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FOOD-BORNE PATHOGENS OF RECENT CONCERN

Michael P. Doyle

The Food Research Institute, University of Wisconsin, Madison, Wisconsin 53706

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INTRODUCTION

Food-associated pathogens are a frequent cause of disease in the United States. Estimates indicate that the annual number of food-borne disease cases is in the millions (53). Epidemiologic data prepared by the Centers for Disease Control indicate that the cause of more than half of reported food-related outbreaks is not identified (Table 1), usually for lack of performing the appropriate laboratory tests (59). One reason for not doing the necessary tests is that the responsible agent was not recognized as a pathogen or a significant pathogen at the time, hence laboratory tests were not available or were not routinely performed. Several outbreaks reported as being caused by agents of unknown etiology were likely due to pathogens not previously recognized as agents of food-borne disease. Within the last decade, five bacterial pathogens have emerged as newly recognized or important agents of food-borne disease. These include *Yersinia enterocolitica, Vibrio cholerae, Campylobacter jejuni, Listeria monocytogenes,* and *Escherichia coli* O157:H7.

YERSINIA ENTEROCOLITICA

Background

Y. enterocolitica was first recognized as a human pathogen in 1939 (114), but its significance as a pathogen was not greatly appreciated until the mid 1970s. As a result of greater awareness of the organism and more laboratories testing for its presence in enteric specimens (86), there has been a dramatic increase in the number of reported cases of *Y. enterocolitica* infection throughout the world. For example, on a worldwide basis only 23 cases of *Y. enterocolitica*

Table 1 Number of food-borne disease outbreaks of confirmed and unknown etiology reported by the Centers for Disease Control (24–26, 29)

Year	Confirmed	Unknown	Total	Percentage unknown
1970	139	227	366	62.0
1971	94	226	320	70.6
1972	136	165	301	54.8
1973	127	180	307	58.6
1974	201	255	456	55.9
1975	191	306	497	61.6
1976	132	306	438	69.9
1977	157	279	436	64.0
1978	154	327	481	68.0
1979	172	287	460	62.4
1980	221	391	612	63.9
1981	250	318	568	56.0

infection were recorded in 1966, 642 in 1970, over 1000 in 1972, and over 4000 in 1974 (85, 86, 134). A 1979 report indicated *Y. enterocolitica* infections to be the most frequent type of enteric infection in Denmark, with about 200,000 cases occurring per year (76).

An interesting yet inexplicable relationship exists among the serotypes of *Y. enterocolitica* associated with human infections in different regions of the world. The predominant serotype isolated from patients in the United States is O:8, whereas serotypes O:3 and to a lesser extent O:9 are predominantly isolated in Canada, Europe, Japan, and South Africa (18, 130). Also, unlike most other enteric pathogens of which the greatest incidence of infections occur during the summer months, infections due to *Y. enterocolitica* are most prevalent during the autumn and winter months (130). The organism is responsible for appreciable morbidity in the cooler regions of Europe and North America. For instance, in parts of the Federal Republic of Germany and Canada, *Y. enterocolitica* is comparable to *Salmonella* and surpasses *Shigella* as a cause of acute enteritis (81, 147).

Outbreaks

A 1976 outbreak in Upstate New York provided the first conclusive evidence linking *Y. enterocolitica* to food-borne transmission (8). In this outbreak a yersiniosis-like illness was reported by more than 220 persons, primarily schoolchildren, of whom 36 were hospitalized and 16 had appendectomies. The responsible organism, *Y. enterocolitica* serotype O:8, was isolated from chocolate milk epidemiologically implicated as the vehicle of transmission.

Since then, several additional food-associated outbreaks of *Y. enterocolitica* infection have been reported. Three of these are of particular interest, as many individuals were infected in each.

One outbreak occurred at a coeducational summer camp in New York State in July 1981 (87, 118). Gastrointestinal disorders were observed in 239 campers and staff members, and 5 of 7 hospitalized patients had appendectomies before the illness was recognized as yersiniosis. *Y. enterocolitica* serotype O:8 was isolated from patients and from dissolved powdered milk and turkey chow mein, which were epidemiologically linked as vehicles of transmission. Information obtained during the investigation suggested that *Yersinia* was introduced by a foodhandler during food preparation (87).

Another outbreak occurred in December 1981 and January 1982 among 87 persons in Washington State (4, 91). The illness was associated with ingestion of tofu packed in untreated spring water. *Y. enterocolitica* serotypes O:8 and O:Tacoma were isolated from hospitalized patients, and serotype O:8 strains were isolated from the processing plant's water supply, hence incriminating the water used in the processing of tofu as the source of infection (4).

The largest outbreak of yersiniosis ever reported in the United States occur-

red during the summer of 1982 in Tennessee, Arkansas, and Mississippi (132). One hundred seventy-two culture-positive *Y. enterocolitica* infections were identified. Drinking pasteurized milk processed by a specific plant was epidemiologically associated with illness. Based on the widespread distribution of milk processed by the implicated plant and the calculated attack rates of individuals who consumed the milk and became ill, it was estimated that several hundred cases actually occurred.

Characteristics of Disease

The most frequently encountered manifestation of *Y. enterocolitica* infection is enterocolitis, which occurs primarily in infants and young children (18). The disease is characterized generally by diarrhea (which may last for several weeks), fever, and severe abdominal pain in the right lower quadrant, which is highly suggestive of acute appendicitis and often leads to surgical intervention. Apart from enterocolitis, *Y. enterocolitica* may cause mesenteric adenitis, hepatosplenic abscesses, septicemia, erythema nodosum, and arthritis.

Reservoirs

Y. enterocolitica is commonly found in the environment (69, 92, 117) and in the alimentary tract of animals (3, 32, 69, 117, 131); however, with the exception of cultures obtained from swine (41–43, 98, 99, 112, 135, 138, 149), most isolates are types not associated with human infection. Swine has been implicated as a major reservoir of pathogenic *Y. enterocolitica,* because the serotypes commonly associated with human infection (i.e. O:3; O:5,27; O:8; and O:9) have been isolated from the tongue, throat, or feces of healthy pigs (3, 41–43, 98, 99, 112, 131, 135, 138, 149). Some European investigators concluded that *Y. enterocolitica* O:3 is a normal inhabitant of the oral cavity of swine and that swine may play a major role in the epidemiology of human infections (98, 138).

Significance as a Food-Borne Pathogen

Unlike other enteropathogens, *Y. enterocolitica* is a psychrotroph that can grow in foods at refrigeration temperature (51, 52, 126). Hence, cold storage, the traditional method of preventing growth and toxin formation by food-poisoning bacteria, is ineffective in controlling the growth of *Y. enterocolitica* in foods. Consequently, measures must be taken to prevent contamination of foods or treat foods with heat or some means of microbial inactivation to destroy *Y. enterocolitica*. Fortunately, with the exception of pork (14, 42, 111), the types of *Y. enterocolitica* primarily associated with human infection are not prevalent in foods; hence it is likely that pathogenic strains are seldom present in most foods. This may explain why food-associated outbreaks of *Y. enterocolitica* gastroenteritis are not more common.

VIBRIO CHOLERAE

Background

V. cholerae O–Group 1 (O1) has long been a scourge of the Eastern Hemisphere, where seven cholera pandemics have occurred since the early 1800s. Although the disease entered the United States and caused many deaths during the 19th century (110), only one culture-proven case of domestically acquired *V. cholerae* O1 infection was identified between 1911 (2) and 1978; this was an unexplained case discovered in Texas in 1973 (140).

Another type of *V. cholerae,* designated *V. cholerae* non O–Group 1 (non-O1) because it does not agglutinate in cholera O–Group 1 antiserum (143), has also been recognized as a pathogen (60); however, this organism does not pose the same pandemic threat as *V. cholerae* O1. Since 1965, non-O1 *V. cholerae* has been associated with several outbreaks and sporadic cases of gastrointestinal illness (60, 143), with most cases reported in the Eastern Hemisphere.

Outbreaks

Concern of *V. cholerae* O1 in the United States was resurrected in 1978 following an outbreak of cholera in Louisiana (9). Eleven cases of *V. cholerae* O1 infection were identified. The vehicle of transmission was cooked crabs caught in Louisiana marshes. The crabs were well-cooked by traditional criteria, but may not have been heated properly or sufficiently to inactivate *V. cholerae*.

Three years later, the largest outbreak of cholera in the United States in over a century occurred on a floating Texas oil rig (64). Sixteen men were infected by *V. cholerae* O1. Infection was associated with eating rice on the oil rig on a particular day when an open valve permitted the rig's drinking water system to be contaminated by canal water containing sewage discharged from the rig. The rice had been rinsed in the contaminated water after cooking.

The first outbreak of non-O1 *V. cholerae* gastroenteritis in the United States occurred in Florida in 1979 (146). A cluster of five confirmed cases was identified, and illness was associated with eating raw oysters. Furthermore, non-O1 *V. cholerae* was isolated from oysters and water samples taken from areas where ill persons had obtained their oysters.

Characteristics of Disease

Both *V. cholerae* O1 and non-O1 *V. cholerae* produce a gastrointestinal syndrome characterized by diarrhea, nausea, and vomiting, but illness caused by *V. cholerae* O1 is typically more severe, with a patient often experiencing severe dehydration and electrolyte depletion through a stool loss of greater than one liter per hour. Because of the tremendous loss of fluids, patients may die quickly unless appropriately treated by fluid replacement therapy.

Reservoirs

Man is the major source of *V. cholerae* O1 infection and the disease is usually spread by symptomatic or asymptomatic persons whose excrements contaminate food and water (143). Vibrios are likely transported to other areas by water contaminated by infected feces. Recent surveys of US coastal waters documented the isolation of *V. cholerae* O1 and non-O1 *V. cholerae* from aquatic environments, including California, Florida, Louisiana, and Maryland (31, 35, 58, 68, 71, 80, 89, 106).

Laboratory studies reveal that *V. cholerae* requires a salinity of at least 0.01% to survive in water beyond 24 hours (84). This may explain why the organism is primarily associated with marine environments. *V. cholerae* has also been isolated from oysters (57, 58, 80, 136), crabs (9, 33, 58), and shrimp (9), which confirms that shellfish can be a source of human infection as occurred in recent outbreaks in the United States. Results of environmental surveys indicate that non-O1 *V. cholerae* is much more prevalent than *V. cholerae* O1. Because of its widespread distribution in sewage, sewage-contaminated surface water, estuarine waters, and seafoods, non-O1 *V. cholerae* is thought to be free-living in the environment (143).

Significance as a Food-Borne Pathogen

Symptoms of cholera, especially those produced by *V. cholerae* O1, are often life-threatening and may, if not quickly and properly treated, result in death. The fatality rate may approach 50% in untreated cases (56). Shellfish, primarily oysters and crabs, are the predominant foods that carry *V. cholerae* and these foods have been identified as vehicles transmitting the organism in cholera outbreaks. Recent outbreaks in the United States primarily resulted from eating raw or improperly cooked seafood containing *V. cholerae*. Shellfish should be thoroughly cooked before eating to eliminate the potential for a cholera infection.

CAMPYLOBACTER JEJUNI

Background

The disease potential of microaerophilic campylobacters (formerly *Vibrio fetus*) was first recognized in 1909 when such organisms were associated with abortion in cattle and sheep (82). However, it was not until this past decade, when simplified procedures became available to isolate such organisms from humans (15, 34, 120), that *C. jejuni* emerged as a leading cause of acute gastroenteritis in humans throughout the world (14, 17, 22, 97, 105, 120, 124). A recent 15-month study at eight hospitals in different parts of the United States revealed that *C. jejuni* was isolated from fecal specimens of patients more often than *Salmonella* and *Shigella* combined (14).

Outbreaks

Food is a likely source of many campylobacter infections and has been identified as the vehicle of transmission of many outbreaks of campylobacter enteritis. Raw milk has been associated most commonly with food-related outbreaks (12, 27, 28, 30, 65, 83, 100, 101, 109, 123, 133) and many of these have been quite large, with as many as 2500 individuals infected (65). Undercooked chicken (19), processed turkey (11), cake icing (13), raw clams (11), and raw hamburger (95) have also been implicated as vehicles of transmission in outbreaks of campylobacter enteritis. All outbreaks have been associated with raw or inadequately cooked foods of animal origin or likely contamination of food by a food handler.

Characteristics of Disease

Symptoms and signs of campylobacter enteritis are not so distinctive that a physician can easily differentiate them from illness caused by other enteric pathogens (10). The features of *C. jejuni* infection vary from a brief insignificant enteritis to an enterocolitis with abdominal pain and profuse diarrhea. The predominant symptoms of individuals whose illness is sufficiently severe to seek medical attention are diarrhea, abdominal pain, malaise, fever, nausea, and vomiting (10). In severe cases, grossly bloody stools are common, and many patients have at least one day with eight or more bowel movements. Most patients recover in less than a week, but 20% may have a relapse or a prolonged or severe illness (15).

Reservoirs

C. jejuni is a commensal in the intestinal flora of many mammalian and avian species. In mammalian studies, the organism has been isolated from feces of healthy swine (90, 94, 102, 125, 128, 129), cattle (38, 90, 102, 108, 129, 133), sheep (102, 125), goats (102), dogs (21, 46, 102, 129, 148), cats (20, 46, 129), rabbits (102, 139), rodents (44, 47, 139), and monkeys (79, 88). *C. jejuni* has also been isolated from feces of 15–100% of chickens, turkeys, or wild birds tested (36, 48, 77, 78, 90, 96, 103, 119, 122, 129, 142), with greater than 10^6 *C. jejuni* often present per gram of fecal specimen (48, 96, 142). After slaughter and processing, more than 50% of poultry carcasses and edible parts are often contaminated with 10^2 to 10^3 *C. jejuni* per gram or organ (96, 142).

Significance as a Food-Borne Pathogen

C. jejuni is the leading cause of acute bacterial gastroenteritis in the United States (14), and hence constitutes a major concern. Food, primarily of animal origin, is the likely source of many campylobacter infections and has been associated with many outbreaks of campylobacter enteritis. Fecal excrement from healthy, wild and domestic animals is likely the major source of *C. jejuni* and the primary vehicle for transmitting the organism to food.

Interestingly, *C. jejuni* is not a hardy survivor outside of its host's environment. The organism is not likely to grow in foods (that remain edible) or on food processing equipment surfaces because the bacterium (*a*) will not grow below 30°C (38), (*b*) requires microaerobic (5% oxygen optimal) and capnophilic (10% carbon dioxide optimal) conditions for growth (121), and (*c*) grows slowly (even under ideal conditions its doubling time is about 1 hour; 37). Furthermore, *C. jejuni* does not survive well in foods. It is sensitive to drying (40), 21% oxygen (74, 121), storage at 25°C (38), acidic conditions (37), sodium chloride (39), and heat (37, 73).

Although *C. jejuni* is not likely to grow or survive well in foods, foods of animal origin may be contaminated initially with large numbers of campylobacters through fecal contamination. Often 45–85% of retail-ready poultry carcasses, parts, and giblets are *C. jejuni* positive (48, 77, 96, 119, 142). Additionally, results of studies done to estimate the number of *C. jejuni* that must be ingested to produce human illness suggest that consuming only 500 cells can produce sickness (7, 54, 107). The organism's apparent high degree of virulence, as indicated by its apparent low infectious dose, and its widespread prevalence in animal-derived foods are important factors in explaining why this sensitive bacterium is a leading cause of human enteric infections.

LISTERIA MONOCYTOGENES

Background

L. monocytogenes was first reported as a cause of human disease in 1929 (93), although infection caused by this bacterium had been recognized as a significant problem in animals since 1911 (61). In humans, the primary manifestations of listeric infections are meningitis, abortion, and perinatal septicemia, with immunocompromised individuals, pregnant women, and infants primarily at risk (1). Listeriosis is not a reportable disease, hence precise data regarding the true incidence of disease are not available. However, available data indicate that *L. monocytogenes* is infrequently identified as a human pathogen. For example, during 1978 only 68 cases of meningitis due to *L. monocytogenes* were reported to the Centers for Disease Control from 38 states participating in the national bacterial meningitis and meningococcemia surveillance study, and 165 strains from patients were received for confirmation of identification and serotyping (1). Recently, two food-associated outbreaks of listeriosis in North America were reported.

Outbreaks

In 1981, a large outbreak of both adult (7 cases) and perinatal (34 cases) infection due to *L. monocytogenes* serotype 4b occurred in the Maritime Provinces of Canada (113). Epidemiologic investigation identified contaminated coleslaw as the probable vehicle of transmission. Coleslaw obtained

from the refrigerator of one patient grew *L. monocytogenes* of the same serotype (4b) as had been isolated from the patient's blood. The coleslaw was prepared by a regional producer who had obtained cabbage from a farm known to have had cases of ovine listeriosis. This cabbage was grown in fields fertilized with both composted and raw manure from the flock of sheep. After the cabbage was harvested, it was held in a large cold-storage shed during the winter and early spring, until it was distributed to wholesalers. It was suggested that subjecting cabbage to cold storage before use may have allowed a small initial inoculum of *L. monocytogenes* to proliferate.

A more recent outbreak occurred in Massachusetts in 1983 in which 49 patients were hospitalized with septicemia or meningitis caused by *L. monocytogenes* (45). Seven patients were newborns and 42 were adults, all of whom were taking medications causing immunosuppression or had underlying illness. Fourteen (29%) patients died. Most (32 of 40) of the isolates available for testing were serotype 4b. Epidemiologic studies revealed that illness was associated with consumption of a specific brand of pasteurized milk. Furthermore, *L. monocytogenes* serotype 4b was isolated from milk in the bulk tank of one of the farms supplying raw milk to the incriminated processing plant.

Characteristics of Disease

A wide variety of clinical manifestations have been described for *L. monocytogenes* infections (49), but most infections are accounted for by (*a*) neonatal sepsis or meningitis, (*b*) sepsis or meningitis in the immunocompromised patient, and (*c*) puerperal sepsis or nonspecific "flu-like" illness during pregnancy usually resulting in premature delivery of stillborn or acutely ill infants (116). Newborns constitute the largest single group of identified infections. Before the development of antibiotics, mortality was very high, to 70% or more, among patients with listeric meningitis, and those who survived were often left with permanent brain damage (49). Today the mortality rate for listeriosis is on the order of about 30%, with mortality occurring most frequently among newborns and patients over 70 years of age.

Reservoirs

Although *L. monocytogenes* is infrequently associated with human infection, the organism is widely distributed in nature (16). Many healthy humans are carriers of this pathogen (66, 67). Studies by Schlech et al (113) indicate that fecal carriage may be present in 5% of the general population. Apparently healthy intestinal carriers also have been identified among cattle (55, 66), poultry (75, 115), swine (75), and sheep (50) populations. *L. monocytogenes* has been isolated from at least 2 mammalian and 22 avian species, including domesticated animals, house pets, and zoo, laboratory, and wild animals (23). Investigators (55, 66) reported a greater than 10% incidence of fecal carriers among healthy cattle.

The organism is occasionally isolated from human milk (62). In cattle, excretion of *L. monocytogenes* in milk is usually intermittent but may persist for periods exceeding a single lactation. Many such cows do not have mastitis and the milk they excrete is apparently normal (62). One study revealed that 13 of 36 cows whose sera were positive for *Listeria* had *Listeria* in their milk but were apparently healthy with no clinical signs of listeric infection (137). However, most often *Listeria*-contaminated milk is from mastitic cows.

Recovery of *L. monocytogenes* from decaying moist vegetation prompted Welshimer & Donker-Voet (145) to suggest that the organism has a saprophytic existence wherein the plant-soil environment may serve as a reservoir. Rather than attribute the presence of *Listeria* in nature solely to past contamination with animal feces, they suggest that the organism may be free-living on plants and soil.

Significance as a Food-Borne Pathogen

The brain-damaging and often fatal consequences of listeric infections in high-risk groups are of serious concern. This concern is compounded by the fact that *L. monocytogenes* purportedly may survive the pasteurization treatment given milk. Available data indicate that the organism is unusually heat resistant when compared to the thermal resistance of other vegetative bacteria. Bearns & Girard (5) found that heating 5×10^4 *L. monocytogenes* per milliliter of milk at 61.7°C for 35 minutes did not completely kill all *Listeria*. When the heat-treated milk was held at 22°C for 48 hours, the survivors multiplied and reached numbers of 10^8 per milliliter, without producing any grossly detectable changes or suspicious odors in the milk. According to current US regulations, such a heat treatment should be sufficient to pasteurize milk, thereby destroying all non-spore-forming pathogenic bacteria.

Additionally, *L. monocytogenes* is a psychrotroph that can grow at refrigeration temperature (72, 144). If indeed *Listeria* can survive the time-temperature treatment currently used for pasteurizing milk and subsequently grow to large numbers during refrigerated storage, then, depending on the organism's prevalence in milk, an important segment of the milk-drinking population may be at considerable risk. In light of the recent outbreak that was associated with pasteurized milk, additional studies are needed to more fully assess the thermal resistance of *L. monocytogenes* in milk.

ESCHERICHIA COLI O157:H7

Background

Before 1982, *E. coli* O157:H7 was identified only once in the United States at the Centers for Disease Control (104). The organism was isolated in 1975 from a patient during an acute illness with severe abdominal cramps followed by grossly bloody diarrhea. In Canada, in screening *E. coli* strains isolated from

diarrheal patients for enterotoxigenicity and cytotoxicity, the Laboratory Centre for Disease Control identified six cytotoxic isolates of *E. coli* O157:H7 during 1978–1982 (63). These data from sporadic cases of gastroenteritis suggested that this organism may be a human pathogen, but these few reports were not sufficient to conclusively identify *E. coli* O157:H7 as a cause of enteric disease. It was not until 1982, following two food-associated outbreaks of an unusual gastrointestinal illness, that epidemiologists were able to firmly link *E. coli* O157:H7 with a clinically distinctive gastrointestinal disorder (104).

Outbreaks

In the first half of 1982, two outbreaks of gastroenteritis characterized by sudden onset of severe abdominal cramps and grossly bloody diarrhea occurred in Oregon and Michigan (104): 26 cases were confirmed in Oregon and 21 cases in Michigan. Both outbreaks were epidemiologically linked to eating ground-beef sandwiches prepared at restaurants belonging to the same chain. Furthermore, *E. coli* O157:H7 was isolated from a raw ground-beef patty from the same lot as that used at the Michigan restaurants during the outbreak (141).

A third outbreak of *E. coli* O157:H7 infection occurred in 1982 and involved 31 residents in a home for the aged in Ontario, Canada (127). It was suggested that the cause of this outbreak was contaminated food because food was the only common factor. However, no specific food was implicated as the vehicle of transmission.

Characteristics of Disease

Symptoms of *E. coli* O157:H7 infection typically begin with the sudden onset of severe abdominal cramps, which are followed by the development of watery then grossly bloody diarrhea, described as "all blood and no stool" (104). Patients experience no fever or low-grade fever and some nausea. Barium enema radiography of several patients demonstrated marked submucosal edema with spasm and a "thumb-printing" pattern in the ascending and transverse colon, and sigmoidoscopy revealed moderately hyperemic mucosa; hence the illness has been termed hemorrhagic colitis.

More recently, *E. coli* O157:H7 was associated with hemolytic uremic syndrome, the leading cause of acute renal failure in childhood (70). Patients experience substantial morbidity, often requiring dialysis and blood transfusions, and may develop central nervous system disease characterized by frequent seizures and prolonged coma. Death may result.

Reservoirs

Although not confirmed, circumstantial information suggests that cattle may be a source of *E. coli* O157:H7. The organism has been isolated from a raw ground-beef patty (141), and ground-beef sandwiches were epidemiologically implicated as vehicles transmitting the pathogen in two outbreaks.

Inoculation studies by Beery et al (6) revealed that *E. coli* O157:H7 can colonize chicken cecae; thus chickens may serve as a host and possibly a reservoir for the organism. Because methods are not yet available to isolate *E. coli* O157:H7 selectively from foods and environmental sources, little is known about the organism's prevalence and habitat in nature.

Significance as a Food-Borne Pathogen

The types of illness associated with *E. coli* O157:H7 are indeed serious and appropriate measures should be taken to control its presence in foods. However, many questions must be answered before the significance of this organism as a food-borne pathogen can be accurately assessed. The immediate need is to identify the source(s) and prevalence of the organism. To accomplish this, an effective and sensitive procedure is needed to detect and isolate *E. coli* O157:H7 from foods. If cattle are a source of the organism, improvements are needed in handling, slaughtering, and processing procedures to prevent contamination of meat. An immediate solution is to thoroughly cook meat and avoid recontaminating cooked meat by contact with raw meat. Cross-contamination through food handlers should also be prevented.

CONCLUDING REMARKS

The brilliant detective efforts of epidemiologists and microbiologists in the past decade uncovered several food-associated pathogens of public health concern. *C. jejuni* is now recognized as the most common cause of acute bacterial gastroenteritis in humans in the United States. Although not frequently associated with human infection, *V. cholerae, L. monocytogenes,* and *E. coli* O157:H7 can produce life-threatening illnesses if patients are not promptly and properly treated. The distressing appendicitis-like symptoms of *Y. enterocolitica* are serious because they often lead to unnecessary appendectomies. Hence, the recognition of these bacteria as food-associated pathogens is indeed significant, as appropriate measures to control these organisms in foods can ultimately be developed and applied to prevent future incidents.

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