Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.

Routes of Transmission in the Food Chain

3

R.A. Stein[1], M. Chirilă[2]

[1]*New York University School of Medicine, New York, NY, United States and Department of Natural Sciences, LaGuardia Community College, City University of New York, Long Island City, NY, United States;* [2]*"Iuliu Hațieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania*

3.1 INTRODUCTION

For a long time through history, diseases were thought to be caused by evil spirits, supernatural forces, or divine punishment, and rituals and sacrifices were used to get rid of them (Patwardhan et al., 2015; Peset, 2015). Infectious disease transmission was explained by influences including witchcraft, religious forces, earthquakes, comets, and meteors (Chakrabarti, 2010; Karamanou et al., 2012). In the mid-1800s, while it was already accepted that some infectious diseases such as smallpox and syphilis were contagious, ample debate surrounded the origin of certain illnesses, such as cholera and typhoid fever, and the miasma theory became the predominant model to explain cholera transmission (Tulodziecki, 2011). The miasma theory, still widespread in the early 19th century, proposed that decaying and decomposing organic matter generates new compounds that are released into the air and are poisonous to people (Halliday, 2001; Julia and Valleron, 2011; Tulodziecki, 2011).

A critical step toward understanding the fecal-oral transmission route came during the 1853–1854 London cholera outbreak, when John Snow, one of the founders of epidemiology, correlated cholera mortality with the water source. Snow applied for and received mortality reports from the General Register Office, and his conclusions relied on two different data sets (Koch and Denike, 2009). In addition to visiting houses affected by cholera to determine the water source that deceased individuals used, Snow mapped the location of their homes and that of the public water pumps (Koch and Denike, 2009; Newsom, 2006). Based on a map of 578 cholera cases that occurred in August and September 1854, Snow concluded that more deaths occurred near the pump on Broad Street, which in the 1930s became Broadwick Street (Koch, 2009; Newsom, 2006; Ramsay, 2006). This confirmed his theory about cholera transmission through contaminated water, contrary to the belief, popular at the time, that the illness was airborne (Shiode et al., 2015). While the city removed the pump handle on September 8, 1854, on Snow's recommendation, the epidemic was already subsiding at the time (Newsom, 2006; Paneth, 2004). The medical community was not convinced by Snow's evidence, and he continued to argue that the ingestion of contaminated water was the cause of cholera, a theory that was accepted only in 1866 (Ramsay, 2006).

3.2 GENERAL CONSIDERATIONS ABOUT ROUTES OF TRANSMISSION

Foodborne pathogens annually affect one-third of the world's population and approximately 48 million individuals or one in six people in the United States (Dhama et al., 2013; Kalyoussef and Feja, 2014; Schlundt et al., 2004). More than 250 different foodborne diseases have been described, and they may be caused by biological agents (such as bacteria, viruses, protozoa and other parasites, and prions), chemicals, or preformed toxins (Epp and Parker, 2009; Kalyoussef and Feja, 2014). Some foodborne pathogens, such as *Salmonella typhi*, do not have an animal reservoir and cause strictly human disease, while others, such as *Escherichia coli* or *Salmonella enteritidis*, may be transmitted to humans from animal reservoirs (see Chapters 5 and 7). Foodborne and waterborne illnesses are underestimated and underreported, and this is particularly true for infections caused by viruses, whose importance has been increasingly recognized (see Chapter 14). To a great extent, this has to do with the fact that many of the foodborne pathogens may also be transmitted via other pathways (Hall et al., 2008). Moreover, medical help is usually not sought for short-lived foodborne illnesses and, as a consequence, these are often not reported (see Chapter 2).

An aging population and increasing global mobility are two factors believed to increase the burden of foodborne infectious diseases (Koopmans et al., 2002). Globalization and centralization of the food supply, and transportation of the food progressively farther from its place of origin, have made it increasingly challenging to investigate foodborne outbreaks (Kalyoussef and Feja, 2014). The lack of signs and symptoms in all the individuals who ingested contaminated food, the fact that people may not remember everything they ate during the period immediately preceding their illness, and the presence of multiple ingredients from several sources in many food products are additional considerations that make the identification of contaminated and contaminating ingredients more challenging (see Chapter 2).

Several groups are more vulnerable to foodborne illnesses, including individuals with immunosuppression or HIV infection, transplant recipients and cancer patients, people with liver or kidney disease, the elderly, the pregnant, the very young, and people taking certain types of medication such as proton pump inhibitors (Lund and O'Brien, 2011).

While most foodborne outbreaks occur in localized areas, increasing numbers of outbreaks in the United States involve multiple states, mostly due to the rapid and widespread distribution of the food (Crowe et al., 2015). Data from the Centers for Disease Control and Prevention (CDC)'s Foodborne Disease Outbreak Surveillance System revealed that between 2010 and 2014, the 120 multistate foodborne disease outbreaks reported to the CDC accounted for 3% of all reported foodborne outbreaks but were responsible for 11% of the illnesses, 34% of the hospitalizations, and 56% of the deaths associated with foodborne outbreaks (Crowe et al., 2015). Also, in May 2011, an outbreak caused by Shiga toxin–producing *Escherichia coli* (STEC) O104:H4 was identified in Germany, and eventually it affected more than 4000 individuals in 16 countries (MMWR, 2013). An unusually high number of infected

patients developed hemolytic-uremic syndrome, making this outbreak the most dramatic since enterohemorrhagic *E. coli* (EHEC) was identified as a cause of human illness (Beutin and Martin, 2012). While initial investigations implicated mixed raw sprouts from a farm in Germany, subsequent investigations identified the source of the outbreak in a lot of fenugreek seeds that had been imported from Egypt (MMWR, 2013) (see Chapter 7).

Food contamination may occur at several steps, including the environment where animals are raised and plants are grown and harvested; during the production process such as transportation, processing, or handling; or as a result of cross-contamination from individuals preparing the food or eating it (Hall et al., 2008). For example, transportation and social stress increase the fecal excretion of *S. typhimurium* in pigs (Callaway et al., 2006; Marg et al., 2001). This is explained by the ability of the bacteria to respond to host stress-related catecholamines by activating growth and virulence gene expression (Bearson and Bearson, 2008; Stevens et al., 2009).

To schematize the routes that fecal-oral pathogens may take through the environment to reach a new host, the F-diagram was developed. This model promotes the view that diarrheal disease could be transmitted through food, flies, fields, fingers, and fluids and played a key role in developing a framework for the multiple pathways of environmental transmission of infectious diseases (Curtis et al., 2000; Eisenberg et al., 2012; Kawata, 1978; Wagner and Lanoix, 1958). Most pathogens that are excreted into the environment usually die. However, some of them reach fingers, fluids, new hosts such as flies, and floor or surfaces that are used for food preparation or eating. From any of these sites, pathogens may reach food and infect a new human host. All these transmission routes can be blocked by changes in domestic hygiene practices (Curtis et al., 2000). This model envisions two types of barriers to reduce disease transmission. Preventing transmission at the level of the primary barriers prevents the entry of the pathogens into the environment, and these initiatives are the most effective interventions to prevent disease transmission. Preventing transmission at the level of the secondary barriers involves initiatives that prevent pathogens that have already entered the environment from multiplying and reaching new hosts (Curtis et al., 2000).

3.3 **IRRIGATION WATER**

Because water is an important part of food production, processing, and preparation, pathogens can be transmitted by sewage or contaminated water that is used for irrigation or for washing or preparing food (Kalyoussef and Feja, 2014). Survival of pathogens in the irrigation water depends on the level of contamination and is generally better for helminths and viruses, which may remain viable outside a human host for months to years, than for bacteria and protozoans (Steele and Odumeru, 2004). A study that examined the transfer of *Listeria innocua* from the soil to the edible parts of lettuce revealed that the bacteria survived in the soil for at least 9 weeks during the spring. Survival rates were comparable when the bacteria were introduced

via either the compost or surface irrigation. Bacteria were transferred to the edible part of the plant, but it was not clear whether this transfer occurred by direct contact, internalization or through vectors (Oliveira et al., 2011) (see Chapter 12). In a large outbreak of verotoxin-producing *E. coli* that occurred in 2005 in Sweden, in which 135 individuals became ill including 11 individuals who developed hemolytic-uremic syndrome, lettuce emerged as the most likely cause of illness. Water samples used for irrigation tested positive for the *stx2* gene, and bacteria from the affected individuals had an identical pulsed-field gel electrophoresis (PFGE) pattern with the ones isolated from cattle upstream from the irrigation point (Soderstrom et al., 2008). In another outbreak, a *Salmonella* Newport strain was implicated in a 2002 outbreak that affected 510 individuals from 26 states in the United States, and the same strain was implicated in a 2005 outbreak that affected people in 16 states. A case–control study conducted during the second outbreak identified eating tomatoes as being associated with illness. The tomatoes were traced back to the eastern shore of Virginia, where the same strain was isolated from pond water that had been used for irrigation (Greene et al., 2008) (see Chapter 5).

From contaminated water or soil, *E. coli* and *Salmonella* were shown to be taken up into lettuce leaves (Franz et al., 2007), and *E. coli* O157:H7 sprayed on the leaves of field-grown lettuce was internalized into the leaves (Erickson et al., 2010). It is relevant that *E. coli* from cattle, sheep, and pig feces can survive on grass for at least 5–6 months, providing opportunities to contaminate animals, plants, or water (Avery et al., 2004). A study that used immunofluorescence and scanning electron microscopy reported that after radish seeds were experimentally contaminated with *E. coli* O157:H7, the bacteria could be detected on the outer surfaces and in the inner tissues of the cotyledons and in stomata (Itoh et al., 1998). *Escherichia coli* O157:H7 was transmitted from manure-contaminated soil and irrigation water to lettuce plants, and viable bacteria could be recovered from the inner tissues (Solomon et al., 2002). A study that grew radishes in soil inoculated with *Listeria monocytogenes* reported that some of the samples were positive for the bacterium for 3 months later (Van Renterghem et al., 1991). Another study revealed that when soil was inoculated with murine norovirus and Tulane virus, surrogates for human norovirus, the viruses became internalized via the roots and reached the leaf and fruit portion of strawberry plants (DiCaprio et al., 2015).

A multinational study conducted in the Czech Republic, Finland, Poland, and Serbia examined the food chain for contamination routes by monitoring for a panel of human and animal enteric viruses. During this study, samples were collected from irrigation water, animal feces, food handlers' hands, toilets on farms, conveyor belts at processing plants, and raspberries or strawberries at points of sale. Human adenovirus was found in samples from irrigation water, toilets, and a swab from a food handler's hand and on raspberries and strawberries at the point of sale. The presence of the virus both in the irrigation water and on food handler's hands indicated that these could act as vehicles allowing human pathogenic viruses to enter the food chain (Maunula et al., 2013), although food-associated adenovirus outbreaks are currently unknown (see Chapter 14).

In May 2000, 20 individuals from a scout camp in New Deer Agricultural Showground, United Kingdom, developed an outbreak caused by *E. coli* O157. An investigation revealed that about 300 sheep have been grazing the pasture, and a test on 28 animals revealed that 17 of them shed *E. coli* O157. Isolates from animal, human, and environmental sources were indistinguishable by PFGE, and in vitro studies revealed that the bacteria can survive in the field for approximately 105 days (Ogden et al., 2002).

3.4 ZOONOTIC TRANSMISSION

Many pathogens that cause foodborne illnesses can be transmitted directly from animals to humans. For example, *Taenia solium* is endemic in most of the world (Gilman et al., 2012). Humans are the only *T. solium* definitive hosts, and they carry the adult tapeworm in the intestine, while pigs are the intermediate hosts and they are infected with the larval stages, usually in the muscle (Coral-Almeida et al., 2015) (see Chapter 15). Humans can become infected during the "normal cycle" of the tapeworm, by ingesting contaminated raw or undercooked pork, and develop teniasis (Del Brutto, 2013; Murrell, 2013; Wu et al., 2016). Pigs develop porcine cysticercosis by ingesting viable *T. solium* eggs contained in the feces of human tapeworm carriers (Coral-Almeida et al., 2015). During the "aberrant transmission," for example as a result of improper food handling by tenia carriers, humans may accidentally ingest *T. solium* eggs by fecal contamination, become the intermediate host, and develop human cysticercosis (Del Brutto, 2013; Garcia and Del Brutto, 2000).

While foodborne and waterborne transmission are frequently implicated in hepatitis E outbreaks (see Chapter 14), pigs and other animals are recognized as a reservoir for the virus, and direct contact with animals or consumption of animal products has been linked to human infections (Casas and Martin, 2010; Cossaboom et al., 2016; Meng, 2013). Rotaviruses can also be transmitted from animals to humans (see Chapter 14), and this is supported by experimental and epidemiological evidence (Cook et al., 2004).

The foodborne transmission of another zoonosis came into the spotlight in 1986, when a bovine spongiform encephalopathy (BSE) epidemic affected about 200,000 cattle in Great Britain, Ireland, several other European countries, Japan, and Canada. In the United Kingdom, the epidemic peaked in January 1993 and then subsided drastically (Nathanson et al., 1997). The infectious agent was thought to originate in cattle from the meat and bone meal (MBM), the protein-rich supplement regularly fed to cattle at the beginning of their weaning, and the total MBM ban in the wake of the outbreak reduced the spread of BSE (de Vos and Heres, 2009). Contamination of the MBM appears to have been initiated by the incomplete inactivation of the agent of scrapie, a fatal prion-caused neurodegenerative disease that has been recognized in sheep from Britain for over 250 years (Nathanson et al., 1997; Taylor and Woodgate, 2003; Woolhouse et al., 2001). Meat and bone meal has been regularly fed to adult cattle, especially dairy cows, for at least half of the 20th century, especially after

1970, in Europe and the United States (Ramasamy, 2004). Meat and bone meal administration in larger amounts to dairy cattle than to beef cattle explains the significantly higher incidence of BSE in dairy cows in the United Kingdom outbreak (Bradley and Wilesmith, 1993).

Prions resist conventional procedures to inactivate viruses and are extremely resistant to disinfection, sterilization, UV radiation, formalin, and acidic conditions (Dormont, 2002; Gibbs et al., 1978; Giles et al., 2008; Jung et al., 2003; Prusiner, 1998). They survive 200°C dry heat for 1–2 h (Jung et al., 2003) and boiling for 15 min in 5% SDS at a neutral pH (Taylor, 1999), and 2 mol/L sodium hydroxide causes substantial but incomplete inactivation (Taylor, 1999).

When cattle are killed, rendering is used to separate the melted fat (tallow) from the animal tissues. Conventionally, this process has used heat, which also has a sterilizing effect (Nathanson et al., 1997; Taylor and Woodgate, 2003). Over a century ago, it was found that the protein-rich material that remains after extracting the tallow can be used as a dietary supplement for animals (Taylor and Woodgate, 2003). Separating tallow from animal tissues requires organic solvents and a high-energy input, and this process has become inefficient after fuel prices increased in the 1970s. This, along with more stringent health and safety regulations, led to a decline in the use of organic solvent for extraction. This, together with the change from high-temperature batch rendering to lower-temperature continuous processing, allowed the infectious agent to better survive the rendering process (Almond, 1998; Brewer, 2001; Nathanson et al., 1997). After the proportion of MBM that was prepared with fat solvents declined between the mid-1970s and the early 1980s from 70% to 10%, the less-effective inactivation of the prion protein is thought to have resulted in the first batches of MBM being contaminated (Almond, 1998; Nathanson et al., 1997). These factors are thought to have allowed prions, which are resistant to heat but can be inactivated by lipid solvents, to maintain their infectivity (Brewer, 2001). This gradual elimination of the solvent step did not occur in the United States, where rendering has used high-temperature treatment of the fatty tissues, and organic solvents have not been used (Brewer, 2001). The relevance of the solvent extraction step is also illustrated by the example from Scotland, where solvent extraction has not been discontinued and the rate of BSE was low. While 12.6% of the dairy herds from southern England had BSE in 1988–1989, this percentage was only 1.8% in Scotland (Nathanson et al., 1997).

Dietary exposure of humans to the BSE agent was implicated in the variant Creutzfeldt-Jakob (vCJD) disease, first reported in the United Kingdom in 1996 (Grobben et al., 2005). Strong epidemiological and laboratory evidence, including strain typing, revealed that BSE and human vCJD were caused by the same prion variant (Collinge et al., 1996; Brown et al., 2001; Belay and Schonberger, 2002; Collee et al., 2006).

Prion diseases are caused by the accumulation in the brain of PrP^{Sc}, an abnormal isoform of the host cellular prion protein PrP^{C}. The two proteins have identical amino acid sequences but differ in their secondary structures, with PrP^{Sc} having a higher beta-sheet and a lower alpha-helix content than PrP^{C} (Liemann

and Glockshuber, 1998). One of the genetic susceptibility factors to prion diseases in humans is a polymorphism at position 129 of the *PRNP* gene. Either a methionine or a valine can be found at this position. As of 2011, all but one individual who developed vCJD were homozygous at codon 129 for methionine, and a single heterozygous individual was reported (Mead et al., 2009; Colby and Prusiner, 2011). Another transmissible spongiform encephalopathy, kuru, a neurodegenerative disease described in Papua New Guinea, had an incubation period sometimes exceeding 50 years and was transmitted by ritualistic endocannibalism (which refers to the eating of relatives, as opposed to the eating of enemies, known as exocannibalism) (Gajdusek, 1977; Colby and Prusiner, 2011; Liberski, 2013). Kuru was restricted to members of the Foré linguistic group and neighboring groups but did not affect groups into which the kuru-affected people did not intermarry. In the Foré language, the word "kuru" means "to tremble with fear or cold" (Liberski, 2013). The incidence of kuru started to decline after the 1950s when endocannibalism had gradually stopped (Collinge et al., 2006). Studies that examined the molecular genetic basis of susceptibility to kuru found that homozygotes for methionine at codon 129 were overrepresented in the younger age group, while valine homozygotes and heterozygotes were overrepresented in the much older age group, and survivors were almost never carried homozygotes for methionine at this position (Liberski, 2013). Heterozygosity at this codon was associated with older patients and with longer incubation times (Mead et al., 2008). It is thought that heterozygotes were protected from disease by the more difficult interactions between the protein heterodimers, while the homologous protein–protein interactions made homozygotes more susceptible to disease (Palmer et al., 1991).

3.5 FOODBORNE PATHOGENS TRANSMITTED THROUGH INSECTS

Insects are well-recognized vectors for foodborne pathogens. Their association with decaying matter, along with their endophily (the ability to enter buildings) and synanthropy (the cohabitation with humans), are behaviors that make flies, cockroaches, and ants particularly relevant for their ability to transmit foodborne illnesses (Pava-Ripoll et al., 2015). A study of ant communities at a municipal hospital from Brazil found several bacterial species, including *Escherichia* and *Salmonella*, associated with ants (Pesquero et al., 2008). Another study trapped cockroaches in multiple buildings in Spain and found several bacterial species, including *Salmonella* (hospital), *E. coli* (food-industry plant and home care), and *Enterobacter* (hospital, catering establishment, grocery stores, and food-industry plant), associated with the insects (Garcia et al., 2012). The examination of cockroaches collected at hospitals, houses, grocery stores, animal sheds, and restaurants from the South Kanara District in southwestern India revealed that more than 4% harbored several *Salmonella* strains (Devi and Murray, 1991).

Knowledge about the food-related health risks of flies is relatively limited but, overall, a 3-fold higher prevalence of foodborne pathogens was found in the guts compared with the body surface of several fly species (Pava-Ripoll et al., 2012). House flies may contribute to pathogen dispersal in four different ways: through body hairs and surface, through the glandular hairs on the feet, by regurgitating vomitus, and by passage through their alimentary tract (Rosef and Kapperud, 1983). The danger of flies as vectors of foodborne pathogens is compounded by the fact that they have been observed to defecate at 4- to 5-min intervals throughout the day (Rosef and Kapperud, 1983). Adult houseflies were able to transmit bacteria from food to their eggs, and some of the bacteria were also transmitted to first-generation adult flies (Pava-Ripoll et al., 2015).

A study conducted in 2005 at farms from northwestern Taiwan collected 114 *Salmonella* isolates from flies and swine stool samples. Of four serotypes that were present in both flies and swine stool samples, eight of 18 PFGE patterns described were present in both, indicating the potential of the flies to function as vectors (Wang et al., 2011).

To examine bacterial populations in the Australian bush fly in three different environments, flies were captured at a cattle farm, an urban shopping center car park, and the site of a barbeque in Australia. The highest bacterial populations per fly were found in the farm environment and the lowest one in the urban environment. Multidrug resistance was detected in 94% of the *Salmonella* and 87% of the *Shigella* isolates, underscoring the potential of these flies to function as vectors for the foodborne dispersal of antimicrobial resistance (Vriesekoop and Shaw, 2010).

A study conducted in 2006 in Dormagen, Germany, trapped 12 fly species at domestic animal-related places, including a dog pound, a poultry house, a cattle barn, a horse stable, and a pigpen. *Musca domestica* was the dominant species and represented 43% of all the flies caught. All individual flies that were captured carried multiple microbial species, including potentially pathogenic and nonpathogenic microorganisms, and their capacity to act as vectors was demonstrated by the successful transfer of the microorganisms from live flies to blood agar plates (Forster et al., 2007).

To assess the ability of *Musca domestica* to transfer bacteria to clean surfaces, fluorescent *E. coli* were used to contaminate a sugar-milk aqueous solution, steak, and potato salad, and the number of bacteria transferred to the flies was quantitated. The bacteria were detected on 43%, 53%, and 62% of the flies from sugar/milk, steak, and potato salad, respectively, and contaminated flies were able to transfer the bacteria to the inner surface of a sterile jar (De Jesus et al., 2004).

In European Union countries, the prevalence of *Campylobacter* in broiler chicken batches meant that elimination of this pathogen from the chicken flocks was identified as an immediate public health priority (see Chapter 8). Flies were implicated as a vector by several studies, and their contribution makes public health initiatives more challenging (Bahrndorff et al., 2013). The housefly *Musca domestica* is the fly species most often found to carry *Campylobacter* species (Bahrndorff et al., 2014; Hald et al., 2008). The seasonality of *Campylobacter* infections, which increase in incidence in

May and June, was hypothesized to reflect the direct or indirect exposure of humans to contaminated material carried by fly species that have been in contact with human, bird, or animal feces or became contaminated by raw food. A study that plotted the time required for *M. domestica* larval development against ambient temperature from England and Wales for 1989 and 1999 found a strong relationship between the weekly number of human *Campylobacter* infections and *M. domestica* larval development times. The periods when human *Campylobacter* cases exceed a 7-day average of 170 cases per day occurred when the *M. domestica* larval development time was less than 3 weeks (Nichols, 2005).

A study that explored the benefits of fly screen interventions on *Campylobacter* prevalence among chicken flocks at broiler chicken houses from Jutland, Denmark, found that fly screens that prevented the entry of the flies into the chicken houses reduced the prevalence of *Campylobacter*-positive flocks from about 41% during the 2003–2005 period (without fly screens) to about 10% during the 2006–2009 period (with fly screens) (Bahrndorff et al., 2013). Even though the prevalence of *Campylobacter* infections and the number of flies generally peak during the summer, *Campylobact*er prevalence did not peak during the summer in the houses with fly screens (Bahrndorff et al., 2013; Nichols, 2005).

3.6 THE FOOD-PROCESSING ENVIRONMENT

Pathogens in the food-processing environment may originate from several sources, including contaminated food, food preparation surfaces, or food handlers (Todd et al., 2009). At each of the locations where food is processed or prepared, multiple factors can shape contamination and transmission. For example, in the kitchen environment, microbial pathogens can be introduced from commercial food products, by cross-contamination of the food from kitchen utensils, or by insufficient cooking or inadequate storage (Hall et al., 2008; Vogt and Dippold, 2005).

Food handlers represent an important reservoir of pathogens. A study that enrolled volunteers from a food company in Portugal found that about 20% had nasal colonization and about 11% had hand colonization with *Staphylococcus aureus*. Approximately 6% of the participants presented both nasal and hand colonization and, in some of them, PFGE analysis identified the same strain at both locations (Castro et al., 2016). Food workers may contaminate the food directly or by fomites (Sharps et al., 2012). An analysis of 191 norovirus and sapovirus outbreaks that occurred in Denmark between 2005 and 2011 revealed that 34% of the outbreaks were linked to contamination during food preparation or serving, and the food handlers were asymptomatic in 64% of them (Franck et al., 2015).

Several categories of outbreaks where food workers are involved have been described. The most typical outbreak scenario is that of an infected food worker contaminating food or food contact surfaces (Todd et al., 2007). A *Giardia lamblia* (also known as *Giardia duodenalis*; see Chapter 15) outbreak in November 1990 affected employees at a Connecticut company and resulted in 18 laboratory-confirmed and

nine suspected cases of giardiasis. A case–control study implicated raw sliced vegetables that were served in the employee cafeteria and prepared by an infected food handler. While this employee always wore gloves when preparing sandwiches, she had been seen without gloves when preparing vegetables for a salad (Mintz et al., 1993). In September 1998, a *Campylobacter* outbreak was reported in Salina, Kansas, among people who attended a luncheon at an elementary school and in several community members. Consumption of gravy or pineapple was associated with illness, and both food products were prepared in a kitchen that served six other schools, none of which experienced outbreaks. The outbreak was traced to a cafeteria worker who had diarrhea, and isolates with identical PFGE pattern were obtained from the worker and from eight lunch attendees. Four community members became ill during the same time, but the strains that they were infected with presented different PFGE patterns. This was the first time when PFGE analysis was used during the epidemiological analysis of a *Campylobacter* outbreak in the United States, and it revealed that the school cases were the result of an outbreak, while the community members were infected in unrelated, sporadic infections (Olsen et al., 2001) (see Chapter 8 for more on *Campylobacter* epidemiology).

In some outbreaks, food workers employed at more than one location may contaminate food at each of those locations. On February 24, 2012, the Long Beach Department of Health and Human Services reported the first three cases of an *S. typhimurium* outbreak. The first three culture-confirmed cases were diagnosed in two siblings aged 3 and 1, whose symptoms started on January 20 and January 24, respectively, and in an 83-year-old man whose illness started on February 2. The three strains were indistinguishable by PFGE (see Chapter 2). An investigation revealed that 19 cases, 15 confirmed and four probable, occurred during a 4-month period, and all isolates had the same electrophoretic pattern. The initial epidemiological investigation focused on several restaurants and grocery stores from the same neighborhood, where several of the affected individuals dined and shopped, but no significant findings were made. When on February 27 a neighboring jurisdiction reported a case, with a PFGE pattern matching the outbreak strain, and the person reported that within the previous 24 h she ate at the restaurant where several other cases ate, in the same neighborhood that other cases reported frequenting, an onsite visit was made to this restaurant. The restaurant owner also owned a second restaurant, which was named by two of the affected individuals. During the environmental health inspections, the owner mentioned that two of the cooks were working at both restaurants. One of these two employees tested positive for *Salmonella*, and the PFGE pattern of the strain matched the outbreak strain (Holman et al., 2014). The food items sampled at both restaurants tested negative, and an epidemiological investigation revealed that the source of the outbreak was this infected food handler, who was working at both restaurants. This example illustrates the complexity of epidemiological investigations and the need to pursue multiple venues when investigating the source of an outbreak (Holman et al., 2014).

Food workers who contaminate food products may be asymptomatic. A large typhoid fever outbreak occurred in the United States in 1989 among convention

attendees and staff at a hotel from Sullivan County, New York. The outbreak involved 43 culture-confirmed and 24 probable infections among guests, one culture-confirmed and one asymptomatic culture-confirmed case among employees, and one culture-confirmed case in the child of a hotel guest, who did not stay at the hotel and was considered to have been infected by secondary transmission. The affected patients were more likely to have consumed orange juice for one of the breakfasts, compared with control individuals. *Salmonella typhi* was isolated from the stool of an asymptomatic worker who had handled the orange juice. A survival study of the outbreak strain revealed that, at room temperature, half of the bacteria survived for 6 h in reconstituted orange juice (Birkhead et al., 1993).

Another outbreak scenario is when a food worker contaminates other workers, who infect consumers in the same or in a different establishment (Todd et al., 2007). An example of such an outbreak was documented in August 2000, when an increase in *Salmonella* Thompson infection was noted in southern California. Most affected individuals ate at a restaurant chain before becoming ill, and a case–control study implicated the consumption of burgers. The earliest onset of illness was in a burger bun packer at a bakery, who had not eaten at the restaurants from the chain but had worked while ill. The bakery supplied burger buns to some of the restaurants. This bakery employee was responsible for removing freshly baked bread and buns from the cooling rack, feeding them through an automatic slicer, and packaging the bread for distribution. She did not wear gloves and she handled every individual bread item. She became ill on July 13 but continued working, and after being hospitalized overnight on July 17 she continued working from July 18, when she was released, until July 23. Her brother became ill on July 17, and his primary duty was mixing the dough. He was removed from work by the Health Department on August 3. It was thought that the employee became ill either from his sister or from consuming contaminated buns (Kimura et al., 2005).

In certain outbreaks, it may not be clear whether food workers were the source or the victims of the outbreak (Greig et al., 2007). In 1996, after a buffet style party on a ward from a Welsh hospital, 80 of the 460 patients and staff became ill with a gastrointestinal infection. Foods associated with the outbreak included ham, coleslaw, bread rolls, cheese, and pineapple on sticks. Participants ate food from the hospital kitchen and food brought in and prepared on the ward by staff and patients. The investigation suggested that food was contaminated either during preparation by staff and patients or during the party when people served themselves (Fone et al., 1999).

Another category of foodborne outbreaks is when food contaminated at one or several sites is delivered to one or multiple locations that experience the outbreaks (Todd et al., 2007). A foodborne cyclosporidiasis outbreak that occurred in 1996 in North America was traced to raspberries imported from Guatemala (Manuel et al., 1999, 2000). In the United States, 1465 cases from 20 states, the District of Columbia, and two provinces were reported during this outbreak (Herwaldt and Ackers, 1997). In the index Canadian cluster of the outbreak, eating strawberry flan, decorated with raspberries and blueberries, was associated with an increased relative risk of acquiring the infection (Manuel et al., 1999). In another example, epidemiological

investigations and genotyping linked one source of imported frozen raspberries to eight independent norovirus outbreaks in Denmark between October 2010 and September 2011. Collectively, these outbreaks caused 242 cases, 32 of which were laboratory confirmed. The outbreaks occurred at different locations, including a company canteen in Viborg and Herlev, a hospital canteen in Køge, a conference center in Aarhus, and a café in Copenhagen. The vehicles in these outbreaks were raspberry cake (in Viborg), raspberry smoothie (in Aarhus and Copenhagen), raspberry mousse (in Herlev), and red cabbage salad with raw raspberries (in Køge). Virus isolated from one patient sample had an identical RNA sequence to the virus isolated from berries. In one of these outbreaks, the raspberries originated from a bag of mixed frozen berries that had been packaged in Belgium and raw materials from Serbia, and subsequently it was determined that they came from the same batch of contaminated raspberries that was responsible for all the other outbreaks (Muller et al., 2015).

Finally, customers, as opposed to workers, may sometimes be the source of food-borne outbreaks (Todd et al., 2007). This is illustrated by three successive gastro-enteritis outbreaks with Norwalk-like viruses that occurred between May 1998 and June 1999 in a Mediterranean-style restaurant in Melbourne, Australia. In the restaurant, food was typically served on platters and eaten with fingers, from a common platter, and guests typically would move from table to table. Phylogenetic analyses revealed that each outbreak was caused by a different norovirus strain, indicating that the virus had been introduced on multiple occasions, rather than having circulated continuously. Due to the fact that the incubation period of norovirus gastroenteritis is about 24–48 h, short incubation periods in this study were interpreted as suggesting infection before visiting the restaurant. In the first outbreak, a guest whose fecal specimens tested positive for the norovirus had a 4-h incubation period and was thought to have been the index case. In the second outbreak, one of the guests had a 2-h incubation period, but a fecal sample was not obtained from him. In the third outbreak, a food handler was positive for the virus and could have been the source of the outbreak. The virus appears to have been introduced by guests or staff, as opposed to a long-term food reservoir (Marshall et al., 2001).

Some outbreaks are linked to inadequate temperatures in the environment where food is processed or served. A foodborne outbreak in Sulyyel, about 400 km from Riyadh, was reported among participants at a wedding ceremony. Nine food items and drinks had been served at the ceremony and, of these, meat, rice, and restaurant-made sweets were significantly associated with illness. Non–typhoid group C *Salmonella* were isolated from 62 stool samples collected from individuals who became ill. Bacteria were present in stool samples from all the restaurant workers, and a health inspection revealed the lack of air conditioning and high ambient temperatures (Aljoudi et al., 2010). On September 8, 2006, an outbreak of botulism (see Chapter 19) caused by carrot juice was reported in the United States. It involved three Georgia patients who consumed carrot juice from the same bottle before becoming ill. This was followed by the recognition of botulism in a woman from Florida, on September 25, who had been hospitalized since September 16, and a few weeks later, on October 2, botulism was reported in two people from Ontario, Canada, who had

been hospitalized weeks earlier in separate hospitals, and they all consumed carrot juice (Sheth et al., 2008). Type A toxin was recovered from carrot juice produced by one manufacturer, and inadequate refrigeration was implicated. This outbreak was characterized by a rapid clinical onset, and two of the patients required mechanical ventilation for over 1 year (Sheth et al., 2008). One of the bottles that was analyzed contained very high concentrations of toxin, and this was thought to explain the rapid progression and the protracted course, despite prompt antitoxin administration (Sheth et al., 2008).

Some foodborne outbreaks are caused by the ingestion of raw food products that are contaminated. For example, several cases of human brucellosis were reported on Jeju Island in South Korea, at a restaurant where, between 2012 and 2013, customers ingested raw parts of fetal calf as a folk remedy (Yoo et al., 2015).

3.7 CROSS-CONTAMINATION AND ENVIRONMENTAL SURFACES

A study that enrolled volunteers from the Netherlands revealed that even though most consumers were knowledgeable about the importance of heating food and preventing cross-contamination, they did not always apply this knowledge in practice. Some of the participants made errors known to cause cross-contamination in the kitchen environment, such as allowing raw meat juices to come into contact with a cooked meal, and one in five participants placed unprocessed food on the same cutting surface as raw meat (Fischer et al., 2007).

To explore consumer awareness about food safety, an Internet survey of a nationally representative group of 1504 adult grocery shoppers from the United States examined their practices when handling raw poultry at home (Kosa et al., 2015). Almost 70% of the consumers reported washing or rinsing raw poultry before cooking it, an unsafe practice that may lead to the splashing of contaminated water and the transfer of pathogens to other foods and kitchen surfaces. Only 17.5% of the consumers reported storing raw poultry in the refrigerator correctly, and of the 62% of consumers who reported owning a food thermometer, only 26% or fewer reported using it to check the internal temperature of small cuts of poultry or ground poultry. Moreover, only 11% of the consumers who thawed raw poultry in cold water reported doing it correctly (Kosa et al., 2015).

Cross-contamination in the kitchen environment represents an important contributor to outbreaks, particularly through the transfer of pathogens to and from cutting boards, kitchen surfaces, sponges, and cloths. To quantitate cross-contamination between fresh-cut produce and common kitchen surfaces, a study used cocktails of rifampin-resistant *Salmonella* and rifampin-resistant *E. coli* O157:H7 for various transfer scenarios. Bacterial transfer was dependent on produce type, surface moisture, and drying time but less dependent on the type of the kitchen surface. More bacteria were transferred from kitchen surfaces to produce than from produce to kitchen surfaces (Jensen et al., 2013). A study that measured *Campylobacter* transfer

from poultry legs purchased in supermarkets to cutting boards found that transfer rates reached 81% when the product had been in contact with the cutting board for 10 min and was significantly lower when contact occurred for 1 min (Fravalo et al., 2009). In another study, both *C. jejuni* and *C. coli* were transferred from naturally contaminated raw poultry to cooked chicken via the cutting board, and transfer was detected even when the samples had low contamination levels (Guyard-Nicodeme et al., 2013).

A study conducted in Rio Grande do Sul, Brazil, collected 24 sponges from industrial kitchens, counted microorganisms on half of them, and used the other half to study the transfer of bacteria to stainless steel and polyethylene. All sponges were contaminated by heterotrophic microorganisms and about 83% were contaminated with fecal coliforms. Large numbers of microorganisms could be transferred from sponges to both surfaces. Survival of the transferred microorganisms at room temperature decreased over time, and even though the reduction was greatest in the first 4 h, viable organisms were still found after 24 h (Rossi et al., 2013).

Salmonella enteritidis, *Staphylococcus aureus*, and *Campylobacter jejuni* were shown to remain viable on dry stainless steel at room temperature and pose cross-contamination risks for a considerable time, depending on the specific pathogen and on the level of contamination (Kusumaningrum et al., 2003). *Staphylococcus aureus* was recovered from the surfaces for at least 4 days for both high (10^5 CFU/cm^2) and moderate (10^3 CFU/cm^2) contamination levels, but the number of viable bacteria decreased below the detection limit within 2 days at low contamination levels (10 CFU/cm^2). *Salmonella enteritidis* was recovered from surfaces for at least 4 days at high contamination levels, and the numbers decreased to the detection limit within 24 h at moderate levels and within 1 h at low levels. *Campylobacter jejuni* was most susceptible to slow air-drying on surfaces, and at high contamination levels their numbers decreased below the detection limit within 4 h. The microorganisms were artificially transmitted from wet sponges to stainless steel surfaces and from these surfaces to food, with transfer rates of 25–100% (Kusumaningrum et al., 2003).

During the preparation of contaminated chickens in domestic kitchens, significant bacterial dissemination may occur onto hands, cloths, and food contact surfaces such as chopping boards (Cogan et al., 1999, 2002). A study found that in the case of *Salmonella*, even low contamination levels can grow after cross-contamination, and removal of bacteria from cloths by washing was more difficult after they had been stored overnight (Cogan et al., 2002).

A study that used bologna slices that had been inoculated with different levels of *L. monocytogenes* revealed that transfer to stainless steel and to polyethylene processing surfaces increased with the inoculation level. Transfer efficiency was not statistically different to wet compared with dry processing surfaces, and greater numbers of bacteria were transferred from bologna to stainless steel than to polyethylene. Transfer was not affected by the roughness or by the finish of the stainless steel surfaces (Rodriguez et al., 2007).

Cross-contamination of food and/or environmental surfaces has been implicated in many outbreaks. In June 2005, an investigation was conducted to examine the source of gastrointestinal illness in individuals who attended luncheons catered by an Adelaide café and in people who ate at the café. To identify the source of the outbreak, 61 respondents who met the case definition of two or more gastrointestinal symptoms after attending a luncheon catered by the café were included in a study, and *S. typhimurium* phage type 64 was confirmed in 32 of them. Of the 61 individuals, 59 (96%) reported having eaten a bread roll. The same strain was detected in raw defrosted chicken recovered from the café's kitchen, suggesting cross-contamination between the chicken and one or several ingredients used to prepare the bread rolls (Moffatt et al., 2006).

In May 2005, a gastroenteritis outbreak caused by *S. enteritidis* phage type 21 was associated with attending an annual traditional fair in a small Austrian village. Exposure in all 85 affected individuals occurred at one point in time, and 20 of the 52 stool specimens that were examined, from patients and two kitchen staff, were positive for *S. enteritidis* phage type 21. Mixed salad, which included potatoes, emerged as the only food exposure that had an independent effect on disease risk, and the causative pathogen was cultured from the stock of eggs used at the fair and from all three drag swabs and one barn dust sample collected from the responsible egg-laying flock. PFGE revealed that the bacterial isolates from eggs, the flock, and humans were indistinguishable. It was hypothesized that the boiled potatoes were contaminated in the kitchen area from raw eggs that were used to prepare an egg dressing (Schmid et al., 2006).

Another outbreak in which cross-contamination was suspected occurred in 1993 at a nursing home in Brooklyn, New York, in a mixed outbreak with *Salmonella* Heidelberg and *C. jejuni* (Layton et al., 1997). From the 93 patients who became ill and submitted specimens, stool cultures were positive for *S.* Heidelberg in 24 (26%), *C. jejuni* in 14 (15%), and both microorganisms in 25 (27%) (Layton et al., 1997). A case–control study revealed that of six different dietary plans that were available, only the pureed diet was strongly associated with positive stool tests. From the 52 pureed food items that were prepared during the week preceding the outbreak, five meat or poultry items were most strongly associated with stool positivity. Stool samples from food handlers and environmental swabs from the kitchen facility were negative for the pathogens. During this outbreak, two nurses also developed gastrointestinal illness, and stool cultures were positive for *S.* Heidelberg in one of them and for *C. jejuni* in the other one. However, during the epidemiological investigation, a food handling error that had occurred several days earlier was unveiled. During the preparation of chopped liver salad, the chopped chicken liver had been placed into a bowl that still contained the juices from the raw chicken liver. The salad was blast-chilled and refrigerated until lunch the following day, but due to a coolant failure in the refrigerator, the temperature of the salad increased to 50°F. The chopped liver salad was the only common food that the two nurses ate in the hospital, and only residents on the pureed diet were offered chopped liver salad for lunch. It is interesting that during the investigation of this outbreak, the case–control study did not identify the chopped liver salad as the source of contamination, but other findings pointed

to this dish. Based on the epidemic curve, contamination of a single food item with multiple bacterial pathogens was suspected, probably due to several batches of raw chicken liver contaminated with multiple pathogens. This outbreak underscored the complexity of investigating mixed outbreaks (Layton et al., 1997).

Foodborne illness caused by multiple microorganisms is relatively rare. Another such outbreak occurred in 1968 in Jersey City, New Jersey, and was linked to a Thanksgiving dinner in the members of four related families (Janeway et al., 1971). The meal implicated in this outbreak was served to 18 people, and 17 of them became ill. Two of the victims, a 17-year-old boy and his 56-year-old mother died, and they were both had previously been in good health. The person who did not become ill ate very little and left because of an argument. *Salmonella enteritidis* was isolated from all those who attended the dinner and from several food items, including turkey stuffing, turkey slices mixed with stuffing and gravy, peas, apple pie, pumpkin pie, and cranberry sauce. It was also isolated from environmental locations in the home and from an 8-year-old female beagle, which was not in the house where the meal was prepared and served, and did not eat any of the dinner food items but was thought to have licked the vomitus of one of the individuals. Additionally, *C. perfringens* was found in the turkey meat and stuffing, and *Enterocccus faecalis* was identified in the stuffing and in the turkey carcass (Janeway et al., 1971). Illness started between 3 and 18h after eating the meal, with a median incubation of 7h. While *S. enteritidis* was initially suspected to have caused the outbreak, the severity of the clinical manifestations was striking, considering that this pathogen mostly causes mild illness, with fatalities observed only in the very young and in those with underlying pathologies. The short incubation period and the severity of the clinical manifestations were explained by the additional involvement of one or both of the other two pathogens (Janeway et al., 1971).

The complexity of transmission routes, the need for multiagency support, and the extensive epidemiological investigations that are sometimes required are illustrated by two listeriosis outbreaks from British Columbia, Canada, caused by soft ripened cheese contaminated from environmental sources (McIntyre et al., 2015). The two outbreaks occurred in February 2002 (49 illnesses) and in September 2002 (86 illnesses). Pasteurized milk and the pasteurization process were ruled out in both outbreaks, and postpasteurization contamination was implicated. In the first outbreak, *L. monocytogenes* was detected in 64% of the cheese samples tested and in about 19% of the environmental samples tested, including food contact surfaces where cheese was aged and nonfood contact surfaces such as drains, air vents, the area around the plant, and the adjacent farm. Environmental transmission of the pathogen is thought to have likely occurred from farm animals to personnel and from them to culture solutions used during cheese production. In the second outbreak, *L. monocytogenes* was detected in 76% of the cheese samples tested and in about 19% of the environmental samples collected, including an ingredient sample, water inside and outside the plant, and farm samples. Isolates that were recovered from a cistern pipe, a water-soaked

rag in the milking house, and a swallow's nest matched the PFGE profile of the outbreak strain (McIntyre et al., 2015). Birds were identified in the second outbreak as likely having contaminated the dairy plant's water supply and the cheese during the curd-washing step (McIntyre et al., 2015).

3.8 **AIRBORNE ROUTE AND FOMITES**

A range of enteric viruses can cause gastrointestinal illness (see Chapter 14). One vomiting incident can distribute up to 30 million viral particles into the environment and this, in part, explains the high potential to transmit these viruses via the airborne route and fomites (Caul, 1994; Tung-Thompson et al., 2015).

Rotavirus is the most important gastrointestinal pathogen in daycare settings (Dennehy, 2000). In addition to the fecal-oral route, rotaviruses are transmitted by nonenteral routes, which include fomites and the respiratory route (Dennehy, 2000; Prince et al., 1986). The stability of viruses in aerosols is relevant for their transmission. A study that examined the stability of aerosolized rotavirus reported that at 20°C, the half-life of an aerosolized human rotavirus strain was about 44 h at a relative humidity of 50% but decreased to about 24 h at a relative humidity of 30% and to about 3.8 h at a relative humidity of 80%. When fecally suspended virus was aerosolized, almost 80% of the airborne virus particles remained infectious after 24 h at 20°C and a relative humidity of 50% (Ijaz et al., 1985). In another study on aerosolized rotavirus, a relative humidity of 50% was optimal for survival, with 45% of the virus remaining viable after 72 h and 3% after 9 days. At 25% relative humidity, ~21% of the virus was viable after 72 h, while at 80% relative humidity, 50% of the virus was not detectable after 2 h (Sattar et al., 1984). Rotavirus RNA has been detected in air samples taken from the hospital rooms of rotavirus-infected children, suggesting that airborne spread may play a role in hospital and daycare settings (Dennehy et al., 1998). Aerosolization is also an important transmission route for noroviruses, which represent an underestimated cause of viral gastroenteritis (see Chapter 14). Airborne Norwalk-like gastroenteritis outbreaks have been reported in which airborne spread of the virus was the most likely possibility (Marks et al., 2000, 2003).

Aerosol transmission may also be another important way by which pathogens enter the food chain. A study that examined bacteria in the air at three beef processing facilities in the United States detected *E. coli* O157:H7 in about 16% and *S. enterica* in 16.5% of the 291 air samples collected from the slaughter area. Aerobic bacteria and Enterobacteriaceae were most prevalent in samples collected near hide removal areas, suggesting that hide removal likely introduces pathogens into the air by the dispersion of liquid droplets, and could be a source of hide-to-carcass contamination (Schmidt et al., 2012). Similarly, a nationwide survey conducted in Japan to assess *Salmonella* prevalence in airborne dust from layer farms surveyed 203 farms between December 2004 and March 2005 and found

that 48 (23.6%) of them were positive for *Salmonella* (Iwabuchi et al., 2010). Experimental studies also supported the possibility of nose-to-nose transmission of *S. typhimurium* among pigs (Oliveira et al., 2006, 2007) and turkeys (Harbaugh et al., 2006).

Relevant for both food establishments and homes, toilet bowls represent another potential source for environmental contamination by aerosols. To mimic an episode of acute diarrhea, a study contaminated the sidewalls and bowl water from a domestic toilet with *Serratia marcescens* or with a bacteriophage. The highest level of airborne microorganisms was achieved immediately after the first flush, when almost twice as many bacteriophage particles were detected than bacteria. Sequential flushing caused their further distribution into the air, but the numbers declined after each flush (Barker and Jones, 2005).

Apart from transmission via fomites in food-processing facilities, an unusual potential transmission route of foodborne pathogens by fomites is via currency. A five-strain mixture of *Escherichia coli* O157:H7 or *S. enteritidis* applied to the surfaces of sterile U.S. coins revealed that *E. coli* survived for up to 11 days and *Salmonella* for up to 9 days (Jiang and Doyle, 1999). In an analysis of banknotes from 10 different countries, the number of bacteria correlated with the type of banknote material. Moreover, the bacterial content on banknotes increased with the decreasing values of the "index of economic freedom," which expresses the social and/or economic status of a country. Pathogens could be isolated only after enrichment, and while their levels did not appear to be alarming, precautionary measures should be considered for the concurrent handling of food and money (Vriesekoop et al., 2010).

3.9 FOODBORNE PATHOGENS WITH MULTIPLE TRANSMISSION VEHICLES/ROUTES

Some of the foodborne illnesses may be transmitted by more than one route, complicating epidemiological investigations and making preventive interventions more challenging.

3.9.1 BOTULISM

Foodborne botulism, caused by preformed heat-labile botulism neurotoxin, is a severe and progressive neuroparalytic disease that may progress to respiratory complications and death as a result of progressive neuromuscular blockade (Brook, 2007; Lawrence et al., 2007; Peck et al., 2011; Proverbio et al., 2016; Sobel, 2005). Botulinum toxin is one of the most powerful toxins known, with commonly cited lethal dose estimates for purified botulinum toxin type A being 70 µg orally and 0.8–0.9 µg inhalationally for a 70-kg man, but lower doses have been reported in other studies, and as little as 30–100 ng could be potentially fatal via the oral route (Peck et al., 2011; Sobel, 2005). Most cases are sporadic or occur as

small outbreaks, but larger outbreaks caused by commercial food have also been reported (Sobel, 2005; Sobel et al., 2004) (see Chapter 19 for more on botulinum toxin).

Foodborne botulism is one the four naturally occurring forms of botulism that have been described, the other ones being infant botulism, adult intestinal colonization botulism, and wound botulism (Lawrence et al., 2007; Sobel, 2005) (see Chapter 19). Infant botulism is the most frequent form encountered (Lawrence et al., 2007) and is a life-threatening condition that usually occurs in infants younger than 12 months (Goonetilleke and Harris, 2004; Fenicia and Anniballi, 2009). Honey and environmental exposure are the main routes of acquiring the microorganisms (Brook, 2007; Tanzi and Gabay, 2002). Adult intestinal colonization botulism is similar to infant botulism and mostly occurs in adults with changes in the intestinal flora, including previous surgery, Crohn disease, achlorhydria, or recent antibiotic treatment (Cherington, 1998; Chia et al., 1986; Lawrence et al., 2007). Wound botulism, caused by wound contamination with *Clostridium botulinum* spores, is usually associated with trauma or intravenous drug use (Burningham et al., 1994; Lawrence et al., 2007) (see Chapter 19).

For foodborne botulism, home-canned food is the most common source of contamination (see Chapter 19). In April 2015, a large outbreak of botulism, associated with a church potluck meal in Ohio, was linked to eating salad with improperly home-canned potatoes, and in a 1983 outbreak from Illinois, sautéed onions in sandwiches from a restaurant were implicated as the vehicle (McCarty et al., 2015).

In recent years, a novel form of botulism has been attributed to pruno, an alcoholic beverage made by inmates (Walters et al., 2015). Pruno is prepared by fermentation from water, fruit, sugar, and miscellaneous ingredients (Walters et al., 2015). It was first recognized as a vehicle for botulism in 2004 and 2005, in two outbreaks from California (Vugia et al., 2009). The first outbreak, at the California state prison in Riverside County, affected four men who drank from the same batch of pruno and developed clinical manifestations 3 days later. Two of the men required intubation and all four survived. The second outbreak, at a California state prison in Monterey County, affected a man who required intubation and survived (Vugia et al., 2009). In early October 2011, foodborne botulism was diagnosed in eight maximum security prison inmates from the Utah State Prison in Draper, Utah (Prevention, 2012). Several batches of pruno were circulating among the inmates, and one of them had been prepared from oranges, grapefruit, canned fruit, water, powdered drink mix, and a baked potato. The baked potato was the only one of these ingredients that had not been used in other pruno batches, and an investigation revealed that the toxin was most likely produced when the potato was added to a mixture of the other ingredients under low-acidity, anaerobic conditions during fermentation (Prevention, 2012). Another outbreak occurred in late November 2012 in Arizona, when eight inmates from a maximum security prison developed botulism after drinking pruno from a single batch (Prevention, 2013).

3.9.2 LISTERIA

An example of a microorganism with multiple transmission routes is *L. monocytogenes*, a human pathogen that is naturally present in the soil (Moshtaghi et al., 2003; Vivant et al., 2013). *L. monocytogenes* has become a serious concern due to its ability to survive pH extremes, high salt concentrations, low water activity, and refrigeration temperatures and to multiply in diverse habitats (see Chapter 12).

The fecal-oral route is the main route of *L. monocytogenes* transmission (Butler et al., 2015; Vally et al., 2014). A study reported that 69% of transmission occurs by food, 7% by the environment, 5% by animals, and 5% by the person-to-person route, and 13% was linked to travel (Havelaar et al., 2008). In addition to food, *Listeria* can be transmitted by water (Linke et al., 2014). A variety of food vehicles have been involved in outbreaks including coleslaw, raw and cooked meat, seafood, dairy products, sandwiches, and other ready-to-eat foods (see Chapter 12). As well as raw materials, environmental sources, including factory equipment such as a commercial meat slicer, have been shown to result in product contamination and foodborne outbreaks (Vorst et al., 2006; Sheen, 2008; Keskinen et al., 2008; Garrido, 2009).

Soil is an environmental niche for *L. monocytogenes*, and the agricultural recycling of organic wastes without adequate sanitation is another transmission mechanism (Garrec et al., 2003; Vivant et al., 2013). From soil, the bacteria can also be transferred to vegetables that enter the food chain, including internalization in salad leaves through the vascular root system after irrigation with contaminated water (Chitarra et al., 2014) (see Chapter 12).

Fecal shedding of *L. monocytogenes* by dairy cattle, which are reservoirs for the pathogen, is also a strong risk factor for the contamination of milk, dairy products, and meat (Haley et al., 2015; Ivanek et al., 2006). A longitudinal study that collected fecal samples from dairy cows for 33 days and examined the daily variability of fecal shedding revealed that 94% of the cows excreted *L. monocytogenes* in feces at least once during the study period, and the prevalence of fecal shedding varied, over time, from 0% to 100%, underscoring the limitations associated with collecting cross-sectional data in a single herd. The prevalence of *L. monocytogenes* in bulk tank milk was reported to vary between 1% and more than 12% in various studies, underscoring the dangers of consuming raw milk and raw milk products (Oliver et al., 2005; Van Kessel et al., 2011). A study that collected environmental samples, milk and milk product samples, and half-udder ovine and caprine foremilk samples from 53 dairy farms in the dairy intensive area of lower Austria detected *L. monocytogenes* in ~1% of the samples, and contamination was found on about 30% of the inspected farms (Schoder et al., 2011). Samples from working boots and fecal matter had a significantly higher overall prevalence than samples from the milk processing environment, and bacterial isolation was 3–7 times more likely on farms where silage was fed to animals throughout the year compared with farms where silage was not fed to the animals (Schoder et al., 2011).

An important consideration is that *L. monocytogenes* has been detected in bio-aerosols from high-throughput chicken-slaughtering facilities, particularly in the receiving-killing, defeathering, and evisceration areas, underscoring its potential to spread to other areas, and the bacteria survived in the aerosols for several hours (Lues et al., 2007; Spurlock and Zottola, 1991).

3.9.3 NOROVIRUSES

Norovirus is one of the most infectious viruses known, and the average probability of a viral particle causing a human infection was estimated to be close to 50% (Teunis et al., 2008). Noroviruses are the most frequent causes of acute viral gastroenteritis and foodborne disease in most of the world (Moore et al., 2015). They are often spread by food, and among simple foods, transmission occurs mostly by fresh vegetables (30–40%), fruits and nuts (10–20%), mollusks (10–15%), and dairy (5–15%) (see Chapter 14). In addition to their foodborne transmission, noroviruses can also be transmitted by the person-to-person route (Moore et al., 2015). A study that analyzed 2895 norovirus outbreaks with a known transmission route that occurred in the United States between 2009 and 2013 reported that person-to-person transmission could be documented in 2425 (83.7%) and foodborne transmissions in 465 (16.1%) of these outbreaks (Vega et al., 2014). In another analysis, of 552 outbreaks documented in 20 states from the United States between 2009 and 2010, foodborne transmission occurred in 78 (14%) and person-to-person transmission in 340 (62%) cases, whereas the transmission route was not reported for the 134 remaining outbreaks (Vega et al., 2011).

About half of norovirus outbreaks have been linked to food handlers (Widdowson et al., 2005). A study examined the transfer of two norovirus strains, originating from clinical stool samples, from gloved fingertips to soft berries and lettuce, and vice versa. Virus transfer was greater from gloves to lettuce than to soft berries, and this was explained by differences in the applied pressure. Transfer from produce to glove was generally larger than transfer from glove to produce, underscoring the possibility to cross-contaminate food products via food handlers (Verhaelen et al., 2013). (see Chapter 14).

Noroviruses can also be transmitted through person-to-person contact. This is exemplified by an acute gastroenteritis outbreak during a 2011 summer camp from Spain, in which a child is thought to have transmitted the virus to other children during a bus ride from Barcelona to the campsite, and cases later also appeared in families that were not in the camp, presumably via person-to-person transmission (Solano et al., 2014).

Moderate evidence, including epidemiological studies, supports the transmission of noroviruses by aerosols and contamination of surfaces (Marks et al., 2000; Jones and Brosseau, 2015) (see Chapter 14). Transmission has also been documented on airplanes, including among staff members working in successive shifts (Kirking et al., 2010; Thornley et al., 2011).

3.10 SUPERSPREADING

Historically, models used to describe the dynamics of infectious diseases have assumed that infected individuals are homogeneous with respect to transmission of the pathogen at the population level. In other words, it was thought that an infected individual has more or less equal chances of infecting susceptible contacts and that each susceptible contact is more or less equally likely to become infected (Bolzoni et al., 2007). However, the number of infectious diseases for which heterogeneity in transmission has been documented is growing (Chao et al., 2013; Garske and Rhodes, 2008; Liebman et al., 2014). One of the key observations on the dynamics of infectious disease outbreaks at the population level is superspreading, a phenomenon that has been described for many pathogens and in many host species, including humans, animals, birds, and plants (Capparelli et al., 2009; Cronin et al., 2010; Paull et al., 2012; Reisen et al., 2009; Stein, 2011). Early in the 20th century, Mary Mallon, more widely known as Typhoid Mary, infected at least 54 people with *S. typhi* and became the first and perhaps the best documented superspreader (Gonzalez-Escobedo et al., 2011; Marineli et al., 2013; Paull et al., 2012).

Two nonexclusive mechanisms of superspreading have been described. Some individuals, known as superspreaders, create many more secondary contacts that most others in a population. Others, known as supershedders, shed larger amounts of pathogen than most others. By this definition, superspreaders reflect more the host–host interaction, while supershedders reflect more the host–microbe interaction (Chase-Topping et al., 2008).

Superspreading has been described at the level of individuals, groups of individuals, and species. For example, during the 2002–2003 severe acute respiratory syndrome outbreak, more than 71% of the cases in Hong Kong and more than 74% of the ones in Singapore were attributed to superspreading events (Li et al., 2004). A study that examined more than 15,000 Scottish sheep farms between 2003 and 2007 found that due to heterogeneities in the animal movement patterns, less than 20% of the farms contributed to more than 80% of the transmission potential (Volkova et al., 2010). Further, a single relatively uncommon avian species, the American robin (*Turdus migratorius*), appeared to be responsible for most West Nile virus–infectious mosquitoes (Kilpatrick et al., 2006).

Superspreading is important both in human and in animal species. A longitudinal study that examined *Brucella abortus* shedding in the milk of 500 water buffaloes from four herds in southern Italy, a region endemic for brucellosis, revealed that 80% of the animal tested were nonshedders, and from those that shed the bacteria, 81% were shedding at low levels ($\leq 10^3$ colony-forming units [CFU]/mL), and almost 16% were shedding large numbers of bacteria ($\geq 10^4$ CFU/mL) and acted as supershedders (Borriello et al., 2013; Capparelli et al., 2009). Culling the supershedding animals significantly reduced the percentage of animals that became positive during a 3-month follow-up period, illustrating that this approach could effectively reduce pathogen transmission in a population by identifying and selectively culling highly shedding animals (Capparelli et al., 2009).

Cattle are the primary reservoir for *E. coli* O157:H7, a bacterium that is distributed worldwide and is most often transmitted by contaminated water or food (Ahmed and Shimamoto, 2015; Bell et al., 1994; Jensen et al., 2015; King et al., 2014; Marder et al., 2014; Riley, 2014; Soon et al., 2011; Wendel et al., 2009). Infection with *E. coli* O157:H7 may be asymptomatic (Griffin et al., 1988; Rahal et al., 2012; Su and Brandt, 1995) and it may cause mild disease or nonbloody diarrhea (Lim et al., 2010; Rodrigue et al., 1995), but the pathogen is feared for the more severe outcomes, which include bloody diarrhea, hemolytic-uremic syndrome, thrombocytic thrombocytopenic purpura, and death (see Chapter 7).

Healthy cattle harbor *E. coli* O157:H7 in their gastrointestinal tract (see Chapter 7), with the terminal rectum and the rectoanal junction being some of the main colonization sites, from where the bacteria are shed into the feces (Gansheroff and O'Brien, 2000; Naylor et al., 2003; Low et al., 2005; Robinson et al., 2009; Lim et al., 2010; Cote et al., 2015; Munns et al., 2015). The bovine rectoanal junction is located between the descending colon and the anal canal, and marks the abrupt transition between two cell types, the follicle-associated columnar epithelium toward the distal colon and the nonkeratinized stratified squamous epithelium toward the anal canal (Kudva and Dean-Nystrom, 2011; Kudva et al., 2012). Even after exposure to doses of bacteria as high as 10^{10} CFU, cattle remain asymptomatic (Baines et al., 2008). As a result, some colonized animals become supershedders and disperse very large numbers of bacteria, exceeding 10^4 CFU/g of feces (Arthur et al., 2010; Cote et al., 2015; Munns et al., 2015; Naylor et al., 2003).

Surveys of cattle groups on Scottish farms have reported that *E. coli* O157 shedding was never seen on some of the farms, some farms had occasional, short periods of shedding, and a few farms showed high levels of shedding (Synge et al., 2003; Matthews et al., 2006) and that sources of variation in *E. coli* O157 prevalence across the farms was best supported by a model in which heterogeneities occurred mostly as the result of within-farm, rather than between-farm, differences (Matthews et al., 2006). In another study, 9% of cattle were high shedders, defined in the study as shedding more than 10^4 CFU/g, and these highly shedding animals were accountable for more than 96% of the bacteria that were isolated from all the animals tested (Omisakin et al., 2003). These results underscored that the high level of shedding in some animals may be of much higher importance than the infection prevalence of an entire cattle population (Omisakin et al., 2003).

E. coli O157 prevalence in cattle has been shown to be higher in the cold than in the warm months, but this trend was reversed compared with the seasonality of human infections, which were more common in the summer. This suggested that interpreting cattle prevalence data in isolation might not accurately reflect their reservoir potential and the risk of human infection. Even though the number of high-shedding cattle was similar during the cold and the warm months, high-shedding cattle were dispersing, on average, 6 times more bacteria during the summer than during the winter. This trend mirrored the number of reported human infections and

pointed toward the need to determine not only the prevalence of shedding in cattle but also the extent of shedding (Ogden et al., 2004).

Supershedding in cattle is shaped by the bacterial strain, the host, and the environment (Munns et al., 2015). For example, analyses of fecal samples in cattle from Scottish farms found a link between *E. coli* O157 phage type 21/28 and supershedding (Chase-Topping et al., 2007, 2008; Halliday et al., 2006). A study that sequenced the whole genome of SS17, a supershedder *E. coli* O157 strain, and compared it with several reference strains identified a genomic signature comprising about 60 genomic targets that could help understand the mechanistic bases of virulence and superspreading (Cote et al., 2015). Host-specific and rearing environment risk factors may also be responsible for high shedding (Chase-Topping et al., 2007; Williams et al., 2014). An important factor that influences supershedding is diet composition (Braden et al., 2004; Jacob et al., 2008; Callaway et al., 2009; Jeong et al., 2011; Munns et al., 2015). Corn-fed cattle had lower average fecal pH and higher fecal *E. coli* O157:H7 concentrations than did barley-fed cattle (Berg et al., 2004). Hay-fed cattle experimentally infected with *E. coli* O157:H7 shed the bacteria for longer than did grain-fed animals, and all animals remained healthy (Hovde et al., 1999). Increased fermentation in the hindgut, making the environment less hospitable for bacterial survival (Fox et al., 2007), is one suggested reason for this latter observation. This illustrate that many factors shape supershedding and superspreading, and these are influenced by intricate mechanisms.

3.11 CONCLUSIONS

Understanding the transmission routes of foodborne pathogens is a challenging, thought-provoking, and critical aspect for food sciences, clinical medicine, and public health. While identifying the source of an outbreak is decisive for successfully limiting it and for preventing recurrences, implementation of the interventions is often delayed or complicated by various factors, such as the increased distances to which food products are transported, the increasing global mobility of populations, and the many processes and locations that are often involved in food preparation. This is compounded by the existence of several transmission routes and various transmission vehicles that can be exploited by certain pathogens. More than 250 known foodborne diseases affect one-third of the world population, and some segments of the population, particularly the very young, the elderly, pregnant women, and immunocompromised individuals, are an a heightened risk, pointing toward the relevance of these conditions for medicine and public health. Foodborne illnesses can be transmitted through vectors, fomites, contaminated food products, directly by person-to-person contact, from animals, or through the airborne route. As an additional layer of complexity in transmission at the level of populations, in what became known as the "20/80 rule," for most infectious disease outbreaks that have been studied, a minority of hosts was found to be responsible for most transmission events.

These heterogeneities in transmission have been observed in both human and animal populations, and understanding their dynamics promises to be transformative for the successful implementation of prophylactic and therapeutic interventions.

BIBLIOGRAPHY

Ahmed, A.M., Shimamoto, T., 2015. Molecular analysis of multidrug resistance in Shiga toxin-producing *Escherichia coli* O157:H7 isolated from meat and dairy products. International Journal of Food Microbiology 193, 68–73.

Aljoudi, A.S., Al-Mazam, A., Choudhry, A.J., 2010. Outbreak of food borne Salmonella among guests of a wedding ceremony: the role of cultural factors. Journal of Family and Community Medicine 17, 29–34.

Almond, J.W., 1998. Bovine spongiform encephalopathy and new variant Creutzfeldt-Jakob disease. British Medical Bulletin 54, 749–759.

Arthur, T.M., Brichta-Harhay, D.M., Bosilevac, J.M., Kalchayanand, N., Shackelford, S.D., Wheeler, T.L., Koohmaraie, M., 2010. Super shedding of *Escherichia coli* O157:H7 by cattle and the impact on beef carcass contamination. Meat Science 86, 32–37.

Avery, S.M., Moore, A., Hutchison, M.L., 2004. Fate of *Escherichia coli* originating from livestock faeces deposited directly onto pasture. Letters in Applied Microbiology 38, 355–359.

Bahrndorff, S., Gill, C., Lowenberger, C., Skovgard, H., Hald, B., 2014. The effects of temperature and innate immunity on transmission of Campylobacter jejuni (Campylobacterales: Campylobacteraceae) between life stages of *Musca domestica* (Diptera: Muscidae). Journal of Medical Entomology 51, 670–677.

Bahrndorff, S., Rangstrup-Christensen, L., Nordentoft, S., Hald, B., 2013. Foodborne disease prevention and broiler chickens with reduced Campylobacter infection. Emerging Infectious Diseases 19, 425–430.

Baines, D., Lee, B., McAllister, T., 2008. Heterogeneity in enterohemorrhagic *Escherichia coli* O157:H7 fecal shedding in cattle is related to *Escherichia coli* O157:H7 colonization of the small and large intestine. Canadian Journal of Microbiology 54, 984–995.

Barker, J., Jones, M.V., 2005. The potential spread of infection caused by aerosol contamination of surfaces after flushing a domestic toilet. Journal of Applied Microbiology 99, 339–347.

Bearson, B.L., Bearson, S.M., 2008. The role of the QseC quorum-sensing sensor kinase in colonization and norepinephrine-enhanced motility of *Salmonella enterica* serovar Typhimurium. Microbial Pathogenesis 44, 271–278.

Belay, E.D., Schonberger, L.B., 2002. Variant Creutzfeldt-Jakob disease and bovine spongiform encephalopathy. Clinics in Laboratory Medicine 22, 849–862 (v–vi).

Bell, B.P., Goldoft, M., Griffin, P.M., Davis, M.A., Gordon, D.C., Tarr, P.I., Bartleson, C.A., Lewis, J.H., Barrett, T.J., Wells, J.G., et al., 1994. A multistate outbreak of *Escherichia coli* O157:H7-associated bloody diarrhea and hemolytic uremic syndrome from hamburgers. The Washington experience. Journal of the American Medical Association 272, 1349–1353.

Berg, J., McAllister, T., Bach, S., Stilborn, R., Hancock, D., LeJeune, J., 2004. *Escherichia coli* O157:H7 excretion by commercial feedlot cattle fed either barley- or corn-based finishing diets. Journal of Food Protection 67, 666–671.

Beutin, L., Martin, A., 2012. Outbreak of Shiga toxin-producing *Escherichia coli* (STEC) O104:H4 infection in Germany causes a paradigm shift with regard to human pathogenicity of STEC strains. Journal of Food Protection 75, 408–418.

Birkhead, G.S., Morse, D.L., Levine, W.C., Fudala, J.K., Kondracki, S.F., Chang, H.G., Shayegani, M., Novick, L., Blake, P.A., 1993. Typhoid fever at a resort hotel in New York: a large outbreak with an unusual vehicle. Journal of Infectious Diseases 167, 1228–1232.

Bolzoni, L., Real, L., De Leo, G., 2007. Transmission heterogeneity and control strategies for infectious disease emergence. PLoS One 2, e747.

Borriello, G., Peletto, S., Lucibelli, M.G., Acutis, P.L., Ercolini, D., Galiero, G., 2013. Link between geographical origin and occurrence of Brucella abortus biovars in cow and water buffalo herds. Applied and Environmental Microbiologyl 79, 1039–1043.

Braden, K.W., Blanton Jr., J.R., Allen, V.G., Pond, K.R., Miller, M.F., 2004. Ascophyllum nodosum supplementation: a preharvest intervention for reducing *Escherichia coli* O157:H7 and Salmonella spp. in feedlot steers. Journal of Food Protection 67, 1824–1828.

Bradley, R., Wilesmith, J.W., 1993. Epidemiology and control of bovine spongiform encephalopathy (BSE). British Medical Bulletin 49, 932–959.

Brewer, M.S., 2001. Bovine spongiform encephalopathy–food safety implications. Advances in Food and Nutrition Research 43, 265–317.

Brook, I., 2007. Infant botulism. Journal of Perinatology 27, 175–180.

Brown, P., Will, R.G., Bradley, R., Asher, D.M., Detwiler, L., 2001. Bovine spongiform encephalopathy and variant Creutzfeldt-Jakob disease: background, evolution, and current concerns. Emerging Infectious Diseases 7, 6–16.

Burningham, M.D., Walter, F.G., Mechem, C., Haber, J., Ekins, B.R., 1994. Wound botulism. Annals of Emergency Medicine 24, 1184–1187.

Butler, A.J., Thomas, M.K., Pintar, K.D., 2015. Expert elicitation as a means to attribute 28 enteric pathogens to foodborne, waterborne, animal contact, and person-to-person transmission routes in Canada. Foodborne Pathogens and Disease 12, 335–344.

Callaway, T.R., Carr, M.A., Edrington, T.S., Anderson, R.C., Nisbet, D.J., 2009. Diet, *Escherichia coli* O157:H7, and cattle: a review after 10 years. Current Issues in Molecular Biology 11, 67–79.

Callaway, T.R., Morrow, J.L., Edrington, T.S., Genovese, K.J., Dowd, S., Carroll, J., Dailey, J.W., Harvey, R.B., Poole, T.L., Anderson, R.C., Nisbet, D.J., 2006. Social stress increases fecal shedding of *Salmonella typhimurium* by early weaned piglets. Current Issues in Intestinal Microbiology 7, 65–71.

Capparelli, R., Parlato, M., Iannaccone, M., Roperto, S., Marabelli, R., Roperto, F., Iannelli, D., 2009. Heterogeneous shedding of Brucella abortus in milk and its effect on the control of animal brucellosis. Journal of Applied Microbiology 106, 2041–2047.

Casas, M., Martin, M., 2010. Hepatitis E virus and pigs: a zoonotic risk in Europe? Veterinary Journal 186, 135–136.

Castro, A., Santos, C., Meireles, H., Silva, J., Teixeira, P., 2016. Food handlers as potential sources of dissemination of virulent strains of *Staphylococcus aureus* in the community. Journal of Infection and Public Health 9 (2), 153–160.

Caul, E.O., 1994. Small round structured viruses: airborne transmission and hospital control. Lancet 343, 1240–1242.

Centers for Disease Control and Prevention, 2012. Botulism from drinking prison-made illicit alcohol – Utah 2011. MMWR Morbidity and Mortality Weekly Report 61, 782–784.

Centers for Disease Control and Prevention, 2013. Notes from the field: botulism from drinking prison-made illicit alcohol – Arizona, 2012. MMWR Morbidity and Mortality Weekly Report 62, 88.

Chakrabarti, P., 2010. Curing cholera: pathogens, places and poverty in South Asia. International Journal of South Asian Studies (New Delhi) 3, 153–168.

Chao, D.L., Longini Jr., I.M., Halloran, M.E., 2013. The effects of vector movement and distribution in a mathematical model of dengue transmission. PLoS One 8, e76044.

Chase-Topping, M., Gally, D., Low, C., Matthews, L., Woolhouse, M., 2008. Super-shedding and the link between human infection and livestock carriage of *Escherichia coli* O157. Nature Reviews in Microbiology 6, 904–912.

Chase-Topping, M.E., McKendrick, I.J., Pearce, M.C., MacDonald, P., Matthews, L., Halliday, J., Allison, L., Fenlon, D., Low, J.C., Gunn, G., Woolhouse, M.E.., 2007. Risk factors for the presence of high-level shedders of *Escherichia coli* O157 on Scottish farms. 45, 1594–1603.

Cherington, M., 1998. Clinical spectrum of botulism. Muscle and Nerve 21, 701–710.

Chia, J.K., Clark, J.B., Ryan, C.A., Pollack, M., 1986. Botulism in an adult associated with food-borne intestinal infection with Clostridium botulinum. New England Journal of Medicine 315, 239–241.

Chitarra, W., Decastelli, L., Garibaldi, A., Gullino, M.L., 2014. Potential uptake of *Escherichia coli* O157:H7 and Listeria monocytogenes from growth substrate into leaves of salad plants and basil grown in soil irrigated with contaminated water. International Journal of Food Microbiology 189, 139–145.

Cogan, T.A., Bloomfield, S.F., Humphrey, T.J., 1999. The effectiveness of hygiene procedures for prevention of cross-contamination from chicken carcases in the domestic kitchen. Letters in Applied Microbiology 29, 354–358.

Cogan, T.A., Slader, J., Bloomfield, S.F., Humphrey, T.J., 2002. Achieving hygiene in the domestic kitchen: the effectiveness of commonly used cleaning procedures. Journal of Applied Microbiology 92, 885–892.

Colby, D.W., Prusiner, S.B., 2011. Prions. Cold Spring Harb Perspect Biol 3, a006833.

Collee, J.G., Bradley, R., Liberski, P.P., 2006. Variant CJD (vCJD) and bovine spongiform encephalopathy (BSE): 10 and 20 years on: part 2. Folia Neuropathologica 44, 102–110.

Collinge, J., Beck, J., Campbell, T., Estibeiro, K., Will, R.G., 1996. Prion protein gene analysis in new variant cases of Creutzfeldt-Jakob disease. Lancet 348, 56.

Collinge, J., Whitfield, J., McKintosh, E., Beck, J., Mead, S., Thomas, D.J., Alpers, M.P., 2006. Kuru in the 21st century–an acquired human prion disease with very long incubation periods. Lancet 367, 2068–2074.

Cook, N., Bridger, J., Kendall, K., Gomara, M.I., El-Attar, L., Gray, J., 2004. The zoonotic potential of rotavirus. The Journal of Infection 48, 289–302.

Coral-Almeida, M., Gabriel, S., Abatih, E.N., Praet, N., Benitez, W., Dorny, P., 2015. Taenia solium human cysticercosis: a systematic review of Sero-epidemiological data from endemic zones around the world. PLoS Neglected Tropical Diseases 9, e0003919.

Cossaboom, C.M., Heffron, C.L., Cao, D., Yugo, D.M., Houk-Miles, A.E., Lindsay, D.S., Zajac, A.M., Bertke, A.S., Elvinger, F., Meng, X.J., 2016. Risk factors and sources of foodborne hepatitis E virus infection in the United States. Journal of Medical Virology 88 (9),1641–1645.

Cossart, P., Lebreton, A., 2014. A trip in the "New Microbiology" with the bacterial pathogen Listeria monocytogenes. FEBS Letters 588, 2437–2445.

Cote, R., Katani, R., Moreau, M.R., Kudva, I.T., Arthur, T.M., DebRoy, C., Mwangi, M.M., Albert, I., Raygoza Garay, J.A., Li, L., Brandl, M.T., Carter, M.Q., Kapur, V., 2015. Comparative analysis of super-shedder strains of *Escherichia coli* O157:H7 reveals distinctive genomic features and a strongly aggregative adherent phenotype on bovine rectoanal junction squamous epithelial cells. PLoS One 10, e0116743.

Cronin, J.P., Welsh, M.E., Dekkers, M.G., Abercrombie, S.T., Mitchell, C.E., 2010. Host physiological phenotype explains pathogen reservoir potential. Ecology Letters 13, 1221–1232.

Crowe, S.J., Mahon, B.E., Vieira, A.R., Gould, L.H., 2015. Vital signs: multistate foodborne outbreaks – United States, 2010–2014. MMWR Morbidity and Mortality Weekly Report 64, 1221–1225.

Curtis, V., Cairncross, S., Yonli, R., 2000. Domestic hygiene and diarrhoea - pinpointing the problem. Tropical Medecine and International Health 5, 22–32.

De Jesus, A.J., Olsen, A.R., Bryce, J.R., Whiting, R.C., 2004. Quantitative contamination and transfer of *Escherichia coli* from foods by houseflies, *Musca domestica* L. (Diptera: Muscidae). International Journal of Food Microbiology 93, 259–262.

de Vos, C.J., Heres, L., 2009. The BSE risk of processing meat and bone meal in nonruminant feed: a quantitative assessment for the Netherlands. Risk Analysis 29, 541–557.

Del Brutto, O.H., 2013. Human cysticercosis (*Taenia solium*). Tropical Parasitology 3, 100–103.

Dennehy, P.H., 2000. Transmission of rotavirus and other enteric pathogens in the home. Pediatric Infectious Disease Journal 19, S103–S105.

Dennehy, P.H., Nelson, S.M., Crowley, B.A., Saracen, C.L., 1998. Detection of rotavirus RNA in hospital air samples by polymerase chain reaction (PCR). Pediatric Research 43, 143A.

Devi, S.J., Murray, C.J., 1991. Cockroaches (Blatta and Periplaneta species) as reservoirs of drug-resistant salmonellas. Epidemiology and Infection 107, 357–361.

Dhama, K., Rajagunalan, S., Chakraborty, S., Verma, A.K., Kumar, A., Tiwari, R., Kapoor, S., 2013. Food-borne pathogens of animal origin-diagnosis, prevention, control and their zoonotic significance: a review. Pakistan Journal of Biologocial Sciences 16, 1076–1085.

DiCaprio, E., Culbertson, D., Li, J., 2015. Evidence of the internalization of animal caliciviruses via the roots of growing strawberry plants and dissemination to the fruit. Applied and Environmental Microbiology 81, 2727–2734.

Dormont, D., 2002. Prions, BSE and food. International Journal of Food Microbiology 78, 181–189.

Eisenberg, J.N., Trostle, J., Sorensen, R.J., Shields, K.F., 2012. Toward a systems approach to enteric pathogen transmission: from individual independence to community interdependence. Annual Review of Public Health 33, 239–257.

Epp, T., Parker, S., 2009. Factors in foodborne disease control: a brief overview of issues in changing zoonotic disease transmission and the roles of public health and veterinary professionals. Journal of Agromedicine 14, 228–234.

Erickson, M.C., Webb, C.C., Diaz-Perez, J.C., Phatak, S.C., Silvoy, J.J., Davey, L., Payton, A.S., Liao, J., Ma, L., Doyle, M.P., 2010. Surface and internalized *Escherichia coli* O157:H7 on field-grown spinach and lettuce treated with spray-contaminated irrigation water. Journal of Food Protection 73, 1023–1029.

Fenicia, L., Anniballi, F., 2009. Infant botulism. Annali dell'Istituto Superiore di Sanita 45, 134–146.

Fischer, A.R., De Jong, A.E., Van Asselt, E.D., De Jonge, R., Frewer, L.J., Nauta, M.J., 2007. Food safety in the domestic environment: an interdisciplinary investigation of microbial hazards during food preparation. Risk Analysis 27, 1065–1082.

Fone, D.L., Lane, W., Salmon, R.L., 1999. Investigation of an outbreak of gastroenteritis at a hospital for patients with learning difficulties. Communicable Disease and Public Health 2, 35–38.

Forster, M., Klimpel, S., Mehlhorn, H., Sievert, K., Messler, S., Pfeffer, K., 2007. Pilot study on synanthropic flies (e.g. Musca, Sarcophaga, Calliphora, Fannia, Lucilia, Stomoxys) as vectors of pathogenic microorganisms. Parasitology Research 101, 243–246.

Fox, J.T., Depenbusch, B.E., Drouillard, J.S., Nagaraja, T.G., 2007. Dry-rolled or steam-flaked grain-based diets and fecal shedding of *Escherichia coli* O157 in feedlot cattle. Journal of Animal Science 85, 1207–1212.

Franck, K.T., Lisby, M., Fonager, J., Schultz, A.C., Bottiger, B., Villif, A., Absalonsen, H., Ethelberg, S., 2015. Sources of Calicivirus contamination in foodborne outbreaks in Denmark, 2005-2011–the role of the asymptomatic food handler. Journal of Infectious Diseases 211, 563–570.

Franz, E., Visser, A.A., Van Diepeningen, A.D., Klerks, M.M., Termorshuizen, A.J., van Bruggen, A.H., 2007. Quantification of contamination of lettuce by GFP-expressing *Escherichia coli* O157:H7 and *Salmonella enterica* serovar Typhimurium. Food Microbiology 24, 106–112.

Fravalo, P., Laisney, M.J., Gillard, M.O., Salvat, G., Chemaly, M., 2009. Campylobacter transfer from naturally contaminated chicken thighs to cutting boards is inversely related to initial load. Journal of Food Protection 72, 1836–1840.

Gajdusek, D.C., 1977. Unconventional viruses and the origin and disappearance of kuru. Science 197, 943–960.

Gansheroff, L.J., O'Brien, A.D., 2000. *Escherichia coli* O157:H7 in beef cattle presented for slaughter in the U.S.: higher prevalence rates than previously estimated. Proceedings of the National Academy of Sciences of the United States of America 97 , 2959–2961.

Garcia, F., Notario, M.J., Cabanas, J.M., Jordano, R., Medina, L.M., 2012. Incidence of bacteria of public health interest carried by cockroaches in different food-related environments. Journal of Medical Entomology 49, 1481–1484.

Garcia, H.H., Del Brutto, O.H., 2000. Taenia solium cysticercosis. Infectious Disease Clinics of North America 14, 97–119 ix.

Garrec, N., Picard-Bonnaud, F., Pourcher, A.M., 2003. Occurrence of Listeria sp and L monocytogenes in sewage sludge used for land application: effect of dewatering, liming and storage in tank on survival of Listeria species. FEMS Immunology and Medical Microbiology 35, 275–283.

Garrido, V., Vitas, A.I., García-Jalón, I., 2009. Survey of Listeria monocytogenes in ready-to-eat products: prevalence by brands and retail establishments for exposure assessment of listeriosis in Northern Spain. Food Control 20, 986–991.

Garske, T., Rhodes, C.J., 2008. The effect of superspreading on epidemic outbreak size distributions. Journal of Theoretical Biology 253, 228–237.

Gibbs Jr., C.J., Gajdusek, D.C., Latarjet, R., 1978. Unusual resistance to ionizing radiation of the viruses of kuru, Creutzfeldt-Jakob disease, and scrapie. Proceedings of the National Academy of Sciences of the United States of America 75, 6268–6270.

Giles, K., Glidden, D.V., Beckwith, R., Seoanes, R., Peretz, D., DeArmond, S.J., Prusiner, S.B., 2008. Resistance of bovine spongiform encephalopathy (BSE) prions to inactivation. PLoS Pathogens 4, e1000206.

Gilman, R.H., Gonzalez, A.E., Llanos-Zavalaga, F., Tsang, V.C., Garcia, H.H., 2012. Prevention and control of Taenia solium taeniasis/cysticercosis in Peru. Pathogens and Global Health 106, 312–318.

Gonzalez-Escobedo, G., Marshall, J.M., Gunn, J.S., 2011. Chronic and acute infection of the gall bladder by Salmonella Typhi: understanding the carrier state. Nature Reviews in Microbiology 9, 9–14.

Goonetilleke, A., Harris, J.B., 2004. Clostridial neurotoxins. Journal of Neurology, Neurosurgery and Psychiatry 75 (Suppl. 3) iii35-39.

Greene, S.K., Daly, E.R., Talbot, E.A., Demma, L.J., Holzbauer, S., Patel, N.J., Hill, T.A., Walderhaug, M.O., Hoekstra, R.M., Lynch, M.F., Painter, J.A., 2008. Recurrent multistate outbreak of Salmonella Newport associated with tomatoes from contaminated fields, 2005. Epidemiology and Infection 136, 157–165.

Greig, J.D., Todd, E.C., Bartleson, C.A., Michaels, B.S., 2007. Outbreaks where food workers have been implicated in the spread of foodborne disease. Part 1. Description of the problem, methods, and agents involved. Journal of Food Protection 70, 1752–1761.

Griffin, P.M., Ostroff, S.M., Tauxe, R.V., Greene, K.D., Wells, J.G., Lewis, J.H., Blake, P.A., 1988. Illnesses associated with *Escherichia coli* O157:H7 infections. A broad clinical spectrum. Annals of Internal Medicine 109, 705–712.

Grobben, A.H., Steele, P.J., Somerville, R.A., Taylor, D.M., Schreuder, B.E., 2005. Inactivation of the BSE agent by the heat and pressure process for manufacturing gelatine. The Veterinary Record 157, 277–281.

Guyard-Nicodeme, M., Tresse, O., Houard, E., Jugiau, F., Courtillon, C., El Manaa, K., Laisney, M.J., Chemaly, M., 2013. Characterization of Campylobacter spp. transferred from naturally contaminated chicken legs to cooked chicken slices via a cutting board. International Journal of Food Microbiologyogy 164, 7–14.

Hald, B., Skovgard, H., Pedersen, K., Bunkenborg, H., 2008. Influxed insects as vectors for Campylobacter jejuni and Campylobacter coli in Danish broiler houses. Poultry Science 87, 1428–1434.

Haley, B.J., Sonnier, J., Schukken, Y.H., Karns, J.S., Van Kessel, J.A., 2015. Diversity of Listeria monocytogenes within a U.S. dairy herd, 2004-2010. Foodborne Pathogens and Disease 12, 844–850.

Hall, G., Vally, H., Kirk, M., 2008. Foodborne illnesses: overview. International Encyclopedia of Public Health 638–653.

Halliday, J.E., Chase-Topping, M.E., Pearce, M.C., McKendrick, I.J., Allison, L., Fenlon, D., Low, C., Mellor, D.J., Gunn, G.J., Woolhouse, M.E., 2006. Herd-level risk factors associated with the presence of Phage type 21/28 E. coli O157 on Scottish cattle farms. BMC Microbiology 6, 99.

Halliday, S., 2001. Death and miasma in Victorian London: an obstinate belief. British Medical Journal 323, 1469–1471.

Harbaugh, E., Trampel, D., Wesley, I., Hoff, S., Griffith, R., Hurd, H.S., 2006. Rapid aerosol transmission of Salmonella among turkeys in a simulated holding-shed environment. Poultry Science 85, 1693–1699.

Havelaar, A.H., Galindo, A.V., Kurowicka, D., Cooke, R.M., 2008. Attribution of foodborne pathogens using structured expert elicitation. Foodborne Pathogens and Disease 5, 649–659.

Herwaldt, B.L., Ackers, M.L., 1997. An outbreak in 1996 of cyclosporiasis associated with imported raspberries. The Cyclospora Working Group. New England Journal of Medicine 336, 1548–1556.

Holman, E.J., Allen, K.S., Holguin, J.R., Torno, M., Lachica, M., 2014. A community outbreak of *Salmonella enterica* serotype Typhimurium associated with an asymptomatic food handler in two local restaurants. Journal of Environmental Health 77, 18–20.

Hovde, C.J., Austin, P.R., Cloud, K.A., Williams, C.J., Hunt, C.W., 1999. Effect of cattle diet on *Escherichia coli* O157:H7 acid resistance. Applied and Environmental Microbiologyl 65, 3233–3235.

Ijaz, M.K., Sattar, S.A., Johnson-Lussenburg, C.M., Springthorpe, V.S., Nair, R.C., 1985. Effect of relative humidity, atmospheric temperature, and suspending medium on the airborne survival of human rotavirus. Canadian Journal of Microbiology 31, 681–685.

Itoh, Y., Sugita-Konishi, Y., Kasuga, F., Iwaki, M., Hara-Kudo, Y., Saito, N., Noguchi, Y., Konuma, H., Kumagai, S., 1998. Enterohemorrhagic *Escherichia coli* O157:H7 present in radish sprouts. Applied and Environmental Microbiology 64, 1532–1535.

Ivanek, R., Grohn, Y.T., Wiedmann, M., 2006. Listeria monocytogenes in multiple habitats and host populations: review of available data for mathematical modeling. Foodborne Pathogens and Disease 3, 319–336.

Iwabuchi, E., Maruyama, N., Hara, A., Nishimura, M., Muramatsu, M., Ochiai, T., Hirai, K., 2010. Nationwide survey of salmonella prevalence in environmental dust from layer farms in Japan. Journal of Food Protection 73, 1993–2000.

Jacob, M.E., Parsons, G.L., Shelor, M.K., Fox, J.T., Drouillard, J.S., Thomson, D.U., Renter, D.G., Nagaraja, T.G., 2008. Feeding supplemental dried distiller's grains increases faecal shedding of *Escherichia coli* O157 in experimentally inoculated calves. Zoonoses Public Health 55, 125–132.

Janeway, C.M., Goldfield, M., Altman, R., Rosenfeld, H., Jedynak, C., Moulton, D., Lezynski, W., 1971. Foodborne outbreak of gastroenteritis possibily of multiple bacterial etiology. American Journal of Epidemiology 94, 135–141.

Jayarao, B.M., Donaldson, S.C., Straley, B.A., Sawant, A.A., Hegde, N.V., Brown, J.L., 2006. A survey of foodborne pathogens in bulk tank milk and raw milk consumption among farm families in Pennsylvania. Journal of Dairy Science 89, 2451–2458.

Jensen, D.A., Friedrich, L.M., Harris, L.J., Danyluk, M.D., Schaffner, D.W., 2013. Quantifying transfer rates of Salmonella and *Escherichia coli* O157:H7 between fresh-cut produce and common kitchen surfaces. Journal of Food Protection 76, 1530–1538.

Jensen, D.A., Friedrich, L.M., Harris, L.J., Danyluk, M.D., Schaffner, D.W., 2015. Cross contamination of *Escherichia coli* O157:H7 between lettuce and wash water during home-scale washing. Food Microbiology 46, 428–433.

Jeong, K.C., Kang, M.Y., Kang, J., Baumler, D.J., Kaspar, C.W., 2011. Reduction of *Escherichia coli* O157:H7 shedding in cattle by addition of chitosan microparticles to feed. Applied and Environmental Microbiologyl 77, 2611–2616.

Jiang, X., Doyle, M.P., 1999. Fate of *Escherichia coli* O157:H7 and Salmonella Enteritidis on currency. Journal of Food Protection 62, 805–807.

Jones, R.M., Brosseau, L.M., 2015. Aerosol transmission of infectious disease. Journal of Occupational and Environmental Medicine 57, 501–508.

Julia, C., Valleron, A.J., 2011. Louis-Rene Villerme (1782–1863), a pioneer in social epidemiology: re-analysis of his data on comparative mortality in Paris in the early 19th century. Journal of Epidemiology and Community Health 65, 666–670.

Jung, M.J., Pistolesi, D., Pana, A., 2003. Prions, prion diseases and decontamination. Igiene e Sanita Pubblica 59, 331–344.

Kalyoussef, S., Feja, K.N., 2014. Foodborne illnesses. Adv Pediatr 61, 287–312.

Karamanou, M., Panayiotakopoulos, G., Tsoucalas, G., Kousoulis, A.A., Androutsos, G., 2012. From miasmas to germs: a historical approach to theories of infectious disease transmission. Le Infezioni in Medicina : Rivista Periodica di Eziologia, Epidemiologia, Diagnostica, Clinica e Terapia delle Patologie Infettive 20, 58–62.

Kawata, K., 1978. Water and other environmental interventions–the minimum investment concept. The American Journal of Clinical Nutrition 31, 2114–2123.

Keskinen, L.A., Todd, E.C., Ryser, E.T., 2008. Transfer of surface-dried Listeria monocytogenes from stainless steel knife blades to roast Turkey breast. Journal of Food Protection 71, 176–181.

Kilpatrick, A.M., Daszak, P., Jones, M.J., Marra, P.P., Kramer, L.D., 2006. Host heterogeneity dominates West Nile virus transmission. Proceedings in Biological Sciences 273, 2327–2333.

Kimura, A.C., Palumbo, M.S., Meyers, H., Abbott, S., Rodriguez, R., Werner, S.B., 2005. A multi-state outbreak of Salmonella serotype Thompson infection from commercially distributed bread contaminated by an ill food handler. Epidemiology and Infection 133, 823–828.

King, L.A., Loukiadis, E., Mariani-Kurkdjian, P., Haeghebaert, S., Weill, F.X., Baliere, C., Ganet, S., Gouali, M., Vaillant, V., Pihier, N., Callon, H., Novo, R., Gaillot, O., Thevenot-Sergentet, D., Bingen, E., Chaud, P., de Valk, H., 2014. Foodborne transmission of sorbitol-fermenting *Escherichia coli* O157:[H7] via ground beef: an outbreak in northern France, 2011. Clinical Microbiology and Infection 20, O1136–O1144.

Kirking, H.L., Cortes, J., Burrer, S., Hall, A.J., Cohen, N.J., Lipman, H., Kim, C., Daly, E.R., Fishbein, D.B., 2010. Likely transmission of norovirus on an airplane, October 2008. Clinical Infectious Diseases 50, 1216–1221.

Koch, T., 2009. Disease mapping. International Encyclopedia of Human Geography 234–241.

Koch, T., Denike, K., 2009. Crediting his critics' concerns: remaking John Snow's map of Broad Street cholera, 1854. Social Science and Medicine 69, 1246–1251.

Koopmans, M., von Bonsdorff, C.H., Vinje, J., de Medici, D., Monroe, S., 2002. Foodborne viruses. FEMS Microbiology Reviews 26, 187–205.

Kosa, K.M., Cates, S.C., Bradley, S., Chambers, E.t., Godwin, S., 2015. Consumer-reported handling of raw poultry products at home: results from a national survey. Journal of Food Protection 78, 180–186.

Kudva, I.T., Dean-Nystrom, E.A., 2011. Bovine recto-anal junction squamous epithelial (RSE) cell adhesion assay for studying *Escherichia coli* O157 adherence. Journal of Applied Microbiology 111, 1283–1294.

Kudva, I.T., Griffin, R.W., Krastins, B., Sarracino, D.A., Calderwood, S.B., John, M., 2012. Proteins other than the locus of enterocyte effacement-encoded proteins contribute to *Escherichia coli* O157:H7 adherence to bovine rectoanal junction stratified squamous epithelial cells. BMC Microbiology 12, 103.

Kusumaningrum, H.D., Riboldi, G., Hazeleger, W.C., Beumer, R.R., 2003. Survival of foodborne pathogens on stainless steel surfaces and cross-contamination to foods. International Journal of Food Microbiology 85, 227–236.

Lawrence, D.T., Dobmeier, S.G., Bechtel, L.K., Holstege, C.P., 2007. Food poisoning. Emergency Medicine Clinics of North America 25, 357–373 abstract ix.

Layton, M.C., Calliste, S.G., Gomez, T.M., Patton, C., Brooks, S., 1997. A mixed foodborne outbreak with Salmonella heidelberg and Campylobacter jejuni in a nursing home. Infection Control and Hospital Epidemiology 18, 115–121.

Li, J., Paredes-Sabja, D., Sarker, M.R., McClane, B.A., 2009. Further characterization of *Clostridium perfringens* small acid soluble protein-4 (Ssp4) properties and expression. PLoS One 4, e6249.

Li, Y., Yu, I.T., Xu, P., Lee, J.H., Wong, T.W., Ooi, P.L., Sleigh, A.C., 2004. Predicting super spreading events during the 2003 severe acute respiratory syndrome epidemics in Hong Kong and Singapore. American Journal of Epidemiology 160, 719–728.

Liberski, P.P., 2013. Kuru: a journey back in time from Papua New Guinea to the neanderthals' extinction. Pathogens 2, 472–505.

Liebman, K.A., Stoddard, S.T., Reiner Jr., R.C., Perkins, T.A., Astete, H., Sihuincha, M., Halsey, E.S., Kochel, T.J., Morrison, A.C., Scott, T.W., 2014. Determinants of heterogeneous blood feeding patterns by *Aedes aegypti* in Iquitos, Peru. PLoS Negl Trop Dis 8, e2702.

Liemann, S., Glockshuber, R., 1998. Transmissible spongiform encephalopathies. Biochemical and Biophysical and Research Communications 250, 187–193.

Lim, J.Y., Yoon, J., Hovde, C.J., 2010. A brief overview of *Escherichia coli* O157:H7 and its plasmid O157. Journal of Microbiology and Biotechnology 20, 5–14.

Linke, K., Ruckerl, I., Brugger, K., Karpiskova, R., Walland, J., Muri-Klinger, S., Tichy, A., Wagner, M., Stessl, B., 2014. Reservoirs of listeria species in three environmental ecosystems. Applied and Environmental Microbiology 80, 5583–5592.

Low, J.C., McKendrick, I.J., McKechnie, C., Fenlon, D., Naylor, S.W., Currie, C., Smith, D.G., Allison, L., Gally, D.L., 2005. Rectal carriage of enterohemorrhagic *Escherichia coli* O157 in slaughtered cattle. Applied and Environmental Microbiologyl 71, 93–97.

Lozniewski, A., Humbert, A., Corsaro, D., Schwartzbrod, J., Weber, M., Le Faou, A., 2001. Comparison of sludge and clinical isolates of Listeria monocytogenes. Letters in Applied Microbiology 32, 336–339.

Lues, J.F., Theron, M.M., Venter, P., Rasephei, M.H., 2007. Microbial composition in bioaerosols of a high-throughput chicken-slaughtering facility. Poultry Science 86, 142–149.

Lund, B.M., O'Brien, S.J., 2011. The occurrence and prevention of foodborne disease in vulnerable people. Foodborne Pathogens and Disease 8, 961–973.

Manuel, D., Neamatullah, S., Shahin, R., Reymond, D., Keystone, J., Carlson, J., Le Ber, C., Herwaldt, B., Werker, D., 2000. An outbreak of cyclosporiasis in 1996 associated with consumption of fresh berries- Ontario. Canadian Journal of Infectious Diseases 11, 86–92.

Manuel, D.G., Shahin, R., Lee, W., Grmusa, M., 1999. The first reported cluster of food-borne cyclosporiasis in Canada. Canadian Journal of Public Health 90, 399–402.

Marder, E.P., Garman, K.N., Ingram, L.A., Dunn, J.R., 2014. Multistate outbreak of *Escherichia coli* O157:H7 associated with bagged salad. Foodborne Pathogens and Disease 11, 593–595.

Marg, H., Scholz, H.C., Arnold, T., Rosler, U., Hensel, A., 2001. Influence of long-time transportation stress on re-activation of *Salmonella typhimurium* DT104 in experimentally infected pigs. Berliner und Munchener tierarztliche Wochenschrift 114, 385–388.

Marineli, F., Tsoucalas, G., Karamanou, M., Androutsos, G., 2013. Mary Mallon (1869-1938) and the history of typhoid fever. Annals of Gastroenterology 26, 132–134.

Marks, P.J., Vipond, I.B., Carlisle, D., Deakin, D., Fey, R.E., Caul, E.O., 2000. Evidence for airborne transmission of Norwalk-like virus (NLV) in a hotel restaurant. Epidemiology and Infection 124, 481–487.

Marks, P.J., Vipond, I.B., Regan, F.M., Wedgwood, K., Fey, R.E., Caul, E.O., 2003. A school outbreak of Norwalk-like virus: evidence for airborne transmission. Epidemiology and Infection 131, 727–736.

Marshall, J.A., Yuen, L.K., Catton, M.G., Gunesekere, I.C., Wright, P.J., Bettelheim, K.A., Griffith, J.M., Lightfoot, D., Hogg, G.G., Gregory, J., Wilby, R., Gaston, J., 2001. Multiple outbreaks of Norwalk-like virus gastro-enteritis associated with a Mediterranean-style restaurant. Journal of Medical Microbiology 50, 143–151.

Matthews, L., McKendrick, I.J., Ternent, H., Gunn, G.J., Synge, B., Woolhouse, M.E., 2006. Super-shedding cattle and the transmission dynamics of *Escherichia coli* O157. Epidemiology and Infection 134, 131–142.

Maunula, L., Kaupke, A., Vasickova, P., Soderberg, K., Kozyra, I., Lazic, S., van der Poel, W.H., Bouwknegt, M., Rutjes, S., Willems, K.A., Moloney, R., D'Agostino, M., de Roda Husman, A.M., von Bonsdorff, C.H., Rzezutka, A., Pavlik, I., Petrovic, T., Cook, N., 2013. Tracing enteric viruses in the European berry fruit supply chain. International Journal of Food Microbiology 167, 177–185.

McCarty, C.L., Angelo, K., Beer, K.D., Cibulskas-White, K., Quinn, K., de Fijter, S., Bokanyi, R., St Germain, E., Baransi, K., Barlow, K., Shafer, G., Hanna, L., Spindler, K., Walz, E., DiOrio, M., Jackson, B.R., Luquez, C., Mahon, B.E., Basler, C., Curran, K., Matanock, A., Walsh, K., Slifka, K.J., Rao, A.K., 2015. Large outbreak of botulism associated with a church potluck Meal–Ohio, 2015. MMWR Morbidity and Mortality Weekly Report 64, 802–803.

McIntyre, L., Wilcott, L., Naus, M., 2015. Listeriosis outbreaks in British Columbia, Canada, caused by soft ripened cheese contaminated from environmental sources. Biomedical Research International 2015, 131623.

Mead, S., Poulter, M., Uphill, J., Beck, J., Whitfield, J., Webb, T.E., Campbell, T., Adamson, G., Deriziotis, P., Tabrizi, S.J., Hummerich, H., Verzilli, C., Alpers, M.P., Whittaker, J.C., Collinge, J., 2009. Genetic risk factors for variant Creutzfeldt-Jakob disease: a genome-wide association study. Lancet Neurology 8, 57–66.

Mead, S., Whitfield, J., Poulter, M., Shah, P., Uphill, J., Beck, J., Campbell, T., Al-Dujaily, H., Hummerich, H., Alpers, M.P., Collinge, J., 2008. Genetic susceptibility, evolution and the kuru epidemic. Philosophical Transactions of the Royal Society of London. Series B Biological Sciences 363, 3741–3746.

Meng, X.J., 2013. Zoonotic and foodborne transmission of hepatitis E virus. Seminars in Liver Disease 33, 41–49.

Mintz, E.D., Hudson-Wragg, M., Mshar, P., Cartter, M.L., Hadler, J.L., 1993. Foodborne giardiasis in a corporate office setting. Journal of Infectious Diseases 167, 250–253.

MMWR, 2013. Outbreak of *Escherichia coli* O104:H4 infections associated with sprout consumption – Europe and North America, May–July 2011. MMWR Morbidity and Mortality Weekly Report 62, 1029–1031.

Moffatt, C.R., Combs, B.G., Mwanri, L., Holland, R., Delroy, B., Cameron, S., Givney, R.C., 2006. An outbreak of Salmonella Typhimurium phage type 64 gastroenteritis linked to catered luncheons in Adelaide, South Australia, June 2005. Communicable Diseases Intelligence Quaterly Report 30, 443–448.

Moore, M.D., Goulter, R.M., Jaykus, L.A., 2015. Human norovirus as a foodborne pathogen: challenges and developments. Annual Review of Food Science and Technology 6, 411–433.

Moshtaghi, H., Garg, S.R., Mandokhot, U.V., 2003. Prevalence of Listeria in soil. Indian Journal of Experimental Biology 41, 1466–1468.

Muller, L., Schultz, A.C., Fonager, J., Jensen, T., Lisby, M., Hindsdal, K., Krusell, L., Eshoj, A., Moller, L.T., Porsbo, L.J., Bottiger, B.E., Kuhn, K., Engberg, J., Ethelberg, S., 2015. Separate norovirus outbreaks linked to one source of imported frozen raspberries by molecular analysis, Denmark, 2010–2011. Epidemiology and Infection 143, 2299–2307.

Munns, K.D., Selinger, L.B., Stanford, K., Guan, L., Callaway, T.R., McAllister, T.A., 2015. Perspectives on super-shedding of *Escherichia coli* O157:H7 by cattle. Foodborne Pathogens and Disease 12, 89–103.

Murrell, K.D., 2013. Zoonotic foodborne parasites and their surveillance. Revue Scietifique et Technique 32, 559–569.

Nathanson, N., Wilesmith, J., Griot, C., 1997. Bovine spongiform encephalopathy (BSE): causes and consequences of a common source epidemic. American Journal of Epidemiology 145, 959–969.

Naylor, S.W., Low, J.C., Besser, T.E., Mahajan, A., Gunn, G.J., Pearce, M.C., McKendrick, I.J., Smith, D.G., Gally, D.L., 2003. Lymphoid follicle-dense mucosa at the terminal rectum is the principal site of colonization of enterohemorrhagic *Escherichia coli* O157:H7 in the bovine host. Infection and Immunity 71, 1505–1512.

Newsom, S.W., 2006. Pioneers in infection control: John Snow, Henry Whitehead, the broad street pump, and the beginnings of geographical epidemiology. The Journal of Hospital Infection 64, 210–216.

Nichols, G.L., 2005. Fly transmission of Campylobacter. Emerging Infectious Diseases 11, 361–364.

Ogden, I.D., Hepburn, N.F., MacRae, M., Strachan, N.J., Fenlon, D.R., Rusbridge, S.M., Pennington, T.H., 2002. Long-term survival of *Escherichia coli* O157 on pasture following an outbreak associated with sheep at a scout camp. Letters in Applied Microbiology 34, 100–104.

Ogden, I.D., MacRae, M., Strachan, N.J., 2004. Is the prevalence and shedding concentrations of *E. coli* O157 in beef cattle in Scotland seasonal? FEMS Microbiology Letters 233, 297–300.

Oliveira, C.J., Carvalho, L.F., Garcia, T.B., 2006. Experimental airborne transmission of Salmonella Agona and Salmonella Typhimurium in weaned pigs. Epidemiology and Infection 134, 199–209.

Oliveira, C.J., Garcia, T.B., Carvalho, L.F., Givisiez, P.E., 2007. Nose-to-nose transmission of Salmonella Typhimurium between weaned pigs. Veterinary Microbiology 125, 355–361.

Oliveira, M., Usall, J., Vinas, I., Solsona, C., Abadias, M., 2011. Transfer of Listeria innocua from contaminated compost and irrigation water to lettuce leaves. Food Microbiology 28, 590–596.

Oliver, S.P., Jayarao, B.M., Almeida, R.A., 2005. Foodborne pathogens in milk and the dairy farm environment: food safety and public health implications. Foodborne Pathogens and Disease 2, 115–129.

Olsen, S.J., Hansen, G.R., Bartlett, L., Fitzgerald, C., Sonder, A., Manjrekar, R., Riggs, T., Kim, J., Flahart, R., Pezzino, G., Swerdlow, D.L., 2001. An outbreak of Campylobacter jejuni infections associated with food handler contamination: the use of pulsed-field gel electrophoresis. Journal of Infectious Diseases 183, 164–167.

Omisakin, F., MacRae, M., Ogden, I.D., Strachan, N.J., 2003. Concentration and prevalence of *Escherichia coli* O157 in cattle feces at slaughter. Applied and Environmental Microbiologyl 69, 2444–2447.

Palmer, M.S., Dryden, A.J., Hughes, J.T., Collinge, J., 1991. Homozygous prion protein genotype predisposes to sporadic Creutzfeldt-Jakob disease. Nature 352, 340–342.

Paneth, N., 2004. Assessing the contributions of John Snow to epidemiology: 150 years after removal of the broad street pump handle. Epidemiology 15, 514–516.

Patwardhan, B., Mutalik, G., Tillu, G., 2015. Chapter 3-concepts of health and disease. Integrative Approaches for Health 53–78.

Paull, S.H., Song, S., McClure, K.M., Sackett, L.C., Kilpatrick, A.M., Johnson, P.T., 2012. From superspreaders to disease hotspots: linking transmission across hosts and space. Frontiers in Ecology and the Environment 10, 75–82.

Pava-Ripoll, M., Pearson, R.E., Miller, A.K., Tall, B.D., Keys, C.E., Ziobro, G.C., 2015a. Ingested *Salmonella enterica*, *Cronobacter sakazakii*, *Escherichia coli* O157:H7, and Listeria monocytogenes: transmission dynamics from adult house flies to their eggs and first filial (F1) generation adults. BMC Microbiology 15, 150.

Pava-Ripoll, M., Pearson, R.E., Miller, A.K., Ziobro, G.C., 2012. Prevalence and relative risk of *Cronobacter* spp., Salmonella spp., and Listeria monocytogenes associated with the body surfaces and guts of individual filth flies. Applied and Environmental Microbiology 78, 7891–7902.

Pava-Ripoll, M., Pearson, R.E., Miller, A.K., Ziobro, G.C., 2015b. Detection of foodborne bacterial pathogens from individual filth flies. Journal of Visualised Experiments e52372.

Peck, M.W., Stringer, S.C., Carter, A.T., 2011. Clostridium botulinum in the post-genomic era. Food Microbiology 28, 183–191.

Peset, J.L., 2015. Plagues and Diseases in History. International Encyclopedia of the Social & Behavioral Sciences, second ed. , pp. 174–179.

Pesquero, M.A., Elias Filho, J., Carneiro, L.C., Feitosa, S.B., Oliveira, M.A., Quintana, R.C., 2008. Ants in a hospital environment and its importance as vector of bacteria. Neotropical Entomology 37, 472–477.

Prince, D.S., Astry, C., Vonderfecht, S., Jakab, G., Shen, F.M., Yolken, R.H., 1986. Aerosol transmission of experimental rotavirus infection. Pediatric Infectioous Disease 5, 218–222.

Proverbio, M.R., Lamba, M., Rossi, A., Siani, P., 2016. Early diagnosis and treatment in a child with foodborne botulism. Anaerobe 39, 189–192.

Prusiner, S.B., 1998. Prions. Proceedings of the National Academy of Sciences of the U.S.A 95, 13363–13383.

Rahal, E.A., Kazzi, N., Nassar, F.J., Matar, G.M., 2012. *Escherichia coli* O157:H7-Clinical aspects and novel treatment approaches. Frontiers in Cellular and Infection Microbiology 2, 138.

Ramasamy, I., 2004. The risk of accidental transmission of transmissible spongiform encephalopathy: identification of emerging issues. Public Health 118, 409–420.

Ramsay, M.A., 2006. John Snow, MD: anaesthetist to the Queen of England and pioneer epidemiologist. Proceedings (Baylor University Medical Center) 19, 24–28.

Reisen, W.K., Carroll, B.D., Takahashi, R., Fang, Y., Garcia, S., Martinez, V.M., Quiring, R., 2009. Repeated West Nile virus epidemic transmission in kern county, California, 2004-2007. Journal of Medical Entomology 46, 139–157.

Riley, L.W., 2014. Pandemic lineages of extraintestinal pathogenic *Escherichia coli*. Clinical Microbiology and Infection 20, 380–390.

Robinson, S.E., Brown, P.E., Wright, E.J., Hart, C.A., French, N.P., 2009. Quantifying within- and between-animal variation and uncertainty associated with counts of *Escherichia coli* O157 occurring in naturally infected cattle faeces. Journal of the Royal Society, Interface 6, 169–177.

Rodrigue, D.C., Mast, E.E., Greene, K.D., Davis, J.P., Hutchinson, M.A., Wells, J.G., Barrett, T.J., Griffin, P.M., 1995. A university outbreak of *Escherichia coli* O157:H7 infections associated with roast beef and an unusually benign clinical course. Journal of Infectious Diseases 172, 1122–1125.

Rodriguez, A., Autio, W.R., McLandsborough, L.A., 2007. Effects of inoculation level, material hydration, and stainless steel surface roughness on the transfer of listeria monocytogenes from inoculated bologna to stainless steel and high-density polyethylene. Journal of Food Protection 70, 1423–1428.

Rosef, O., Kapperud, G., 1983. House flies (*Musca domestica*) as possible vectors of *Campylobacter fetus* subsp. jejuni. Applied and Environmental Microbiology 45, 381–383.

Rossi, E.M., Scapin, D., Tondo, E.C., 2013. Survival and transfer of microorganisms from kitchen sponges to surfaces of stainless steel and polyethylene. Journal of Infection in Developing Countries 7, 229–234.

Sattar, S.A., Ijaz, M.K., Johnson-Lussenburg, C.M., Springthorpe, V.S., 1984. Effect of relative humidity on the airborne survival of rotavirus SA11. Applied and Environmental Microbiology 47, 879–881.

Schlundt, J., Toyofuku, H., Jansen, J., Herbst, S.A., 2004. Emerging food-borne zoonoses. Revue Scietifique et Technique 23, 513–533.

Schmid, D., Schandl, S., Pichler, A.M., Kornschober, C., Berghold, C., Beranek, A., Neubauer, G., Neuhold-Wassermann, M., Schwender, W., Klauber, A., Deutz, A., Pless, P., Allerberger, F., 2006. Salmonella Enteritidis phage type 21 outbreak in Austria, 2005. European Surveillance Bulletin 11, 67–69.

Schmidt, J.W., Arthur, T.M., Bosilevac, J.M., Kalchayanand, N., Wheeler, T.L., 2012. Detection of *Escherichia coli* O157:H7 and *Salmonella enterica* in air and droplets at three U.S. commercial beef processing plants. Journal of Food Protection 75, 2213–2218.

Schoder, D., Melzner, D., Schmalwieser, A., Zangana, A., Winter, P., Wagner, M., 2011. Important vectors for Listeria monocytogenes transmission at farm dairies manufacturing fresh sheep and goat cheese from raw milk. Journal of Food Protection 74, 919–924.

Sharps, C.P., Kotwal, G., Cannon, J.L., 2012. Human norovirus transfer to stainless steel and small fruits during handling. Journal of Food Protection 75, 1437–1446.

Sheen, S., 2008. Modeling surface transfer of Listeria monocytogenes on salami during slicing. Journal of Food Science 73, E304–E311.

Sheth, A.N., Wiersma, P., Atrubin, D., Dubey, V., Zink, D., Skinner, G., Doerr, F., Juliao, P., Gonzalez, G., Burnett, C., Drenzek, C., Shuler, C., Austin, J., Ellis, A., Maslanka, S., Sobel, J., 2008. International outbreak of severe botulism with prolonged toxemia caused by commercial carrot juice. Clinical Infectious Diseases 47, 1245–1251.

Shiode, N., Shiode, S., Rod-Thatcher, E., Rana, S., Vinten-Johansen, P., 2015. The mortality rates and the space-time patterns of John Snow's cholera epidemic map. International Journal of Health Geography 14, 21.

Sobel, J., 2005. Botulism. Clinical Infectious Diseases 41, 1167–1173.

Sobel, J., Tucker, N., Sulka, A., McLaughlin, J., Maslanka, S., 2004. Foodborne botulism in the United States, 1990–2000. Emerging Infectious Diseases 10, 1606–1611.

Soderstrom, A., Osterberg, P., Lindqvist, A., Jonsson, B., Lindberg, A., Blide Ulander, S., Welinder-Olsson, C., Lofdahl, S., Kaijser, B., De Jong, B., Kuhlmann-Berenzon, S., Boqvist, S., Eriksson, E., Szanto, E., Andersson, S., Allestam, G., Hedenstrom, I., Ledet Muller, L., Andersson, Y., 2008. A large *Escherichia coli* O157 outbreak in Sweden associated with locally produced lettuce. Foodborne Pathogens and Disease 5, 339–349.

Solano, R., Alseda, M., Godoy, P., Sanz, M., Bartolome, R., Manzanares-Laya, S., Dominguez, A., Cayla, J.A., 2014. Person-to-person transmission of norovirus resulting in an outbreak of acute gastroenteritis at a summer camp. European Journal of Gastroenterology and Hepatology 26, 1160–1166.

Solomon, E.B., Yaron, S., Matthews, K.R., 2002. Transmission of *Escherichia coli* O157:H7 from contaminated manure and irrigation water to lettuce plant tissue and its subsequent internalization. Applied and Environmental Microbiology 68, 397–400.

Soon, J.M., Chadd, S.A., Baines, R.N., 2011. *Escherichia coli* O157:H7 in beef cattle: on farm contamination and pre-slaughter control methods. Animal Health Research Reviews 12, 197–211.

Spurlock, A.T., Zottola, E.A., 1991. The survival of Listeria monocytogenes in aerosols. Journal of Food Protection 54, 910–912.

Steele, M., Odumeru, J., 2004. Irrigation water as source of foodborne pathogens on fruit and vegetables. Journal of Food Protection 67, 2839–2849.

Stein, R.A., 2011. Super-spreaders in infectious diseases. International Journal of Infectious Diseases 15, e510–513.

Stevens, M.P., Humphrey, T.J., Maskell, D.J., 2009. Molecular insights into farm animal and zoonotic Salmonella infections. Philosophical Transactions of the Royal Society of London. Series B Biological Sciences 364, 2709–2723.

Su, C., Brandt, L.J., 1995. *Escherichia coli* O157:H7 infection in humans. Annals of Internal Medicine 123, 698–714.

Synge, B.A., Chase-Topping, M.E., Hopkins, G.F., McKendrick, I.J., Thomson-Carter, F., Gray, D., Rusbridge, S.M., Munro, F.I., Foster, G., Gunn, G.J., 2003. Factors influencing the shedding of verocytotoxin-producing *Escherichia coli* O157 by beef suckler cows. Epidemiology and Infection 130, 301–312.

Tanzi, M.G., Gabay, M.P., 2002. Association between honey consumption and infant botulism. Pharmacotherapy 22, 1479–1483.

Taylor, D.M., 1999. Inactivation of prions by physical and chemical means. The Journal of Hospital Infection 43 (Suppl.), S69–S76.

Taylor, D.M., Woodgate, S.L., 2003. Rendering practices and inactivation of transmissible spongiform encephalopathy agents. Revue Scietifique et Technique 22, 297–310.

Teunis, P.F., Moe, C.L., Liu, P., Miller, S.E., Lindesmith, L., Baric, R.S., Le Pendu, J., Calderon, R.L., 2008. Norwalk virus: how infectious is it? Journal of Medical Virology 80, 1468–1476.

Thornley, C.N., Emslie, N.A., Sprott, T.W., Greening, G.E., Rapana, J.P., 2011. Recurring norovirus transmission on an airplane. Clinical Infectious Diseases 53, 515–520.

Todd, E.C., Greig, J.D., Bartleson, C.A., Michaels, B.S., 2007. Outbreaks where food workers have been implicated in the spread of foodborne disease. Part 3. Factors contributing to outbreaks and description of outbreak categories. Journal of Food Protection 70, 2199–2217.

Todd, E.C., Greig, J.D., Bartleson, C.A., Michaels, B.S., 2009. Outbreaks where food workers have been implicated in the spread of foodborne disease. Part 6. Transmission and survival of pathogens in the food processing and preparation environment. Journal of Food Protection 72, 202–219.

Tulodziecki, D., 2011. A case study in explanatory power: John Snow's conclusions about the pathology and transmission of cholera. Studies in History and Philosophy of Biological and Biomedical Sciences 42, 306–316.

Tung-Thompson, G., Libera, D.A., Koch, K.L., de Los Reyes 3rd, F.L., Jaykus, L.A., 2015. Aerosolization of a human norovirus surrogate, bacteriophage MS2, during simulated vomiting. PLoS One 10, e0134277.

Vally, H., Glass, K., Ford, L., Hall, G., Kirk, M.D., Shadbolt, C., Veitch, M., Fullerton, K.E., Musto, J., Becker, N., 2014. Proportion of illness acquired by foodborne transmission for nine enteric pathogens in Australia: an expert elicitation. Foodborne Pathogens and Disease 11, 727–733.

Van Kessel, J.A., Karns, J.S., Lombard, J.E., Kopral, C.A., 2011. Prevalence of *Salmonella enterica*, Listeria monocytogenes, and *Escherichia coli* virulence factors in bulk tank milk and in-line filters from U.S. dairies. Journal of Food Protection 74, 759–768.

Van Renterghem, B., Huysman, F., Rygole, R., Verstraete, W., 1991. Detection and prevalence of Listeria monocytogenes in the agricultural ecosystem. Journal of Applied Bacteriology 71, 211–217.

Vega, E., Barclay, L., Gregoricus, N., Shirley, S.H., Lee, D., Vinje, J., 2014. Genotypic and epidemiologic trends of norovirus outbreaks in the United States, 2009 to 2013. Journal of Clinical Microbiology 52, 147–155.

Vega, E., Barclay, L., Gregoricus, N., Williams, K., Lee, D., Vinje, J., 2011. Novel surveillance network for norovirus gastroenteritis outbreaks, United States. Emerging Infectious Diseases 17, 1389–1395.

Verhaelen, K., Bouwknegt, M., Carratala, A., Lodder-Verschoor, F., Diez-Valcarce, M., Rodriguez-Lazaro, D., de Roda Husman, A.M., Rutjes, S.A., 2013. Virus transfer proportions between gloved fingertips, soft berries, and lettuce, and associated health risks. International Journal of Food Microbiology 166, 419–425.

Vivant, A.L., Garmyn, D., Piveteau, P., 2013. Listeria monocytogenes, a down-to-earth pathogen. Frontiers in Cellular and Infection Microbiology 3, 87.

Vogt, R.L., Dippold, L., 2005. *Escherichia coli* O157:H7 outbreak associated with consumption of ground beef, June–July 2002. Public Health Rep 120, 174–178.

Volkova, V.V., Howey, R., Savill, N.J., Woolhouse, M.E., 2010. Sheep movement networks and the transmission of infectious diseases. PLoS One 5, e11185.

Vorst, K.L., Todd, E.C., Rysert, E.T., 2006. Transfer of Listeria monocytogenes during mechanical slicing of Turkey breast, bologna, and salami. Journal of Food Protection 69, 619–626.

Vriesekoop, F., Russell, C., Alvarez-Mayorga, B., Aidoo, K., Yuan, Q., Scannell, A., Beumer, R.R., Jiang, X., Barro, N., Otokunefor, K., Smith-Arnold, C., Heap, A., Chen, J., Iturriage, M.H., Hazeleger, W., DeSlandes, J., Kinley, B., Wilson, K., Menz, G., 2010. Dirty money: an investigation into the hygiene status of some of the world's currencies as obtained from food outlets. Foodborne Pathogens and Disease 7, 1497–1502.

Vriesekoop, F., Shaw, R., 2010. The Australian bush fly (Musca vetustissima) as a potential vector in the transmission of foodborne pathogens at outdoor eateries. Foodborne Pathogens and Disease 7, 275–279.

Vugia, D.J., Mase, S.R., Cole, B., Stiles, J., Rosenberg, J., Velasquez, L., Radner, A., Inami, G., 2009. Botulism from drinking pruno. Emerging Infectious Diseases 15, 69–71.

Wagner, E.G., Lanoix, J.N., 1958. Excreta Disposal for Rural Areas and Small Communities. WHO Monograph series No 39. WHO, Geneva.

Walters, M.S., Sreenivasan, N., Person, B., Shew, M., Wheeler, D., Hall, J., Bogdanow, L., Leniek, K., Rao, A., 2015. A qualitative Inquiry about pruno, an illicit alcoholic beverage linked to botulism outbreaks in United States prisons. American Journal of Public Health 105, 2256–2261.

Wang, Y.C., Chang, Y.C., Chuang, H.L., Chiu, C.C., Yeh, K.S., Chang, C.C., Hsuan, S.L., Lin, W.H., Chen, T.H., 2011. Transmission of Salmonella between swine farms by the housefly (*Musca domestica*). Journal of Food Protection 74, 1012–1016.

Wendel, A.M., Johnson, D.H., Sharapov, U., Grant, J., Archer, J.R., Monson, T., Koschmann, C., Davis, J.P., 2009. Multistate outbreak of *Escherichia coli* O157:H7 infection associated with consumption of packaged spinach, August–September 2006: the Wisconsin investigation. Clinical Infectious Diseases 48, 1079–1086.

Widdowson, M.A., Sulka, A., Bulens, S.N., Beard, R.S., Chaves, S.S., Hammond, R., Salehi, E.D., Swanson, E., Totaro, J., Woron, R., Mead, P.S., Bresee, J.S., Monroe, S.S., Glass, R.I., 2005. Norovirus and foodborne disease, United States, 1991–2000. Emerging Infectious Diseases 11, 95–102.

Williams, K.J., Ward, M.P., Dhungyel, O.P., Hall, E.J., 2014. Risk factors for *Escherichia coli* O157 shedding and super-shedding by dairy heifers at pasture. Epidemiology and Infection 1–12.

Woolhouse, M.E., Coen, P., Matthews, L., Foster, J.D., Elsen, J.M., Lewis, R.M., Haydon, D.T., Hunter, N., 2001. A centuries-long epidemic of scrapie in British sheep? Trends in Microbiology 9, 67–70.

Wu, H.W., Ito, A., Ai, L., Zhou, X.N., Acosta, L.P., Lee Willingham Iii, A., 2016. Cysticercosis/taeniasis endemicity in Southeast Asia: current status and control measures. Acta Tropica. pii: S0001-706X(16)30013-4. (Epub ahead of print).

Yoo, J.R., Heo, S.T., Lee, K.H., Kim, Y.R., Yoo, S.J., 2015. Foodborne outbreak of human brucellosis caused by ingested raw materials of fetal calf on Jeju Island. The American Journal of Tropical Medicine and Hygiene 92, 267–269.