ and bone marrow transplant recipients, those with malignancies and on anticancer chemotherapy) (Dorny *et al.*, 2009; Smith and Evans, 2009).

Human toxoplasmosis can be contracted by the ingestion of sporulated oocysts present in cat feces and the environment (Dorny *et al.*, 2009). Furthermore, *T. gondii*, by being a zoonotic parasite present worldwide, has been detected in many animals used for meat production; viable parasites have been isolated from the meat of game, sheep, goat, horse, chicken, and pig (Dorny *et al.*, 2009; Smith and Evans, 2009). With particular reference to swine, a reemergence of toxoplasmosis has been observed in pigs raised in organic farms with outdoor access (Kijlstra *et al.*, 2004). Hence, *T. gondii* can also be transmitted to humans by consumption of raw or undercooked meat contaminated with tissue cysts of the parasite (Dorny *et al.*, 2009; Smith and Evans, 2009). Indeed, foodborne outbreaks of toxoplasmosis have been frequently linked to raw or rare meat, whereas additional food products associated with human infections include raw liver, goat's milk and ice cream (Smith and Evans, 2009). Nevertheless, as supported by epidemiological data, consumption of unwashed fresh vegetables or fruit has also been identified as an important risk factor with regard to toxoplasmosis (Dorny *et al.*, 2009).

Trichinella spp.

Trichinella spp. are zoonotic pathogens with widespread distribution and have been recognized as important agents of meat-borne parasitic infections in humans for a long time. The disease caused by the nematodes of the genus *Trichinella*, referred to as trichinellosis, is contracted in humans by the ingestion of larvae of the parasite that are encysted in muscle tissue of domestic or wild animal meat, with the domestic pig being identified as the most important source of the infection worldwide (Dorny *et al.*, 2009). Although, until recently, all cases of infections in both animals and humans were attributed to the species *Trichinella spiralis*, eight species and four genotypes are today recognized in the genus *Trichinella* (Dorny *et al.*, 2009). Clinical disease in humans is characterized by an intestinal phase and a subsequent parenteral (tissue) phase. Adult worms in the intestine may cause diarrhea, nausea, vomiting, fever, and abdominal pain, whereas the parenteral phase is usually accompanied by heavy muscle pains, fever, and eosinophilia (Dorny *et al.*, 2009; Murrell and Crompton, 2009). Potential complications attributed to larvae of the parasite during the early parenteral phase include facial edema, skin rash, convulsions, weight loss, meningitis, encephalitis, and vertigo, whereas death may also occur in heavily infected individuals (Murrell and Crompton, 2009).

Other Parasites

In addition to the parasites mentioned above, the taeniid tapeworms of the genus *Taenia* are also important zoonotic pathogens likely to be transmitted by food, and particularly by meat, to humans. More specifically, the species *Taenia saginata* (main reservoir: cattle), *Taenia saginata asiatica* (main reservoir: swine), and *Taenia solium* (main reservoir: swine) are the species that have been associated with human infections, and the terms cysticercosis and taeniosis refer to infections with larval and adult tapeworms, respectively (Dorny *et al.*, 2009). *Taenia saginata* is a cosmopolitan parasite found in both industrialized and developing countries; whereas *T. saginata asiatica* (which, although genetically is considered a subspecies of *T. saginata*, has distinct morphological and biological characteristics) is restricted to Asian countries (Dorny *et al.*, 2009). Intestinal taeniosis is usually contracted as the result of consumption of raw or undercooked meat, liver, or viscera, and may cause abdominal discomfort, nausea, weight loss, and occasionally more severe symptoms such as intestinal perforation and peritonitis (Dorny *et al.*, 2009).

Additional emerging parasites with a foodborne transmission potential are the intestinal trematodes of the genera *Fasciola* and *Fasciolopsis*. These parasites, and particularly the species *Fasciola hepatica*, *Fasciola gigantica*, and *Fasciolopsis buski*, may have a significant impact on the livestock sector, and cause infections in humans that are usually acquired by consumption or handling of freshwater aquatic plants (e.g., watercress and water chestnut) (Dorny *et al.*, 2009; Murrell and Crompton, 2009). Moreover, parasites that have recently gained interest are *Trypanosoma cruzi* and *Echinococcus* spp. (especially *Echinococcus granulosus* and *Echinococcus multilocularis*); infective stages of these parasites may be shed in the environment via feces and contaminate foods such as vegetables, fruits, or fruit juices (Dorny *et al.*, 2009). Finally, there is a wide variety of parasites including trematodes, cestodes, nematodes, and pentastomides that can be transmitted to humans by fish, crustaceans, reptiles, amphibians and snails when the meat of these animals is consumed raw or undercooked. Although, traditionally, such parasitic zoonoses are

most common in Asian countries (due to their particular culinary habits and the importance of aquaculture), some of them may emerge in other countries as a result of aquaculture, improved transportation and distribution systems, and tourism (Dorny *et al.*, 2009).

Concluding Remarks

Emerging foodborne pathogens, including bacteria, viruses, and parasites, are undoubtedly one of the most important food safety concerns for the food industry and public health authorities. As proposed by Buchanan (1997) several years ago, there are three research areas of interest with regard to emerging pathogens, which are still relevant and include: (1) research seeking improvements or alternatives to detect and control emerging pathogens; (2) research aiming at reducing the response between the emergence of a pathogen and its control; and (3) research identifying factors that will allow new food safety threats to be anticipated. In general, the challenge of foodborne pathogen emergence is expected to be successfully handled primarily via the development and implementation of robust and effective surveillance programs; such programs will allow for early detection and investigation of emerging (or reemerging) foodborne diseases and for the use of effective strategies for their control and prevention. Furthermore, food safety education of food handlers and consumers and prudent use of antimicrobials also are important for the control of emerging pathogens. Finally, the development and utilization of novel molecular techniques for studying foodborne pathogens, as this has been observed in the past decade, is expected to provide information that will improve one's understanding of the factors underlying the emergence of foodborne pathogens.

See also: Food Microbiology

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