FRI BRIEFINGS

Human Illness Caused by *E. coli* O157:H7 from Food and Non-food Sources

M. Ellin Doyle[1*], John Archer[2], Charles W. Kaspar[1], and Ronald Weiss[1]

[1]Food Research Institute, University of Wisconsin–Madison, Madison, WI 53706

[2]Wisconsin Division of Public Health, Bureau of Communicable Diseases and Preparedness, Communicable Disease Epidemiology Section, Madison, WI 53702

Contents

*Corresponding author: M. Ellin Doyle, Ph.D., medoyle@wisc.edu
http://fri.wisc.edu/docs/pdf/FRIBrief_EcoliO157H7humanillness.pdf

Food Research Institute, UW–Madison, August/October 2006
Funded in part by the American Meat Institute Foundation

INTRODUCTION

E. coli O157:H7 was first identified as a possible human pathogen in 1975 in a California patient with bloody diarrhea and was first associated with a foodborne (ground beef) outbreak of disease in 1982 (*299;353*). This serotype (defined by its O and H surface antigens) and some non-O157 serotypes of *E. coli* produce verocytotoxins, also called Shiga-like toxins because of their similarity to toxins produced by *Shigella dysenteriae*. These *E. coli* are called VTEC (verocytotoxin-producing *E. coli*), STEC (Shiga-toxin producing *E. coli*), and also EHEC (enterohemorrhagic *E. coli*) because of the symptoms they produce. Serotypes of VTEC bacteria may include different strains that differ in some virulence factors or other characteristics such as motility and sorbitol fermentation. It has been estimated that *E. coli* O157:H7 causes about 73,500 cases of illness and 60 deaths annually in the U.S. while non-O157 VTEC serotypes cause about 37,000 cases annually (*73;254*).

Epidemiological studies of foodborne outbreaks have indicated that fewer than 40 cells of *E. coli* O157:H7 can cause illness in some people (*162;328;335*). Waterborne outbreaks, involving both drinking water and pools and lakes, also indicate a very low infectious dose because of the great dilution factor in large quantities of water. Depending on their age, immune status, general health, dose of bacteria, and virulence factors in the bacteria, persons exposed to VTEC may experience mild diarrhea, severe bloody diarrhea, hemorrhagic colitis, or hemolytic uremic syndrome (HUS) with kidney failure. Some cases, usually among young children and older people, are fatal. Children <5.5 years of age are more likely to develop HUS and some studies have indicated that antibiotics or antimotility agents increase risk for HUS (*53;101*). Other recent studies indicated that correlations of HUS with antibiotic use were not very strong (*304;339*). Patients with higher temperatures, higher white blood cell counts, and a longer periods of diarrhea appear to be more likely to develop HUS (*181;339*).

Although this literature review is focused on epidemiology of *E. coli* O157:H7, which is the primary serotype causing outbreaks of VTEC in the U.S., more than 200 different serotypes of *E. coli* produce shiga-like toxins and many have been implicated in outbreaks and cases of HUS in the U.S. and other countries (*73;196;251*). Verotoxin-producing *E. coli* can be sorted into 4 clonal groups according to different virulence factors and other characteristics that are encoded by genes on their chromosomes, plasmids, and phages. Many of these genes, including those encoding Shiga toxins, were apparently acquired from other organisms while some other functions, such as motility and sorbitol fermentation, were lost during the evolution of *E. coli* O157:H7 from avirulent ancestors. [Some recent European isolates of *E. coli* O157:H7 have been found to ferment sorbitol (*43*).] Approximately 25% of the *E. coli* O157:H7 genome is derived from bacteriophages indicating that these viruses were very important in the horizontal transfer of genes (*80;213; 344;355*). VTEC bacteria continue to evolve even during the course of infections as strains isolated from stool samples early in infection sometimes differ from those isolated later (*195;240;355*). Analyses by CDC indicate that 10.8% of *E. coli* O157:H7 tested in 2003 have become resistant to one or more antibiotics (*85*).

Non-O157:H7 strains vary in their ability to cause severe human illness and outbreaks but there is evidence that many are associated with cattle and other ruminants as is *E. coli* O157:H7 (*295*). During 2004, 110 non-O157 VTEC infections were identified in the ten states surveyed by FoodNet with serogroups O111, O103, and O26 most frequently detected (*84*). Outbreaks in the U.S. include: a 1994 outbreak in Montana associated with milk (serogroup O104) (*245*), a 1999 outbreak in Connecticut associated with lake water (serogroup O121) (*235*), a 1999 outbreak in Texas associated with a salad (serogroup O111) (*72*), a 2000 outbreak in Washington associated with punch (serogroup O103), a 2001 outbreak in South Dakota in a day care (serogroup O111), and a 2001 outbreak in Minnesota associated with lake water (serogroup O26) (*243*). In Australia, *E. coli* O157:H7 is not as commonly isolated in cases of bloody diarrhea and HUS as are other serotypes. Serogroup O111 was identified in a 1995 outbreak associated with sausage (*271*) and a 2003 outbreak thought to result from person to person transfer (*106*). Other Australian outbreaks most likely resulting from contact with cattle were caused by serotype O86:H27 (*247*) and O48:H21 (*154*). Non-O157 VTEC have also caused outbreaks in several European countries and Argentina (*10;155;192;257;310;354*).

EPIDEMIOLOGY OF *E. coli* O157:H7

Outbreak Data

E. coli O157:H7 has been isolated from ill people around the world. It tends to be reported more often from more developed countries but this may be an artifact caused by the paucity of sophisticated diagnostic laboratories in developing countries. Appendix 1 lists, chronologically, published details of 207 reported outbreaks of *E. coli* O157:H7.

Corresponding author: M. Ellin Doyle, Ph.D., medoyle@wisc.edu

Funded in part by the American Meat Institute Foundation

Undoubtedly, other outbreaks have occurred but details have not been published in accessible journals. A review of data on outbreaks of infectious intestinal disease in the UK from 1992 to 2003 pointed out that there were 1763 outbreaks reported to authorities but only 55 of these were presented in peer-reviewed journals. Of 45 VTEC outbreaks reported to National Surveillance Program, only 7 were described in the literature (*261*).

FoodNet data indicate that *E. coli* O157:H7 causes significantly more cases of sporadic infections than cases linked to an outbreak (*84*). For example, in 2004, only 9% of 402 confirmed cases of infection with *E. coli* O157:H7 were associated with outbreaks (www.cdc.gov/foodnet/annual/2004/report.pdf). Sporadic infections appear to be associated with some of the same factors that cause outbreaks: undercooked hamburgers and exposure to farms and cattle. Some sporadic infections are also associated with use of immunosuppressive medications and dining at table service restaurants (*197*).

Vehicles of infection, suspected or confirmed, have been identified for most outbreaks listed in the appendix. Figures 1 and 2 depict the relative importance of different vehicles in terms of number of outbreaks and cases. Table 1 lists outbreaks with >100 cases. Of the eight largest outbreaks, four were associated with meat, three with drinking water, and the largest outbreak to date, in Japan, was associated with radish sprouts shipped from one farm.

Importance of different vehicles of infection has changed somewhat over time. Table 2 lists the first recognized outbreaks for different vehicles of infection starting with the first outbreak in 1982 linked to ground beef. Figure 3 shows this data on a timeline. During the 1980s most outbreaks of *E. coli* O157:H7 were associated with inadequately cooked hamburgers and unpasteurized milk (*80;176;178;305*). Some later outbreaks have been traced to other dairy products such as cheese and yogurt (*24;172;248*). In most cases, dairy products were made from unpasteurized milk (*121*); in others, there was a problem with post-pasteurization contamination (*342*).

Increasingly, contaminated water has been reported as a source of human infection. This includes drinking water sources contaminated with animal feces and also contaminated lake and pool water used for swimming and playing (*112*). Fruits and vegetables have also been cited as vehicles for human infection with *E. coli* O157:H7. They have presumably been exposed to untreated manure in the environment or else foods have been washed or irrigated with contaminated water. In a 2005 letter, FDA stated that it was aware of 18 outbreaks of *E. coli* O157:H7 associated with lettuce and one associated with spinach in the U.S. since 1995. (www.cfsan.fda.gov/~dms/prodltr2.html) A large outbreak in Japan, affecting more than 12,000 persons, was associated with contaminated radish sprouts (*145;241*), and other outbreaks have been associated with contaminated fruit juices, melon, and salad greens (*3;59;105;162;294*).

More recently a number of outbreaks have occurred among children visiting farms and petting zoos where they come into direct contact with animals carrying *E. coli* O157:H7 and their environment (*158*). Outbreaks at county fairs may also result from airborne dispersion of bacteria in buildings used to show animals (*57;345*). Finally, direct person-to-person infection occurs particularly among children and their caregivers, such as in day care facilities and also within families (*205;357*).

Reservoirs of *E. coli* O157:H7

Understanding the epidemiology of this organism requires a knowledge of where these bacteria live and grow in nature (their reservoir) and of how humans come into contact them. Ruminants have been identified as the major reservoir of *E. coli* O157:H7, with cattle as the most important source of human infections. Other ruminants known to harbor these bacteria include sheep, goats, and deer. STEC bacteria are occasionally isolated from other animals but it is believed that the bacteria are present as transients and that the animals acquired these bacteria from meat, foods, or water contaminated by fecal material from ruminants (*80*). STEC bacteria usually do not cause illness in animals—with a few exceptions such as diarrhea in calves (*194*).

Cattle—the primary reservoir

Cattle are probably the most important ultimate source of infections for humans. Of the outbreaks listed in Appendix 1, 64 appear to be associated directly with cattle. These include 32 associated with beef, 10 with "meat," and 22 with dairy products from cows. In addition there were 15 outbreaks associated with contact with animals at farms or petting zoos and 5 outbreaks linked to exposure to mud, dust or other environmental sources around farm fields and buildings where farm animals are shown. (Some of these animal-associated outbreaks may have been due to infected sheep or goats.) Many other outbreaks associated with contaminated water and fresh produce may be indirectly associated with cattle.

VTEC have been detected in calves, dairy cows and beef cattle worldwide. Prevalence of these bacteria in cattle and their excreta appears to vary seasonally as well as with the age of the animals and other factors but is generally <10%. Examination of naturally and experimentally infected calves and cattle demonstrated that most *E. coli* O157:H7 adhere to

Corresponding author: M. Ellin Doyle, Ph.D., medoyle@wisc.edu
http://fri.wisc.edu/docs/pdf/FRIBrief_EcoliO157H7humanillness.pdf
Food Research Institute, UW–Madison, August/October 2006
Funded in part by the American Meat Institute Foundation

mucosal epithelium in a short 5 cm long region just proximal from the recto-anal junction. As a result, *E. coli* O157:H7 is present predominantly on the surface of the cow pats (*223;255*).

Evidence has accumulated indicating that some infected animals may shed *E. coli* at much higher levels than others. Analyses of 440 *E. coli*-positive fecal pats in Scotland revealed that *E. coli* O157:H7 levels ranged from <100 cfu/g (in about 75% of pats) to a high of about 9×10^5 cfu/g in the most contaminated pat. Data on cattle from more than 900 farms suggested that the 20% of animals that are most infectious are responsible for approximately 80% of transmission of this pathogen on the farms. It is not presently known if these "super-shedders" are genetically more hospitable to *E. coli* O157:H7 (*232;233*). Generally, shedding of *E. coli* O157:H7 is higher during warm months and higher in calves just after weaning than earlier or later in life (*80;149*).

Other ruminants

Sheep are another significant source of *E. coli* O157:H7 for human infection and these bacteria have been detected in meat (*48;93;94*) and in animals from several countries (*62–64;90;166;167;179;199;236;259*). Monitoring of a flock of sheep for 16 months revealed that the animals shed *E. coli* O157:H7 only during the summer, several different strains were shed by sheep in a flock at a single time, and the strains shed by different sheep changed over time (*209*). Diet may affect shedding of *E. coli* (*208*). Sheep may shed as many as 10^4 cfu of *E. coli* O157:H7 per gram feces (*265*) and these bacteria were found to survive for 15 weeks in manure in a field associated with an outbreak of *E. coli* O157:H7 among scouts at a camp (*264*). Infected lambs may have been associated with outbreaks involving visits to farms or petting zoos.

Goats are another reservoir of *E. coli* O157:H7 and other verotoxin producing *E. coli* (*63;124;199*) and outbreaks of human disease have been linked to cheese made from unpasteurized goats' milk (*135*) and to petting zoos with goats (*116;166*). Following an oral dose of *E. coli* O157:H7, attaching and effacing lesions, similar to those seen in cattle, develop in the colon and recto-anal junction of goats (*210*).

Deer are present in significant numbers in some environments also used by cattle, sheep and goats. Since they are also ruminants, deer may serve as a reservoir for *E. coli* O157:H7 and their droppings may contaminate some fresh fruits and vegetables such as apples (*105;139*). Numerous studies in the U.S. and abroad have documented the presence of *E. coli* O157:H7 and other verotoxin-producing *E. coli* in wild deer (*46;129;139;211;296;298;307;349*). *E. coli* O157:H7 has also been detected in some farmed deer (*91*) and in raw deer meat (*253;336*). Several cases of human infection with *E. coli* O157:H7 have been traced to contaminated deer meat (*202;288*).

Other animals

E. coli O157:H7 has been detected in numerous other animals but none of them are considered a significant source of human infection. In one case, a farmer handling a horse infected with *E. coli* O157:H7 subsequently developed an infection with the same bacterial strain (*89*). VTEC bacteria have been detected in several domestic and wild animals including horses (*50;161;280*), dogs (*62;63;161;198*), rats (*102;103*), an opossum (*297*) and cats (*62*) and in a few zoo animals including monkeys and lemurs (*50*) and an orangutan (*64*).

Swine have been found, in several studies, to be infected with *E. coli* O157:H7 but usually only a small percentage (0.4–14%) of animals test positive (*62;63;68;95;168;191;198;198;199;203;269;313;369*). Experimentally infected pigs shed these bacteria in their feces for at least two months (*108*).

Rabbits, both wild individuals and animals being raised commercially on some farms, were reported to harbor enterohemorrhagic *E. coli* (*150;214;286*). Fecal pellets collected in the summer from wild rabbits on four of six UK farms that harbored VTEC-shedding cattle tested positive for VTEC, including *E. coli* O157:H7. Rabbit fecal pellets collected in winter tested negative (*309*).

Poultry meat sometimes has *E. coli* O157:H7 on its surface and these bacteria do persist in the ceca of experimentally infected chicks for as long as 11 months (*80;312;326*). It was recently reported that 26 of 720 cloacal swab samples from living layer hens in Italian intensive management layer hen farms tested positive for *E. coli* O157:H7 (*123*). There are a few reports of the isolation of *E. coli* O157:H7 in chicken feces (*281;313*) and turkey feces (*168*).

Shellfish in contaminated waters are known to concentrate some pathogens such as *Cryptosporidium*. *E. coli* can be detected in sewage and the possibility exists that pathogenic strains such as *E. coli* O157:H7 could be present in water contaminated by sanitary sewer overflow or runoff from farm fields. Although there has been one recent report of several strains of STEC, including *E. coli* O157:H7, isolated from shellfish collected from coastal areas of France (*156*), it appears that *E. coli* O157:H7 does not significantly contaminate shellfish as yet (*227*).

Transport Hosts

Birds are thought to be possible transport hosts for *E. coli* O157:H7. Some wild birds harbor these bacteria, and pigeons, for example, might spread these bacteria around a farm environment. *E. coli* O157:H7

Corresponding author: M. Ellin Doyle, Ph.D., medoyle@wisc.edu

Funded in part by the American Meat Institute Foundation

has been isolated from gulls (*347*), a rook (relative of crows) (*133*), and pigeons (*159;246;311;316*). Experimentally infected pigeons continue to shed these bacteria for about two weeks (*103*). However, a recent study in Colorado suggests that pigeons may not be a major route of transmission of *E. coli* O157:H7. None of the *E. coli* isolated from 406 pigeon samples collected at dairies produced shiga-like toxins (*276*).

Flies and beetles, including houseflies and filth flies of several species (*7;161;199;249;289;316;332*) and dung beetles (*362*), collected on farms with animals shedding *E. coli* O157:H7, contain detectable levels of these bacteria. These insects frequent fecal deposits and may be able to transfer these bacteria to foods, feed and water. In experiments with houseflies, *E. coli* O157:H7 survived and replicated in the mouthparts and crop of the flies for up to 4 days (*204;308*).

Fruit flies collected from a compost pile of decaying apples and peaches contaminated with *E. coli* contained these bacteria both internally and externally and were able to transfer them to wounds in uncontaminated apples. *E. coli* O157:H7 can grow rapidly in apple wounds. Fruit flies could contribute to widespread contamination of wounded apples that may be processed into cider (*186*).

Slugs are known vegetarian pests that frequently traverse leafy vegetables and may be present on these foods when harvested. Slugs ingest bacteria from the environment and also accumulate bacteria in the mucus surrounding their bodies. Some common gray field slugs collected on a farm in Scotland were found to carry the same pathogenic strain of *E. coli* as detected in feces from sheep grazing there. Slugs may travel 12 m or more per night so there is a potential for slugs to carry *E. coli* O157:H7 from manure to vegetables (*325*).

Routes of Human Infection

Various routes for human infection with *E. coli* O157:H7 were reviewed in a recent article on the epidemiology of outbreaks of this bacterium in the U.S. (1982–2002) (*291*). Studies in Canada and France demonstrated that the incidence of HUS and VTEC infection in humans is correlated with indicators of cattle density (*165;343*). *E. coli* O157:H7 in ruminant feces may be directly ingested by persons interacting or working with animals. Fecal material may contaminate meat during slaughter, may enter lakes or drinking water sources by action of rain or wind, and may be deposited on fruits and vegetables inadvertently or by use of manure for fertilization. In addition, some animals may transport these bacteria from a fecal source to drinking water or foods. All of these routes are variations of a pattern "from turd to tongue" (*278*).

Direct contact

E. coli O157:H7 shed by infected animals may be spread to many surfaces in enclosures where ruminants are kept including the hides of other animals (*98*). Depending on moisture and humidity, these bacteria may persist on gates, stiles and other farm surfaces for more than four weeks (*356*). *E. coli* O157:H7 survives in cattle feces for up to 18 weeks at 15ºC (*146*). This helps explain why a substantial number of people residing on dairy farms have evidence of current (stool cultures) or past (serologic status) infection with VTEC (*361*). Several outbreaks among children who visited farms or petting zoos resulted from direct exposure to these bacteria followed by inadequate handwashing.

Person-to-person spread of *E. coli* O157:H7 has been the primary mode of infection in many outbreaks in day cares, schools and hospitals, particularly where there have been lapses in hygiene (*55;278;293*). In many other outbreaks, some of the cases who consumed contaminated food or water passed the infection directly to others. Although a majority of children infected with *E. coli* O157:H7 shed these bacteria in their feces for only a few days, in more seriously ill children, cells of *E. coli* O157:H7 may be shed for 20–30 days or longer. VTEC bacteria may be present in stool samples even after children become asymptomatic (*195*).

Contaminated food

Beef, lamb, and mutton can be contaminated during slaughter and processing by exposure to feces or hides containing *E. coli* O157:H7. In a recent study in the Midwest, more than 45% of over 330 carcasses tested during July–August contained detectable levels of *E. coli* O157:H7 (*134*). Prevalence of these bacteria on carcasses was 43% at pre-evisceration (immediately after hide removal), 18% at postevisceration (after evisceration, splitting and trimming), and 2% after postprocessing (after antimicrobial treatments including hot water and organic acid washes and steam pasteurization). The initial high level of contamination was greatly reduced during processing, suggesting that sanitary procedures within these plants were effective. However, it was also true that some carcass samples, from lots in which no preslaughter hide or fecal samples contained *E. coli* O157:H7, were found to test positive for these bacteria. This suggests that cross-contamination can occur in processing plants.

Milk from dairy cows, sheep, and goats may be contaminated with *E. coli* and other bacteria from the environment. Proper pasteurization will kill these

Corresponding author: M. Ellin Doyle, Ph.D., medoyle@wisc.edu

Funded in part by the American Meat Institute Foundation

bacteria. Outbreaks of *E. coli* O157:H7 due to contaminated dairy products are usually associated with unpasteurized milk but there have been some cases of post-pasteurization contamination.

Manure is a valuable fertilizer for crops but manure containing *E. coli* O157:H7 may be a source of contamination for vegetables or fruits that are not normally cooked before eating. In one study, these bacteria were able to survive for 42 days in manure heaps that were turned and for 90 days in unturned heaps (*141*) while another study found that *E. coli* O157:H7 was undetectable after 4 weeks in biowaste compost piles (*218*). *E. coli* O157:H7 did not grow or survive in dairy wastewater lagoons (*292*) but did survive for more than two months in garden soil treated with contaminated manure (*183;251*).

Field and greenhouse experiments have demonstrated that both *E. coli* O157:H7-contaminated manure and irrigation water may cause contamination of vegetables. Onions and carrots grown in soils treated with contaminated manure or irrigated with contaminated water had detectable levels of *E. coli* O157:H7 on their subterranean parts for 2.5 to 5.5 months (*183*). Lettuce grown in soil amended with contaminated manure did not contain *E. coli* O157:H7 in leaves (*189;190*) but spray irrigation of lettuce with contaminated water deposited *E. coli* O157:H7 on lettuce leaves and these bacteria persisted for up to 30 days (*324*). Experiments with shredded lettuce, carrots, and cucumbers demonstrated that *E. coli* O157:H7 could survive and grow on these vegetables even under modified atmospheres used in commercial packaging (*2*).

Foods can also be contaminated with *E. coli* O157:H7 by cross-contamination during food preparation and by infected workers who don't practice good hygiene. Several restaurant outbreaks in Oregon and Washington in 1993 were associated with a variety of items from the salad bar but not with steak. All the restaurants obtained their beef from the same source, and it was the practice to trim, macerate, and marinate the beef in the same kitchens used for preparation of fruits and vegetables for the salad bar. It appeared that the beef itself was cooked well enough to destroy *E. coli* O157:H7 but that some raw beef was the source of contamination for the fresh produce (*185*).

Contaminated water

Water used for drinking or recreation has been reported as the vehicle of infection for 49 outbreaks: 6 outbreaks associated with water parks and pools, 18 with lakes, springs, canals, and streams, 10 with well water, 12 with "drinking water," and 3 with tap water. Fecal material from ruminant animals, domestic and/or wild, is the probable source of *E. coli* O157:H7 in lakes, streams, and wells and for some "drinking water" outbreaks. Drinking water from an unchlorinated source was implicated in a large Missouri outbreak (*331*). Infected persons are likely the source of bacteria in the pools and water parks and possibly for some other waterborne outbreaks.

RESPONSES TO *E. coli* O157:H7 OUTBREAKS

Today, outbreaks of foodborne disease are featured in almost instant and broad media coverage. This causes anxiety among the general population, lawsuits, and requests for action to protect the health and safety of consumers. The Federal Government through the Centers for Disease Control and Prevention (CDC), United States Department of Agriculture (USDA), Food and Drug Administration (FDA), and the Environmental Protection Agency (EPA) is under pressure and scrutiny and this in turn results in new guidelines, directives, and regulations. State and local public health departments may also be pressured to increase surveillance activities and respond more rapidly to outbreaks. In addition, reports of foodborne disease prompt formation of organizations such as STOP (Safe Tables Our Priority) dedicated to the support of survivors and families of outbreak victims and to dissemination of information on methods for preventing foodborne disease.

Surveillance

Animals

There is no regular surveillance program to monitor prevalence of infection with *E. coli* O157:H7 among ruminant animals in the U.S. Some other countries, including Sweden (*8*), Denmark (*256*), and The Netherlands (*313*), have established national monitoring programs to detect VTEC in herds or cattle at slaughter. Some U.S. surveys indicated that prevalence of *E. coli* O157:H7 was 13% in feces of one group of summer feedlot cattle (*217*) and 28% in feedlot cattle presented for slaughter at Midwestern plants (*134*). A recent review reported that the prevalence of *E. coli* O157:H7 in beef cattle ranged from 0.2 to 27.8% in published data. In addition, a number of non-O157 VTEC serotypes have been reported from beef cattle (*177*).

A review of numerous papers on dairy cattle in the U.S., Canada, Europe, Japan, and Brazil indicated that reported prevalence of *E. coli* O157:H7 varied widely. (Sampling and analytical methods also differed among these studies.) While there were many reports of prevalences of <20%, there were several very high rates of 40–70%. Ranges of prevalence rates in the U.S. for cows, heifers, and calves were,

Corresponding author: M. Ellin Doyle, Ph.D., medoyle@wisc.edu

Food Research Institute, UW–Madison, August/October 2006
Funded in part by the American Meat Institute Foundation

respectively, 0.2–8.4%, 1.6–3.0%, and 0.4–40% (*178*). Recent surveys of dairy farms in Minnesota (*99*) indicated that 4.5–5.2% of cows harbored *E. coli* O157:H7 while 6.5% of dairy cattle in Louisiana tested positive for these bacteria (*130*). Downer dairy cattle in Wisconsin were found to be three times as likely as healthy cattle to harbor *E. coli* O157:H7 (*77*).

Many surveys may underestimate the actual carriage of *E. coli* O157:H7 because they are based on analyses of single samples from fecal pats. Rectoanal mucosal swab cultures were found to detect a higher prevalence of infection with *E. coli* O157:H7 (9.5%) than fecal cultures (4.7%) (*157*). A higher prevalence of infection was also observed when fecal pats were sampled in several locations rather than at just one site (*132*).

A survey of fecal samples from livestock at 32 county and state fairs found that some of the animals at 31 fairs were shedding *E. coli* O157:H7. Prevalence among cattle was 11.4% and among sheep and goats was 3.6%. VTEC were also isolated from flies and from environmental samples even after the livestock left the fair (*199*). Nine of 12 county fairs in Minnesota had cattle infected with *E. coli* O157:H7 with 11% of manure samples testing positive (*99*). The relatively high prevalence of *E. coli* O157:H7 in fair animals was surprising because these animals are usually raised individually or in small groups and are washed frequently.

Foods

Although *E. coli* O157:H7 has been detected in a variety of foods, there is no regulatory surveillance of foods other than ground beef. FSIS began a microbiological testing program for ground beef in October 1994. A subset of plants and retail outlets that grind beef are selected randomly each month for testing. This sampling and testing program is based on data on outbreaks of foodborne illness and on information from the Office of Public Health Science. Testing results are available on the FSIS website (www.fsis.usda.gov/Science/Ground_Beef_E.Coli_Testing_Results/index.asp). Figure 4 shows the number of ground beef samples tested from 2001–2005. Figure 5 shows a dramatic decrease in the number of samples positive for *E. coli* O157:H7 between 2002 and 2003. This is believed to be a result of a 2002 order by FSIS that beef plants reassess their food safety plans. Most plants made major changes to their operations by installing and validating new technologies to control and test for *E. coli* O157:H7.

Results from 2006 testing so far indicate that *E. coli* O157:H7 contamination is continuing to decrease with 14 positive samples detected among 7,295 samples tested. However, a further recall of approximately 900 lb. ground beef reported on August 18 demonstrates that there may still be some issues to be addressed at some plants.

Human illness

FoodNet (Foodborne Diseases Active Surveillance Network) is a collaborative active surveillance project to track foodborne illness and involves CDC, USDA, FDA, and ten states in the Emerging Infections Program (CA, CO, CT, GA, MD, MN, NM, NY, OR, and TN). FoodNet began collecting information from five sites in 1996 and has now expanded to monitor about 15% of the U.S. population. In the ten states, public health officials frequently contact directors of over 650 laboratories testing stool samples to find new cases of foodborne disease and HUS and report these to CDC. Goals of FoodNet include: determining the burden of foodborne illness in the U. S., monitoring trends in specific foodborne illness, determining specific foods and settings associated with foodborne illness, and developing and assessing interventions to reduce foodborne illness.

Reports of foodborne illness from clinical laboratories are reported by all state health departments to CDC under The National Notifiable Diseases Surveillance System (NNDSS). However, there is some variation among states in the priority and funding given to investigation of foodborne illness and notifiable illnesses and their aggressiveness in tracking down causes of outbreaks and sporadic cases. Many persons with foodborne illness are not seriously ill and do not seek medical care, and it is likely that only a fraction of cases is reported to CDC by passive surveillance systems.

Incidence of *E. coli* O157:H7 infections in the U.S. has declined in recent years according to available surveillance data although there was a slight increase in 2005 (Table 3). According to the latest data from FoodNet (2005), incidence of several other foodborne infections has also decreased, including *Listeria, Campylobacter*, and *Salmonella*. However, incidence of infections with *Vibrio* has increased (*86*). The most dramatic decline in cases, as noted by both FoodNet data and cases included in Notifiable Diseases, was between 2002 and 2003 when there was also a dramatic decrease in positive samples of ground beef detected by FSIS sampling (Figure 6). This trend was also detected in Wisconsin (Figure 7). Enter-Net reports that infections with *E. coli* O157:H7 decreased by 6% between 2000 and 2004 but infections with non-O157 VTEC serotypes has increased. These serogroups are likely underdiagnosed and under-reported in most countries, including the U.S. (*140*).

In response to the 1992–1993 Jack in the Box outbreak, scientists at CDC subjected bacterial isolates from several western states to DNA fingerprinting by pulsed-field gel electrophoresis (PFGE) thereby aiding in identifying the vehicle of infection and the extent of the outbreak. Prompt recognition of the outbreak probably prevented hundreds of cases of illness. CDC then developed standardized PFGE methods for several foodborne pathogens and created a network of national and state public health and regulatory laboratories that can submit PFGE patterns of bacterial isolates to a central database. In 1998, the inauguration of PulseNet was announced by Vice President Gore, and it has since become instrumental in facilitating early recognition of common source outbreaks (www.cdc.gov/pulsenet/). Database administrators analyze PFGE data submitted by participating laboratories to identify clusters of infection and can electronically alert participants to outbreaks. Epidemiologists throughout the country can also rapidly compare PFGE patterns of local foodborne bacterial pathogens and determine whether there are similar strains causing illness elsewhere (*151;160*).

Regional and national meetings, such as the First Foodborne Epidemiologist Meeting held in 2004, bring together foodborne epidemiologists from throughout the country. These meetings are being held more frequently and serve to educate public health workers on the latest methods and research and allow participants to share information on state programs. State and federal laboratory personnel present PulseNet data on sporadic and outbreak cases of foodborne illness.

Regulations

Federal regulations for various aspects of food handling and processing are promulgated by FDA and USDA while EPA is in charge of clean water regulations. In addition, many state and local agencies have regulations that impact food, drinking water, swimming beaches and pools, and agricultural fairs and petting zoos. Many of these regulations were established after significant human disease outbreaks and deaths and/or broad media coverage highlighted shortcomings in handling of food, water, or animals. Figure 8 depicts a timeline showing the association between significant outbreaks and new regulations.

Public outrage aroused by the book *The Jungle* lead to the passage and implementation of food inspection laws, including the Federal Meat Inspection Act (FMIA) and the Pure Food and Drug Act for non-meat products in 1906. The Poultry Products Inspection Act (PPIA) was enacted in 1956. FMIA and PPIA require mandatory inspection of livestock before slaughter and mandatory post-mortem inspection of all carcasses, establish sanitary standards for slaughterhouses and meat processing establishments, and authorize USDA to inspect meat processing and slaughtering operations. Only unadulterated carcasses are approved for further distribution to customers. Meat is considered adulterated "if it bears or contains any poisonous or deleterious substance which may render it injurious to health" or "if it consists in whole or in part of any filthy, putrid, or decomposed substance or is for any other reason unsound, unhealthful, unwholesome, or otherwise unfit for human food" (www.fda.gov/opacom/laws/meat.htm#SUBCHAPTER_1).

USDA regulations implementing FMIA and PPIA are found in Title 9 of the Code of Federal Regulations, and FSIS periodically issues Directives that provide instructions for inspectors. In 1967, the Wholesome Meat Act updated FMIA to require inspection of all meat processed and sold within the same state. The Wholesome Poultry Act of 1968 instituted similar requirements for intrastate processing of poultry.

In addition, at least twenty-eight states have their own meat and/or poultry inspection programs covering small and very small establishments. These programs are run cooperatively with FSIS.

Beef

E. coli O157:H7 was first recognized as a foodborne pathogen after a 1982 outbreak affecting 20 people who had consumed undercooked hamburger. During the next 9 years several other outbreaks associated with beef caused illness in 30–70 persons each. Then in 1991 there was a major ground meat-related outbreak in Canada, and in 1992–1993 an outbreak associated with a fast food restaurant affected over 700 people who had eaten undercooked hamburger in the western U.S., with 37 children developing HUS and 4 deaths. This prompted the passage of several regulations by FSIS to improve meat safety (*254*).

- **1994**: Under the authority of the Federal Meat Inspection Act, Michael Taylor of FSIS, in a speech in September, declared *E. coli* O157:H7 to be an adulterant in ground beef. This was challenged in a Texas court but the court decided that USDA had good reason to consider these bacteria as adulterants.
- **1994**: FSIS Directive 7235.1 required the placement of safe handling labels on packages of raw meat and poultry. These labels address storage, cooking, and holding practices to minimize or prevent growth of pathogenic bacteria (www.fsis.usda.gov/OPPDE/rdad/FSISDirectives/7235.1.pdf).
- **1994**: FSIS Notice 50-94 instituted testing of raw ground beef for *E. coli* O157:H7. This notice was later replaced in **1998** by Directive10,010.1.

Corresponding author: M. Ellin Doyle, Ph.D., medoyle@wisc.edu

Funded in part by the American Meat Institute Foundation

- **1996**: HACCP (Hazard Analysis and Critical Control Point) systems were mandated as a systematic procedure for determining critical points during processing when meat could be contaminated and instituting appropriate controls to prevent contamination. This rule establishes a testing program for the pathogen, requires slaughter plants to routinely test carcasses for generic *E. coli*, and requires all plants to incorporate an antimicrobial process and have in place sanitation standard operating procedures (SSOPs) (www.fsis.usda.gov/OPPDE/rdad/FRPubs/93-016F.pdf) (Fed. Register 61(144):38806-38989).
- **1998**: FSIS Directives 6150.1, rev 1 and 6420.1 told inspectors to enforce zero tolerance for visible fecal, ingesta and milk contamination of poultry and livestock carcasses at slaughter. This directive was revised in **2004** to include head, cheek and wesand meats because these may be included in ground beef (www.fsis.usda.gov/OPPDE/rdad/FSISDirectives/6420.2.pdf).
- **1998**: FSIS Directive10,010.1 revised policy for sample collection and testing to emphasize establishments perceived to be a greater risk. Establishments using validated pathogen reduction interventions on beef carcasses and that had not identified a positive sample within the previous six months would not need to be tested by FSIS (www.fsis.usda.gov/OPPDE/rdad/FSISDirectives/10.010.1.pdf).
- **1998**: Performance standards for lethality and stabilization for cooking of meat were updated (9CFR 318.17).
- **1999**: USDA issued rules allowing irradiation of refrigerated or frozen/uncooked red meat and meat products to destroy pathogenic bacteria including *E. coli* O157:H7 (64 FR 72150) (www.fsis.usda.gov/OPPDE/rdad/FSISDirectives/7700.1Rev1.pdf).
- **1999**: Raw ground beef products, including trimmings, would also be considered adulterated if they contain *E. coli* O157:H7 (Fed. Register 64:2803-2805).
- **2002:** FSIS directed producers of ground beef to reassess HACCP plans in light of new epidemiological data on *E. coli* O157:H7, including improvements in analytical tests, data on conditions in feedlot pens as related to shedding of VTEC, and data on contamination of carcasses as related to prevalence of VTEC on hides and in feces. Implementation of critical controls was required (Fed. Register 67(194):62325).
- **2003**: USDA banned all downer cattle from the human food chain. This was intended to prevent possible transmission of BSE but may also have decreased the prevalence of *E. coli* O157:H7 in meat from culled animals since downer dairy cattle have been shown to have a higher prevalence of *E. coli* O157:H7 (*77*) (www.usda.gov/news/releases/2004/01/0457.htm).
- **2004**: Directive 10.010.1 was revised to require all federally inspected plants that produce raw ground beef products or components be subject to testing for *E. coli* O157:H7. This directive also provided instructions for follow up actions if a sample tested positive and for verifying control of products that are presumptive or proven positive for *E. coli* O157:H7 (www.fsis.usda.gov/OPPDE/rdad/FSISDirectives/10.010.1.pdf).
- **2005:** Directive 7700.1, FSIS revised and updated instructions regarding irradiation of meat and poultry products in official establishments, including off-site irradiation of product (www.fsis.usda.gov/OPPDE/rdad/FSISDirectives/7700.1Rev1.pdf).
- **2006**: Directive 7120.1, Amend 8 provided updated information on safe and suitable ingredients used in the production of meat and poultry products. This includes substances that could be used to destroy bacteria or inhibit their growth.

Juice

Following a multistate outbreak of disease linked to *E. coli* O157:H7 in unpasteurized apple juice in 1996 (*105*), FDA published a final rule (Federal Register 63(130):37029-37056) requiring a warning label on fruit juices that have not been processed to prevent, reduce, or eliminate pathogenic microorganisms. Following another juice-borne outbreak of *Salmonella* in orange juice in 2000, another final rule published by FDA in 2001 required all juice manufacturers to develop and implement a HACCP plan to prevent contamination of juices with dangerous pathogens. Treatments should reduce pathogen levels by 5 logs (Federal Register 66(13):6138-6201).

Fresh produce

FDA has published regulations requiring current good manufacturing practices (GMPs) for food processors under its jurisdiction. However, these regulations do not include "*establishments engaged solely in the harvesting, storage, or distribution of one or more* *raw agricultural commodities. …FDA will, however, issue special regulations if it is necessary to cover these excluded operations*" (21U.S.C. §321(r)). Despite this exclusion, FDA can still regulate fresh produce as "food" subject to the adulteration provisions of the Food, Drug, and Cosmetic Act. A food shall be deemed to be adulterated "if it has been prepared, packed, or held under insanitary conditions whereby it may have become contaminated with filth, or whereby it may have been rendered injurious to health." Therefore, FDA can take enforcement action against an agricultural producer if it determines that food is being produced under insanitary conditions.

In 1998, FDA issued a "Guide to Minimize Microbial Food Safety Hazards for Fruits and Vegetables" that recommended good agricultural practices and GMPs (www.fda.gov/ohrms/dockets/98fr/97n0451a.pdf). However, foodborne outbreaks related to fresh produce continued. In a 2005 letter, FDA noted that 8 outbreaks of *E. coli* O157:H7 associated with lettuce and spinach could be traced to Salinas, California. Creeks and rivers in this area test positive for *E. coli* O157:H7 periodically and some areas are highly susceptible to localized flooding. FDA reminded producers that it considers ready-to-eat crops (such as lettuce) to be adulterated if they have been in contact with flood waters due to potential exposure to sewage, animal waste, pathogens, and other contaminants (www.cfsan.fda.gov/~dms/prodltr2.html).

Drinking water

According to EPA regulations, a system that operates at least 60 days per year and serves 25 people or more or has 15 or more service connections is regulated as a public water system under the Safe Drinking Water Act of 1974. If a system is not a public water system as defined by EPA's regulations, it is not regulated under the Safe Drinking Water Act, although it may be regulated by state or local authorities. Revised National Primary Drinking Water regulations are published in the Code of Federal Regulations (40CFR141.1).

Under the Safe Drinking Water Act, EPA requires public water systems to monitor for coliform bacteria. Systems analyze first for total coliforms, because this test is fast. Any time a sample is positive for total coliforms, the same sample must be analyzed for either fecal coliforms or *E. coli*. Both are indicators of contamination with animal waste or human sewage (www.epa.gov/safewater/sdwa/index.html).

Systems serving 25 to 1,000 people typically take one sample per month. Some states reduce this frequency to quarterly for ground water systems if a recent sanitary survey shows that the system is free of sanitary defects. Systems using surface water, rather than ground water, are required to take extra steps to protect against bacterial contamination because surface water sources are more vulnerable to such contamination. At a minimum, all systems using surface waters must disinfect. Disinfection, including chlorination, will kill *E. coli* O157:H7.

Bottled water is regulated by FDA as a food. Standards of quality are listed in the Code of Federal Regulations (21CFR165.110); these include allowable limits for turbidity, color, odor, coliforms, radioactivity and for 70 chemicals. Coliform levels should not exceed 4 cfu/100 mL in any single sample. State and local governments also regulate bottled water.

Swimming pools and beaches

These are regulated by state and local authorities. Pools and water parks are usually tested regularly for fecal contamination that may result from accidents with young children. Lakes and rivers can also be contaminated by human visitors but are perhaps more at risk of contamination during heavy rains when manure may wash into nearby bodies of water. Water at swimming beaches may not be tested as frequently for coliforms but should be tested when there is likely to be a problem with run off.

Fairs and petting zoos

These are regulated by state and county agriculture or public health departments. During the past several years, regulations requiring more hand-washing stations, warning signs, disinfecting of handrails, etc., and cleanliness in animal enclosures have been enacted in a number of states, including North Carolina and Washington, that experienced outbreaks of *E. coli* O157:H7 at county fairs. The CDC has published a compendium of standardized recommendations to prevent disease associated with animals in public settings. The single most important recommendation was proper washing of hands (*57*).

Industry Initiatives: Intervention Strategies

The emergence of *E. coli* O157:H7 as an important foodborne pathogen has prompted industry initiatives to reduce contamination and improve food safety. These included the use of better equipment and testing procedures as well as improved management systems (HACCP). From 1996–2000, the Economic Research Service of USDA estimates that the meat and poultry industry spent about $180 million per year on improvements in food safety. More than $7 million has been spent since 1994 on applied research related to control of *E. coli* O157:H7. This research led to commercialization of several processes to reduce pathogens on carcasses during processing: (*a*) vacuuming of carcasses with steam or hot water (*126*);

Corresponding author: M. Ellin Doyle, Ph.D., medoyle@wisc.edu

Funded in part by the American Meat Institute Foundation

(*b*) thermal pasteurization in which carcasses are rinsed with water at 180ºF; and (*c*) rinsing with mild organic acids. Steam pasteurization was found to be particularly effective (*279*). By 1997, the two largest beef packing companies ordered such equipment for all their plants.

Following the large 1992–1993 outbreak in the western U.S. linked to hamburgers, the beef industry created a blue-ribbon task force to develop plans to aggressively address the problem of *E. coli* O157:H7 in cattle and beef. Among the accomplishments of this task force were development and implementation strategies for HACCP programs, safe handling labels on packages of beef, and steam vacuuming technology. The Beef Industry Food Safety Council was formed in 1997 to address the problem of foodborne pathogens in beef. This council facilitates research activities and develops and implements education programs for both consumers and the industry.

Because it is not readily apparent to consumers whether meat or produce is contaminated with *E. coli* or other bacteria, if a significant outbreak of foodborne disease is associated with a particular company, results can be economically devastating. One of the most well known cases was Hudson Foods which went out of business after its hamburger was implicated in an outbreak and 25 million pounds of product had to be recalled. The industry has instituted a 100% test and hold program for any product that will be ground. Product from positive lots is diverted to cooking using a validated thermal process or else the product is destroyed. It should be noted that this testing, because of sampling limitations, will not detect all lots that contain *E. coli* O157:H7. However, improvements in cleaning carcasses and testing programs have greatly reduced positive samples of ground beef detected by FSIS in recent years (Figure 5).

Demands by large meat and poultry buyers and many foreign buyers have driven some improvements in food safety practices. After the 1992–1993 outbreak, Jack in the Box canceled all its current contracts with hamburger suppliers and required superior food safety controls including more stringent testing and strict temperature control from its future suppliers. Safer hamburgers were produced using this Bacterial Pathogen Sampling and Testing Program. Many other major meat buyers, including McDonald's, Burger King, Kroger and other fast food and grocery chains, have also mandated their own safety standards in contracts with their suppliers. Ground beef manufacturers are using test and hold procedures to ensure the safety of ground that is shipped out to restaurants and consumers.

Fresh salad vegetables have also been implicated as a source of foodborne illness. Following the 1998 Guidance from the FDA (www.fda.gov/ohrms/dockets/98fr/97n0451.pdf) to producers of fresh fruits and vegetables, a majority of the lettuce/leafy green industry adopted the suggested good agricultural practices (GAPs). In April 2006, representatives of trade associations for growers and marketers of fresh produce published a document to aid their members in implementing safeguards during growing, harvesting, value-added operations, distribution, retail, and food service operations: Commodity Specific Food Safety Guidelines for the Lettuce and Leafy Greens Supply Chain (www.cfsan.fda.gov/~acrobat/lettsup.pdf).

Restaurants have also established their own procedures for ensuring that meat, particularly hamburger, is well-cooked to destroy bacterial pathogens and to prevent cross-contamination during food preparation. The 2005 FDA Model Food Code also was directed toward a more proactive approach through active managerial control in reducing foodborne illness rather than reacting to reported illnesses and outbreaks. This along with the more strenuous recommendation related to no bare hand contact of foods, single use of gloves, and more enhanced reporting of employee health from the employee and restrictions by employers may also have contributed to improved public health as witnessed by the yearly reduction in outbreaks by enteric bacterial pathogens in the U. S. (CDC, *personal communication*). An important part of this process is education of employees on the importance of cleanliness and proper cooking. Menus contain a warning that the restaurant will not be responsible for illness if patrons request that meat not be fully cooked.

DISCUSSION AND SUMMARY

Epidemiology

Evaluation of current practices

Active surveillance programs, such as FoodNet, identify a greater proportion of cases of *E. coli* O157:H7 than passive programs. It has been estimated that as many as 4–8 times as many symptomatic cases occur for each case identified by active surveillance (56). Sporadic cases of *E. coli* O157:H7, confirmed in laboratories, have remained stable over several years but incidence varies for different states. Many factors may affect whether cases are ascertained:

- It is not always possible to obtain samples of implicated foods after an outbreak is recognized but greater efforts could probably be made to identify pathogens and food vehicles if more resources were put into epidemiological investigations. In Japan, it is the practice to reserve a portion of foods served and store it in the freezer for later analysis, if necessary (*162*).

- Successful investigation of foodborne disease outbreaks requires sufficient resources for collecting and analyzing food and clinical samples. Of 336 foodborne outbreaks reported to FoodNet in 1998–1999, 71% had no identified etiology. Reports on these 237 outbreaks indicated that no stool samples were collected in 156 outbreaks and neither food nor stool samples were analyzed in 130 outbreaks. Causative agents cannot be determined if food and/or clinical samples are not examined.
- Patients may not seek medical attention or doctors may not order analyses of stool samples. For example, the percentage of stool samples with bloody diarrhea that were cultured for *E. coli* O157:H7 was 58% in Georgia and 96% in Connecticut (*56*).
- Not all laboratories routinely culture stool samples for *E. coli* O157:H7 and results are not always reported (*56*). Stool samples from suspected VTEC infections may be cultured for *E. coli* O157:H7 but test negative if a non-O157 serotype is responsible.

Extrapolating from data provided by FoodNet and from data in peer-reviewed articles in the literature can be problematic on two counts. (*a*) FoodNet sites were not chosen to be representative of the U.S. population as a whole A demographic comparison of the population in the FoodNet sites with the total U.S. population reveals a similar composition by age. However, Hispanics are underrepresented in FoodNet populations as are those in lower socio-economic groups (*163*). (*b*) Only a small fraction of outbreaks reported to national surveillance programs are subsequently described in peer-reviewed literature. A review of data on outbreaks of infectious intestinal disease in the UK from 1992 to 2003 pointed out that there were 1763 outbreaks reported to authorities but only 55 were presented in peer-reviewed journals. Of 45 VTEC outbreaks reported to the national surveillance program, only 7 were described in the literature (*261*). Outbreaks reported in the literature tend to be those for which microbiological and epidemiological evidence is strongest or more out of the ordinary. This may skew perceptions on the frequency of different vehicles of infection as the UK study found that reports in the literature overemphasized the importance of milk/milk products, deserts, and miscellaneous foods and underestimated importance of meat, fish, poultry and eggs.

In addition, if incidence reports are not adjusted for outbreaks, then data may also be misleading. For example, in one day care outbreak in Minnesota, 43 cases were confirmed but most of these were identified because all children and staff at the day care were cultured and many would probably not have been identified if the outbreak hadn't occurred (*56*).

Recommendations:

- Greater uniformity is needed in statewide investigations and reporting of foodborne illness. Public health systems in some states, such as Minnesota, are very well organized and all specimens are sent to state labs for analysis. Other states with different priorities are less aggressive and rigorous in testing samples and conducting investigations. They should be encouraged to improve funding for public health and to adopt and incorporate better epidemiological procedures and improved laboratory methods.
- Federal grants could be targeted to improving laboratory facilities and training for state epidemiologists, as needed. Greater participation by all states will aid in the rapid identification of multistate outbreaks. (Improving laboratory facilities and expertise at state levels can be considered part of Homeland Security preparations to detect possible outbreaks associated with biological or chemical weapons.)

Interventions—Recent Improvements

Interventions undertaken during recent years by numerous organization concerned with food production and food safety have led to a decline in outbreaks and cases of *E. coli* O157:H7 (Figures 6 and 7). These include:

- Increased emphasis on HACCP and pathogen control by industry and government. This includes improvements at slaughter and processing plants (hide washing, steam vacuuming, hot water and antimicrobial rinses, hold and test programs), at restaurants (proper cooking of hamburger, more care about cross-contamination, notes on menus), and improvements at packing plants and retail stores.
- Increased educational efforts targeted at doctors (1999, American Medical Association Program), workers, and consumers. Numerous web sites and publications maintained by academic institutions, industry trade groups, state and local health departments, and the federal government (e.g. www.fightbac.org/, originally launched in 1996) have made the public more aware of the potential hazard of undercooked hamburger and the

importance of hand washing. Articles in the popular press also reinforce these concepts. Increasingly, agricultural fairs and petting zoos have been posting more signs and providing more hand washing facilities.

- Increased cooperation between federal and state agencies with CDC and FDA sharing more PFGE patterns through PulseNet and federal regulators attending more state and regional meetings on epidemiology and foodborne disease.
- Improved training to make FSIS inspectors more knowledgeable about risk-based inspection.
- Litigation surrounding some high profile outbreaks of foodborne disease. While not primarily intended to be an educational tool, publicity surrounding some outbreaks has undoubtedly alerted consumers to important food safety issues.

Further Research

Much of the research on controlling *E. coli* O157:H7 has been devoted to ensuring the safety of meat after slaughter. Yet outbreaks of illness associated with meat and fresh produce still occur, indicating that further efforts need to address preslaughter conditions of cattle (*78;344;371*). Several studies have documented that some individual animals shed very high levels of *E. coli* O157:H7 while fecal material from other animals in the same herd contains much lower concentrations of these bacteria (*223;232;233*). Carcass contamination with *E. coli* O157:H7 is more likely when feces contain higher concentrations of this pathogen (*136;267*). Research to determine why some cattle are “supershedders” and how to identify them would help reduce transmission in herds and carcass contamination. If super-shedding cattle can be identified, then some interventions noted below could be targeted at those animals.

Although it is impossible to completely eliminate pathogenic bacteria from all cattle, some products and procedures are being tested for efficacy in reducing levels of *E. coli* O157:H7 in cattle. Further research is needed to establish parameters that will make these interventions more consistently effective and commercially useful. These include:

probiotics and competitive exclusion: Probiotic bacteria are harmless or beneficial and compete with pathogens to reduce or prevent their colonization of the gut. Results have not been consistent in “real world” studies but research should be directed at finding more effective combinations of probiotic microbes (*337;365;367*).

antibiotics: These compounds are used to treat illness in animals and to improve feed efficiency and growth. Neomycin treatment has been shown to significantly decrease fecal shedding of *E. coli* O157:H7 in cattle and might be useful under some conditions (*78*). However, there is a trend to reduce antibiotic use in animals to minimize the potential for inducing drug-resistant pathogenic bacteria. This should be considered in assessing recommendations for future use of antibiotics.

bacteriophages: Phages are viruses that attack bacteria. Some are very specific in regard to the bacteria they destroy. As with probiotics, results in real world studies are not consistent but research should continue (*263;315*). FDA has recently approved the use of certain phages on foods to reduce or prevent growth of pathogens.

vaccines: Virulence factors secreted by the type III system of *E. coli* O157:H7 were found to be effective components of a vaccine that significantly reduced shedding of these bacteria by cattle in a feedlot setting (*284*). Further field tests of different vaccine types should determine whether this is a cost effective way to reduce *E. coli* O157:H7 levels in cattle.

chlorate: Chlorate can be metabolized by *E. coli* O157:H7 to form chlorite but as chlorite accumulates the bacteria die. Some experiments indicate this might be a useful way to reduce carriage of *E. coli* O157:H7 shortly before slaughter (*13;321*).

ruminant diet: In order to improve performance, dairy cattle and finishing beef cattle are often fed high grain diets rather than forage. Some of the starch from grains escapes degradation in the rumen and passes to the colon where it may be fermented by *E. coli*, including VTEC strains. Several studies have demonstrated that feeding barley grain increases shedding of *E. coli* O157:H7 (*76;114*). Other studies have indicated that an abrupt change from finishing grain rations to forage decreases *E. coli* O157:H7 (*78*). Long-term feeding of forage rather than grain does not have a dramatic effect on *E. coli* populations in the gut. However, switching cattle from grain to forage just before slaughter may reduce VTEC populations (*372*).

water trough hygiene: *E. coli* O157:H7 can be present and persist in water troughs on farms and in feedlots (*316;317*). These bacteria can survive in sediments in troughs for as long as eight months and serve as a continuing source of reinfection for a herd (*216*). More data on the effects of frequency of cleaning troughs and on antimicrobials that might be used for cleaning or

Corresponding author: M. Ellin Doyle, Ph.D., medoyle@wisc.edu
http://fri.wisc.edu/docs/pdf/FRIBrief_EcoliO157H7humanillness.pdf
Food Research Institute, UW–Madison, August/October 2006
Funded in part by the American Meat Institute Foundation

added to water could suggest methods for reducing transmission among cattle on farms and in feedlots.

In addition to reducing levels of *E. coli* O157:H7 in cattle, further efforts are needed to ensure safety of fresh produce. It is very difficult to sanitize raw fruits and vegetables after harvest because pathogens may reside in inaccessible parts of the food. Therefore, strategies for prevention of preharvest contamination from manure or runoff from livestock operations is essential and should be further investigated (*60;61*).

Outbreaks at petting zoos and fairs can be decreased by hand washing but animal owners and caretakers should be aware that *E. coli* O157:H7 can survive for weeks in feces if conditions are right (*58*) and can also persist for days on surfaces (*356*). *E. coli* O157:H7 can also survive in bedding, such as wood chips and sawdust, and can even grow in bedding contaminated with urine (*120*). There is some controversy as to whether bedding for animals should be cleaned out daily (with the attendant risk of creating aerosols of the pathogens that may be inhaled or deposited on other surfaces) or whether new clean bedding should be placed on top of the old bedding for several days to cover up and keep the pathogens in place during the fair. Further data on effects of different methods of handling bedding and wastes and on sanitizing surfaces may reduce outbreaks at fairs and petting zoos.

More effective educational efforts may also be useful in minimizing outbreaks at swimming pools, water parks, and beaches.

Disease caused by *E. coli* O157:H7 in humans is still not well understood. Reported data on the use of antibiotics for treatment of infections with *E. coli* O157:H7 is conflicting and should be resolved. As yet there is no good animal model for HUS to test various hypotheses about mechanisms for dispersal of shiga toxins through the body, in particular to the kidney. Mechanisms of action of the toxins, interactions of different virulence factors, and potential for transfer of virulence to other bacteria need to be further delineated.

OCTOBER 2006 UPDATE

A large multistate outbreak of *E. coli* O157:H7 associated with packaged fresh spinach occurred between August 19 and September 5, with 203 cases in the U.S. and 1 case in Canada. Cases were first identified in Wisconsin, and this state has the highest number of cases (50) and one confirmed death associated with the outbreak strain. The *E. coli* O157:H7 strain responsible for this outbreak appears to be unusually virulent, with 104 cases requiring hospitalization and 31 cases of HUS (*373;376*).

E. coli O157:H7 has been isolated from open bags of spinach in 10 affected states. This allowed tracing of the spinach to four ranches in the Salinas Valley of California. The outbreak strain of *E. coli* O157:H7 has been isolated from cattle feces and stream water on one ranch (*374*). Samples taken from a wild pig in the area also contained the outbreak strain, and it may be that wild boar trampled fences around the spinach fields and spread the outbreak strain to these fields (*377*).

As noted in a December 2005 FDA letter (http://www.cfsan.fda.gov/~dms/prodltr.html), at least 8 outbreaks of *E. coli* O157:H7 associated with lettuce and spinach in the past ten years have been traced to Salinas, California. This outbreak has resulted in 6 recalls of bagged sinach and salad mixes.

Deposition of *E. coli* O157:H7 on lettuce and spinach leaves may occur from use of contaminated irrigation or washing water or from runoff water flooding the fields. Experiments with shredded lettuce have demonstrated that *E. coli* O157:H7 can survive and grow under modified atmospheres used in commercial packaging (*2*). Recent data indicate that lettuce plants exposed to soil or water (not sprayed on leaves) containing *E. coli* O157:H7 can take up these bacteria which are then present in internal parts of the plant. This would make it impossible to wash off the bacteria (*375*). Further research is needed to better define routes of contamination of fresh produce at farms and packaging facilities and methods to minimize such contamination.

Corresponding author: M. Ellin Doyle, Ph.D., medoyle@wisc.edu

Funded in part by the American Meat Institute Foundation

E. coli O157:H7 Outbreaks Worldwide, 1982–present

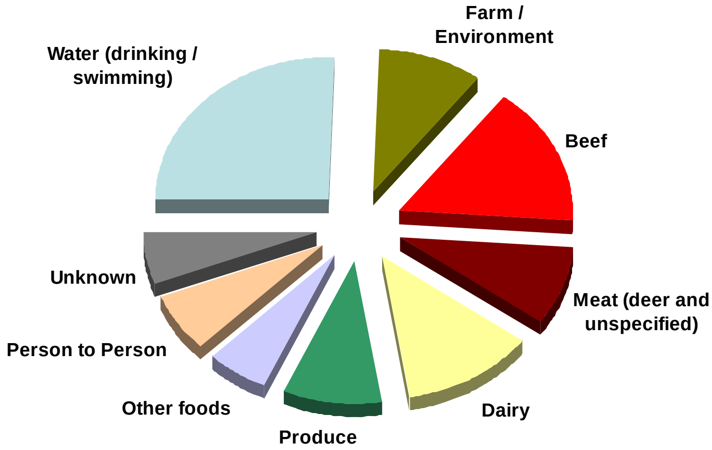

Figure 1. *E. coli* O157:H7 outbreaks worldwide, 1982–present. *Source:* 207 total outbreaks reported in published scientific and government literature.

E. coli O157:H7 Cases Worldwide, 1982–present

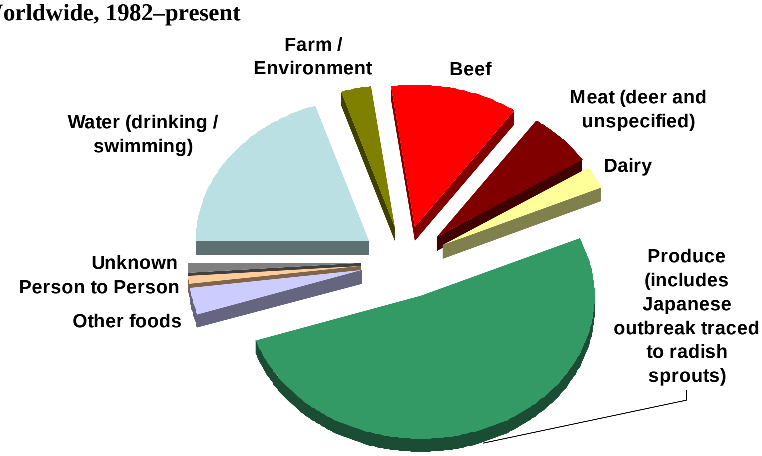

Figure 2a. *E. coli* O157:H7 cases worldwide, 1982–present. *Source:* 26,179 total cases reported in published scientific and government literature, including 12,680 cases in a Japanese outbreak traced to radish sprouts.

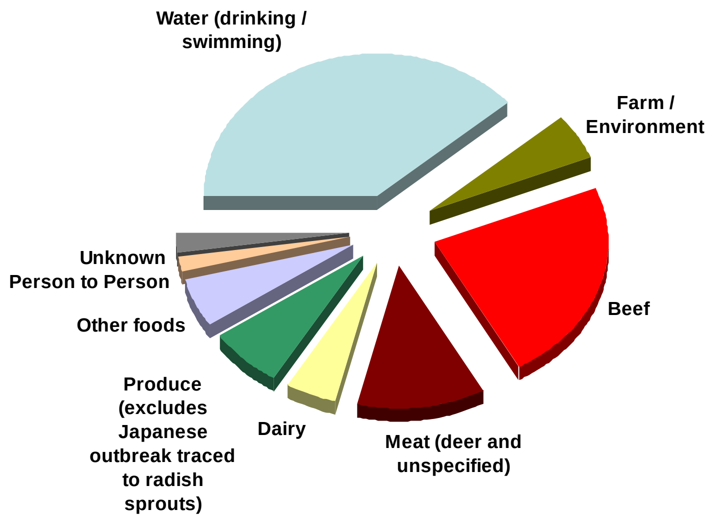

Figure 2b. *E. coli* O157:H7 cases worldwide, 1982–present. *Source:* same data as Fig. 2a but excluding the Japanese outbreak traced to radish sprouts.

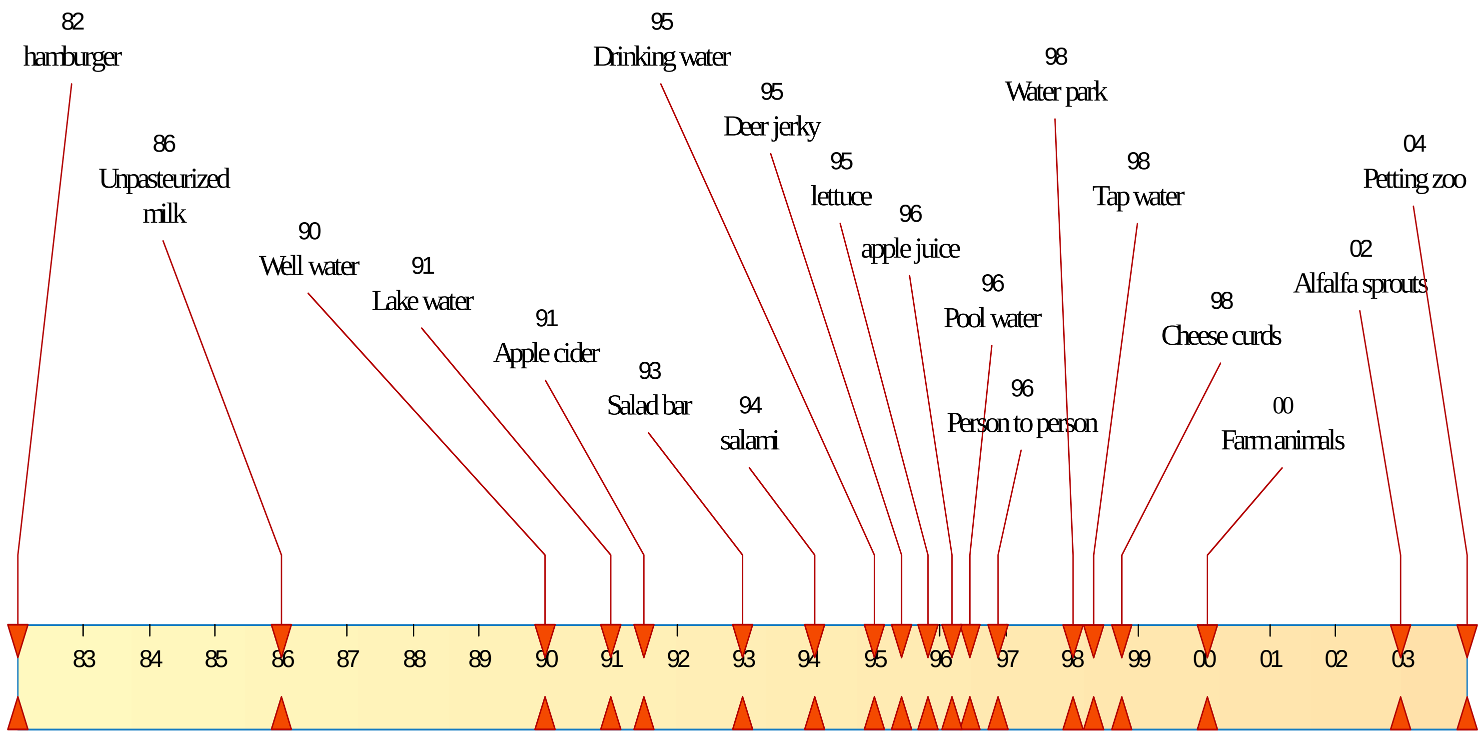

Figure 3. Timeline of appearance of different vehicles for human infection with *E. coli* O157:H7.

Corresponding author: M. Ellin Doyle, Ph.D., medoyle@wisc.edu
http://fri.wisc.edu/docs/pdf/FRIBrief_EcoliO157H7humanillness.pdf

Food Research Institute, UW–Madison, August/October 2006
Funded in part by the American Meat Institute Foundation

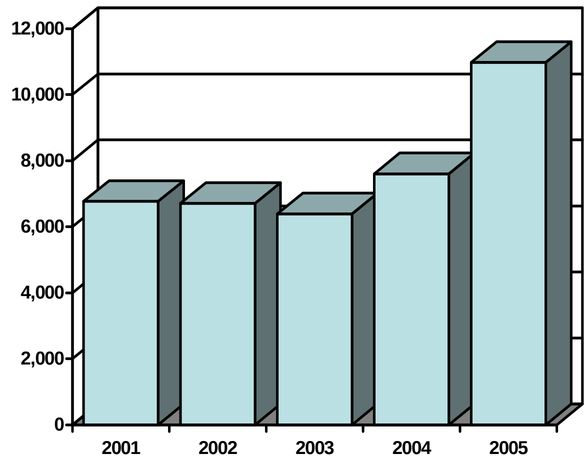

Figure 4. FSIS ground beef samples analyzed by year 2001-2005

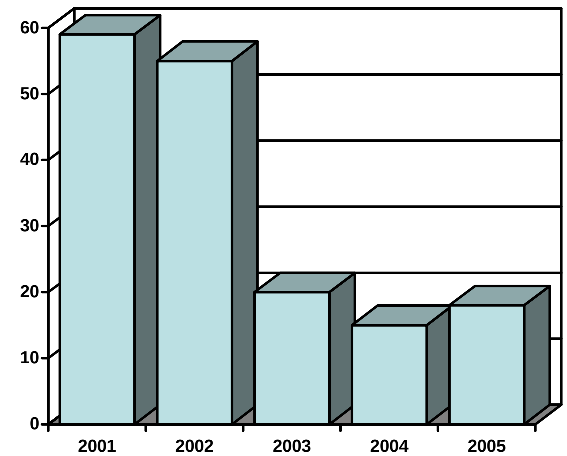

Figure 5. *E. coli* O157:H7 isolates from FSIS sampled ground beef 2001-2005

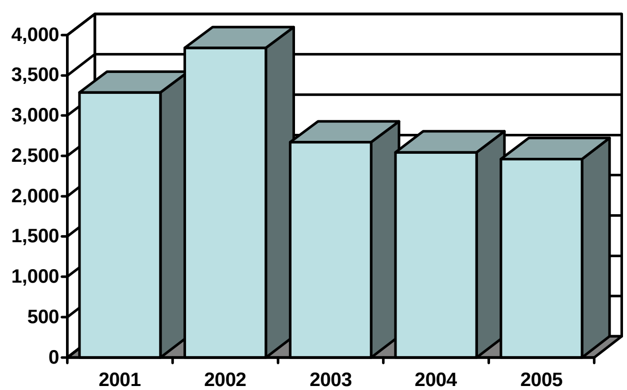

Figure 6. Nationwide *E.coli* O157:H7 cases by year , 2001–2005

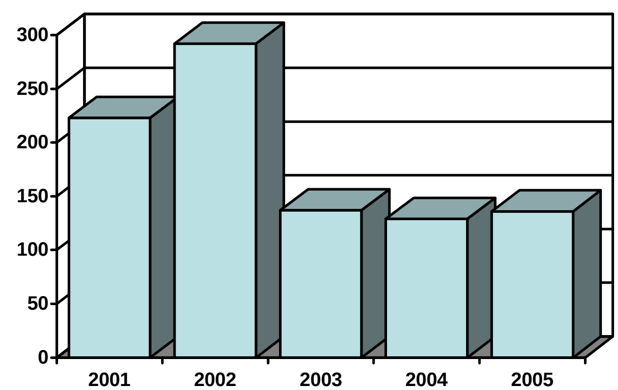

Figure 7. Wisconsin *E. coli* O157:H7 cases by year, 2001–2005

Corresponding author: M. Ellin Doyle, Ph.D., medoyle@wisc.edu
http://fri.wisc.edu/docs/pdf/FRIBrief_EcoliO157H7humanillness.pdf

Funded in part by the American Meat Institute Foundation

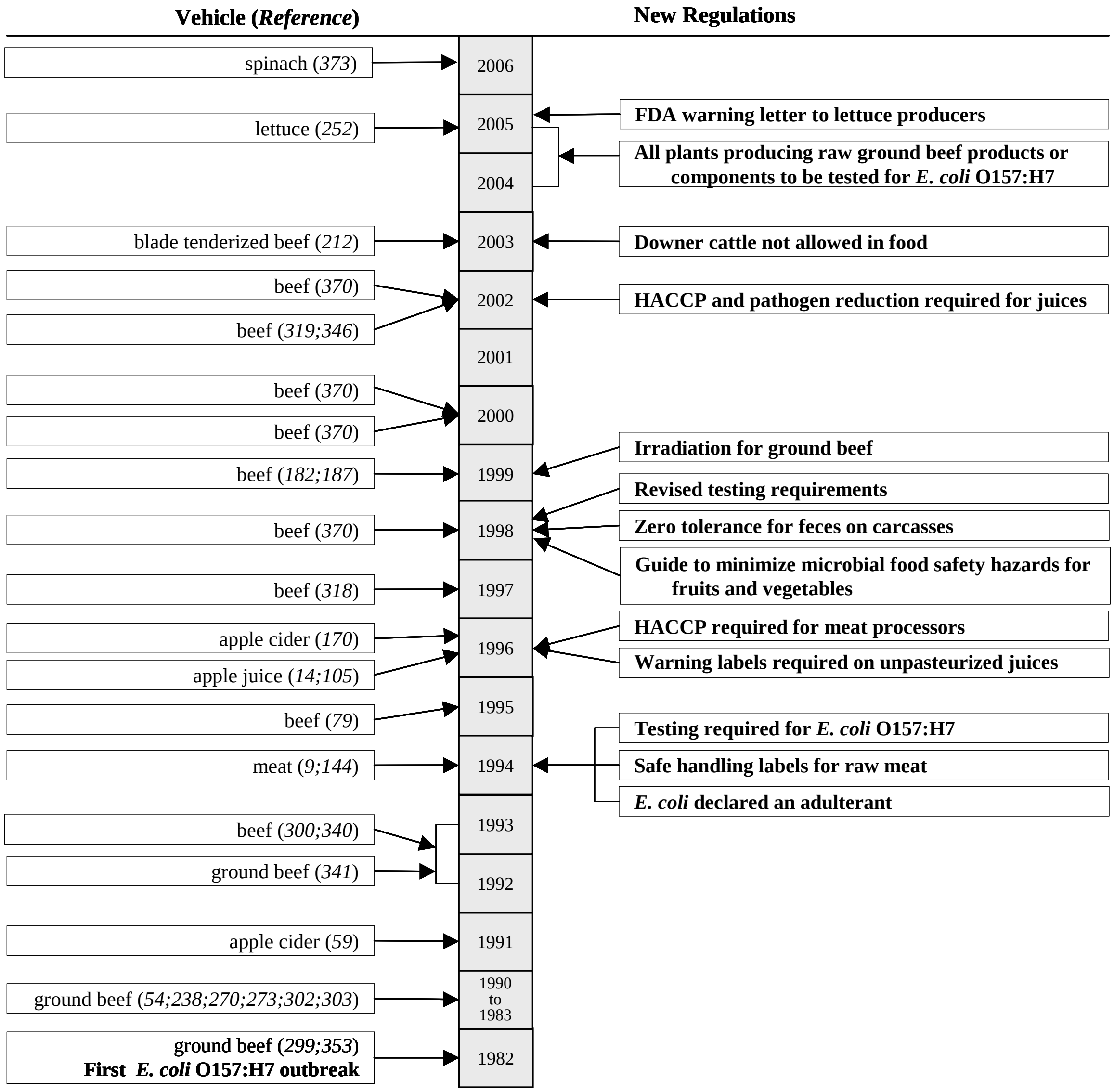

Figure 8. Timeline of important U.S. outbreaks and new regulations to control *E. coli* O157:H7.

Table 1. Large outbreaks of *E. coli* O157:H7 (>100 cases).

Year	# Cases	Location; Vehicle	Reference
1996	12,680	Sakai, Japan; radish sprouts	(*145*;*241*;*363*)
2000	2,300	Canada; drinking water (Walkerton)	(*175*)
1999	>1,000	US; well water (New York)	(*97*)
2000	788	US; raw beef, cross contamination of other foods (Sizzler)	(*370*)
1992–93	>700	US; hamburger at fast food restaurants (Jack in the Box)	(*52*;*69*;*119*;*341*)
1995	633	Fife, Scotland; sewage contamination of drinking water	(*193*)
1991	521	Canada; minced beef and caribou	(*268*)
1996–67	512	Scotland; meat from one shop	(*109*)
1996	503	Scotland; lunch foods	(*277*)
1997	332	UK; restaurant food	(*17*)
1999	329	US: IL, KY, MO; beef	(*182*)
1990	243	US: MO; well water	(*331*)
1999	159	Canada; petting zoo	(*283*)
2005	157	UK; sliced cooked meat	(*305*)
1998	157	US: WY; tap water	(*266*)
1999	143	Canada; salami	(*226*)
1999	127	US: NY; well water, total of 781 cases, some *C. jejuni*	(*258*;*260*)
2005	120	Sweden; lettuce	(*109*)
1999	114	UK; milk, pasteurized	(*153*)
1995–96	110	Sweden; unknown	(*368*)
2002	109	Canada; salads	(*67*)
1997	108	US; alfalfa sprouts	(*107*)
2004	108	US: NC; petting zoo	(*116*)
2000	102	US: UT; irrigation water used for drinking	(*215*)
1994	>100	Scotland; pasteurized milk	(*342*)

Table 2. *E. coli* O157:H7: First reports of vehicles/modes of infection.

Year	Vehicle	Location	Reference
1982	beef, ground (1st reported outbreak)	US	(*299*;*353*)
1985	meat, cooked	Canada	(*81*;*206*)
1986	milk cows, unpasteurized	US; Canada	(*178*;*230*)
1988	beef, roast	US: WI	(*302*)
1989	water, well	Canada	(*294*)
1990	food, restaurant	UK	(*229*)
1990	person-person transfer	Scotland and Israel	(*205*;*219*)
1990	water, drinking	UK	(*122*)
1990	water, tap	Japan	(*5*)
1991	yogurt	UK	(*248*)
1991	water, lake	US: OR	(*201*)
1991	apple cider	US: MA	(*59*)
1992–93	cheese, cows', unpasteurized	France	(*121*)
1992	water, pool	UK: Scotland	(*71*)
1993	salads	US: OR, WA	(*184*)
1994	milk, cows pasteurized	UK: Scotland	(*301*;*342*)
1994	salami	US: WA, CA	(*9*)
1994	sandwiches	US: WI	(*370*)
1994	animals, farm	UK: England	(*320*)
1995	milk, goats, unpasteurized	Czech Republic	(*65*)
1995	ham, cooked	UK: England	(*359*)
1995	lettuce	US and Canada	(*3*;*285*)
1995	deer jerky, meat	US: OR	(*202*;*288*)
1995	potatoes	UK	(*96*)
1995–96	sausage	Germany	(*12*)
1996	apple juice (Odwalla)	US and Canada	(*105*)
1996	food handler	Australia	(*234*)
1996	meat, butcher shop	UK: Scotland	(*109*)
1996	sprouts, radish	Japan	(*145*;*241*)
1997	sprouts, alfalfa	US: MI, VA	(*70*;*107*)
1997	environment (farm field)	UK	(*110*)
1997	cakes, cream filled	UK	(*262*)
1998	cheese curds	US: WI	(*131*)
1998	cream, unpasteurized	UK	(*24*)
1998	water park	US: GA	(*49*)
1999	beach	UK	(*31*;*164*)
1999	animals, petting zoo	Canada	(*283*)
2000	foods, deli	UK	(*36*)
2000	water, stream	US: CA	(*215*)
2003	beef, steak	US: MN	(*212*)
2003	spinach	US: CA	(*294*)
2004	cheese, goats'	France	(*135*)

Table 3. Data from surveillance summaries of foodborne and notifiable diseases from CDC.

	Notifiable cases (*87;88*)		Foodborne Outbreaks (*E. coli*) (*82;83*)			FoodNet Data (*E. coli* O157:H7) (*84;86*)	
Year	*E. coli* O157:H7	non-O157	Outbreaks	Cases	Deaths	cases/100,000	relative rates
2006	*2,141						
2005	2,621	908				1.06	
2004	2,544	626				0.9	0.6
2003	2,671	408				1.06	0.7
2002	3,840	254				1.68	1.05
2001	3,287	191				1.60	0.95
2000	4,528					2.0	1.2
1999	4,513					2.0	1.25
1998	3,161					2.4	1.0
1997	2,555					2.1	1.0
1996	2,741		11	325	1	3.0	1.0
1995	2,139		25	393	1		
1994	1,420		25	902	0		
1993			15	1,340	5		
1992			3	19	0		
1991			3	33	0		
1990			2	80	0		
1989			1	3	0		
1988			2	109	0		

*includes all serotypes of VTEC *E. coli* as of October 1, 2006.

Reference List

1. Abbas Z, Balram C, MacDonald BW, Giffin CS, Armini J, and Panaro L. 2005. An investigation of two simultaneous *E. coli* O157:H7 outbreaks in health region 3, New Brunswick, August to September 2003. Can Commun Dis Rep 31(22):229–235.
2. Abdul-Raouf UM, Beuchat LR, and Ammar MS. 1993. Survival and growth of *Escherichia coli* O157:H7 on salad vegetables. Appl Environ Microbiol 59:1999–2006.
3. Ackers ML, Mahon BE, Leahy E, Goode B, Damrow T, Hayes PS, Bibb WF, Rice DH, Barrett TJ, Hutwagner L, Griffin PM, and Slutsker L. 1998. An outbreak of *Escherichia coli* O157:H7 infections associated with leaf lettuce consumption. J Infect Dis 177:1588–1593.
4. Ackman D, Marks S, Mack P, Caldwell M, Root T, and Birkhead G. 1997. Swimming-associated haemorrhagic colitis due to *Escherichia coli* O157:H7 infection—evidence of prolonged contamination of a fresh water lake. Epidemiol Infect 119:1–8.
5. Akashi S, Joh K, Tsuji A, Ito H, Hoshi H, Hayakawa T, Ihara J, Abe T, Hatori M, Mori T, and Nakamura T. 1994. A severe outbreak of haemorrhagic colitis and haemolytic uraemic syndrome associated with *Escherichia coli* O157:H7 in Japan. Eur J Pediatr 153:650–655.
6. Al-Jader L, Salmon RL, Walker AM, Williams HM, Willshaw GA, and Cheasty T. 1999. Outbreak of *Escherichia coli* O157 in a nursery: lessons for prevention. Arch Dis Child 81:60–63.
7. Alam MJ and Zurek L. 2004. Association of *Escherichia coli* O157:H7 with houseflies on a cattle farm. Appl Environ Microbiol 70:7578–7580.
8. Albihn A, Eriksson E, Wallen C, and Aspan A. 2003. Verotoxinogenic *Escherichia coli* (VTEC) O157:H7 a nationwide Swedish survey of bovine faeces. Act Vet Scan 44:43–52.
9. Alexander ER, Boase J, Davis M, Kirchner L, Osaki C, Tanino T, Samadpour M, Tarr P, Goldoft M, Lankford S, Kobyashi J, Stehrgreen P, Bradley P, Hinton B, Tighe P, Pearson B, Flores GR, Abbott S, Bryant R, Werner SB, and Vugia DJ. 1995. *Escherichia coli* O157:H7 outbreak linked to commercially distributed dry-cured salami—Washington and California, 1994. Morbid Mortal Weekly Rep 44:157–160.
10. Allerberger F, Rossboth D, Dierich MP, Aleksic S, Schmidt H, and Karch H. 1996. Prevalance and clinical manifestations of shiga-toxin producing *Escherichia coli* infections in Austrian children. Eur J Clin Microbiol Infect Dis 15:545–550.
11. Allerberger F, Wagner M, Schweiger P, Rammer HP, Resch A, Dierich MP, Friedrich AW, and Karch H. 2001. *Escherichia coli* O157 infections and unpasteurised milk. Euro Surveill 6(10):147–151.
12. Ammon A, Petersen LR, and Karch H. 1999. A large outbreak of hemolytic uremic syndrome caused by an unusual sorbitol-fermenting strain of *Escherichia coli* O157:H–. J Infect Dis 179:1274–1277.
13. Anderson RC, Carr MA, Miller RK, King DA, Carstens GE, Genovese KJ, Callaway TR, Edrington TS, Jung YS, and McReynolds JL. 2005. Effects of experimental chlorate preparations as feed and water supplements on *Escherichia coli* colonization and contamination of beef cattle and carcasses. Food Microbiol 22:439–447.
14. Anonymous. 1996. Outbreak of *Escherichia coli* O157:H7 infections associated with drinking unpasteurized commercial apple juice—British Columbia, California, Colorado, and Washington, October 1996 Morbid Mortal Weekly Rep 45(44)::975.
15. Anonymous. 1996. VTEC O157 infection in West Yorkshire associated with the consumption of raw milk. CDR Weekly 6:181.
16. Anonymous. 1997. *Escherichia coli* O157 family outbreak in the Highlands. SCIEH Weekly Rep 31:77.
17. Anonymous. 1997. *Escherichia coli* O157 outbreak in Lincolnshire. CDR Weekly 7:101.
18. Anonymous. 1997. European collaboration identifies an outbreak of *Escherichia coli* O157 infection in visitors to Fuerteventura, Canary Islands. CDR Weekly 7(15):127.
19. Anonymous. 1997. Outbreak of *E. coli* infection in a nursing home in Arboath. SCIEH Weekly Rep 31:29.
20. Anonymous. 1997. Outbreak of *E. coli* O157 infection associated with a butchers shop in Hawick. SCIEH Weekly Rep 31:51.
21. Anonymous. 1997. Two outbreaks of Vero cytotoxin producing *Escherichia coli* O157 infection associated with farms. CDR Weekly 7(30):263, 266.
22. Anonymous. 1997. Update on the nosocomial outbreak of *E. coli* O157 at Falkirk and District Royal Infirmary. SCIEH Weekly Rep 31:117.
23. Anonymous. 1997. Vero cytotoxin producing *Escherichia coli* O157. CDR Weekly 7:409.
24. Anonymous. 1998. Cases of *Escherichia coli* O157 infection associated with unpasteurized cream. CDR Weekly 8:389, 392.
25. Anonymous. 1998. *E. coli* O157 contamination of unpasteurised cheese in England. SCIEH Weekly Rep 32:108.
26. Anonymous. 1998. Outbreak of Vero cytotoxin producing *Escherichia coli* O157 infection in Dorset. CDR Weekly 8:183, 186.
27. Anonymous. 1999. *Escherichia coli* O157 associated with eating unpasteurized cheese—update. CDR Weekly 9:131, 134.
28. Anonymous. 1999. Outbreak of VTEC O157 infection at a prison in the Midlands. CDR Weekly 9:281, 284.
29. Anonymous. 1999. Sporadic cases of VTEC O157 infection associated with travel to southern Turkey. CDR Weekly 9:443, 446.
30. Anonymous. 1999. VTEC O157 infection in three nurseries in Preston. CDR Weekly 9:167, 170.
31. Anonymous. 1999. VTEC outbreak linked to beach holidays. CDR Weekly 9:327, 330.
32. Anonymous. 2000. *E. coli* O157 infection in Grampian: update 13 June 2000. SCIEH Weekly Rep 34:137.
33. Anonymous. 2000. Outbreak of VTEC O157 in South Yorkshire. CDR Weekly 10:359.
34. Anonymous. 2000. Outbreaks of *Escherichia coli* O157 infection in two prisons. CDR Weekly 10:375.
35. Anonymous. 2000. Outbreaks of VTEC O157 infection linked to consumption of unpasteurised milk. CDR Weekly 10:203, 206.
36. Anonymous. 2000. Two outbreaks of VTEC O157 infection in northern England. CDR Weekly 10:229.
37. Anonymous. 2001. *E. coli* O157, Inverclyde. SCIEH Weekly Rep 35:153.
38. Anonymous. 2001. Outbreak of vero cytotoxin producing *Escherichia coli* O157 infection in a children's nursery in Suffolk. CDR Weekly 11(26) (28 June 2001).
39. Anonymous. 2002. *E. coli* O157 outbreak. SCIEH Weekly Rep 36:206, 208.
40. Anonymous. 2002. Vero cytotoxic-producing *E. coli* VTEC O157 PT21/28 outbreak associated with a nursery. CDR Weekly 12(50) (12 Dec 2002).
41. Anonymous. 2003. Outbreak of verocytotoxin-producing *Escherichia coli* O157 (VTEC O157) and *Campylobacter* spp associated with a campsite in North Wales. CDR Weekly 13(26) (26 June 2003).
42. Anonymous. 2004. *E. coli* O157 outbreak in Highland. SCIEH Weekly Rep 38:169.

43. Anonymous. 2006. *E. coli* O157 infections in the UK. Euro Surveill 11(6):E060601.2 www.eurosurveillance.org/ew/2006/060601.asp#2.
44. Anonymous. 2006. National increase of vero-cytotoxin producing *E. coli* O157 phage type 8- case-control study. CDR Weekly 16(17) (27 April 2006).
45. Anonymous. 2006. Outbreak of vero cytotoxin-producing *E. coli* (O157 VTEC) associated with a producing butcher in Leeds. CDR Weekly 16(28) (13 July 2006).
46. Asakura H, Makino S, Shirahata T, Tsukamoto T, Kurazono H, Ikeda T, and Takeshi K. 1998. Detection and genetical characterization of shiga toxin-producing *Escherichia coli* from wild deer. Microbial Immunol 42:815–822.
47. Banatvala N, Magnano AR, Cartter ML, Barrett TJ, Bibb WF, Vasile LL, Mshar P, Lambertfair MA, Green JH, Bean NH, and Tauxe RV. 1996. Meat grinders and molecular epidemiology—two supermarket outbreaks of *Escherichia coli* O157:H7 infection. J Infect Dis 173:480–483.
48. Barlow RS, Gobius KS, and Desmarchelier PM. 2006. Shiga toxin-producing *Escherichia coli* in ground beef and lamb cuts: Results of a one-year study. Int J Food Microbiol 111(1):1–5.
49. Barwick RS, Levy DA, Craun GF, Beach MJ, and Calderon RL. 2000. Surveillance for waterborne disease outbreaks—United States, 1997–1998. Morbid Mortal Weekly Rep 49:SS04:1–35.
50. Bauwens L, De Meurichy W, and Vercammen F. 2000. Isolation of *Escherichia coli* O157 from zoo animals. Vlaams Diergeneeskundig Tijdschr 69:76–79.
51. Beecher C. 2005. Shareholder dairy's milk tests positive for *E. coli*. Capital Press. www.ecolilitigation.com/deecreek008.htm
52. Bell BP, Goldoft M, Griffin PM, Davis MA, Gordon DC, Tarr PI, Bartleson CA, Lewis JH, Barrett TJ, Wells JG, and et al. 1994. A multistate outbreak of *Escherichia coli* O157:H7 associated bloody diarrhea and hemolytic uremic syndrome from hamburgers. The Washington experience. J Am Med Assoc 272:1349–1353.
53. Bell BP, Griffin PM, Lozano P, Christie DL, Kobayashi JM, and Tarr PI. 1997. Predictors of hemolytic uremic syndrome in children during a large outbreak of *Escherichia coli* O157:H7 infections. Pediatrics 100:E121–E126.
54. Belongia EA, MacDonald KL, Parham GL, White KE, Korlath JA, Lobato MN, Strand SM, K. Casale, and Osterholm MT. 1991. An outbreak of *Escherichia coli* O157:H7 colitis associated with consumption of precooked meat patties. J Infect Dis 164:338–343.
55. Belongia EA, Osterholm MT, Soler JT, Ammend DA, Braun JE, and MacDonald KL. 1993. Transmission of *Escherichia coli* O157:H7 infection in Minnesota child day-care facilities. J Am Med Assoc 269:883–888.
56. Bender JB, Smith KE, McNees AA, Rabatsky-Ehr TR, Segler SD, Hawkins MA, Spina NL, Keene WE, Kennedy MH, Van Gilder TJ, and Hedberg CW. 2004. Factors affecting surveillance data on *Escherichia coli* O157 infections collected from FoodNet sites, 1996–1999. Clin Infect Dis 38:S157–S164.
57. Bender JB, N. 2005. Compendium of measures to prevent disease associated with animals in public settings, 2005. Morbid Mortal Weekly Rep 54:1–13.
58. Berry ED and Miller DN. 2005. Cattle feedlot soil moisture and manure content: II. Impact on *Escherichia coli* O157. J Environ Qual 34:656–663.
59. Besser RE, Lett SM, Weber JT, Doyle MP, Barrett TJ, Wells JG, and Griffin PM. 1993. An outbreak of diarrhea and hemolytic uremic syndrome from *Escherichia coli* O157:H7 in fresh-pressed apple cider. J Am Med Assoc 269:2217–2220.
60. Beuchat LR. 2002. Ecological factors influencing survival and growth of human pathogens on raw fruits and vegetables. Microb Infect 4:413–423.
61. Beuchat LR. 2006. Vectors and conditions for preharvest contamination of fruits and vegetables with pathogens capable of causing enteric diseases. Brit Food J 108:38–53.
62. Beutin L, Geier D, Steinruck H, Zimmermann S, and Scheutz F. 1993. Prevalence and some properties of verocytotoxin (shiga-like toxin)-producing *Escherichia coli* in seven different species of healthy domestic animals. J Clin Microbiol 31:2483–2488.
63. Beutin L, Geier D, Zimmermann S, and Karch H. 1995. Virulence markers of shiga-like toxin-producing *Escherichia coli* strains from healthy domestic animals of different species. J Clin Microbiol 33:631–635.
64. Beutin L, Knollmann-Schanbacher G, Rietschel W, and Seeger H. 1996. Animal reservoirs of *Escherichia coli* O157:H7. Vet Rec 139:70–71.
65. Bielaszewska M, Janda J, Blahova K, Minarikova H, Jikova E, Karmali MA, Laubova J, Sikulova J, Preston MA, Khakhria R, Karch H, Klazarova H, and Nyc O. 1997. Human *Escherichia coli* O157:H7 infection associated with the consumption of unpasteurized goats milk. Epidemiol Infect 119:299–305.
66. Blackburn BG, Craun GF, Yoder JS, Hill V, Calderon RL, Chen N, Lee SH, Levy DA, and Beach MJ. 2004. Surveillance for waterborne disease outbreaks associated with drinking water—United States, 2001–2002. Morbid Mortal Weekly Rep 53(SS08):23–45.
67. Bolduc D, Srour LR, Sweet L, Neatby A, Issacs A, and Lim G. 2004. Severe outbreak of *Escherichia coli* O157:H7 in health care institutions in Charlottetown, Prince Edward Island, Fall, 2002. Can Commun Dis Rep 30(9):81–88.
68. Borie C, Monreal Z, Guerrero P, Sanchez ML, Martinez J, Arellano C, and Prado V. 1997. Prevalence and characterization of enterohaemorrhagic *Escherichia coli* isolated from healthy cattle and pigs slaughtered in Santiago, Chile. Arch Med Vet 29:205–212.
69. Brandt JR, Fouser LS, Watkins SL, Zelikovic I, Tarr PI, Nazar-Stewart V, and Avner ED. 1994. *Escherichia coli* O157:H7-associated hemolytic-uremic syndrome after ingestion of contaminated hamburgers. J Pediatr 125:519–526.
70. Breuer T, Benkel DH, Shapiro RL, Hall WN, Winnett MM, Linn MJ, Neimann J, Barrett TJ, Dietrich S, Downes FP, Toney DM, Pearson JL, Rolka H, Slutsker L, and Griffin PM. 2001. A multistate outbreak of *Escherichia coli* O157:H7 infections linked to alfalfa sprouts grown from contaminated seeds. Emerg Infect Dis 7:977–982.
71. Brewster DH, Brown MI, Robertson D, Houghton GL, Bimson J, and Sharp JC. 1994. An outbreak of *Escherichia coli* O157 associated with a children's paddling pool. Epidemiol Infect 112:441–447.
72. Brooks JT, Bergmire-Sweat D, Kennedy M, Hendricks K, Garcia M, Marengo L, Wells J, Ying M, Bibb W, Griffin PM, Hoekstra RM, and Friedman CR. 2004. Outbreak of Shiga toxin-producing *Escherichia coli* O111:H8 infections among attendees of a high school cheerleading camp. Clin Infect Dis 38:190–198.
73. Brooks JT, Sowers EG, Wells JG, Greene KD, Griffin PM, Hoekstra RM, and Strockbine NA. 2005. Non-O157 shiga toxin-producing *Escherichia coli* infections in the United States, 1983–2002. J Infect Dis 192:1422–1429.
74. Bruce MG, Curtis MB, Payne MM, Gautom RK, Thompson EC, Bennett AL, and Kobayashi JI. 2003. Lake-associated outbreak of *Escherichia coli* O157:H7 in Clark County, Washington, August 1999. Arch Pediatr Adoles Med 157:1016–1021.
75. Bruneau A, Rodrigue H, Ismäel J, Dion R, and Allard R. 2004. Outbreak of *E. coli* O157:H7 associated with bathing

at a public beach in the Montreal-Centre region. Can Commun Dis Rep 30(15)133–136.
76. Buchko SJ, Holley RA, Olson WO, Gannon VPJ, and Veira DM. 2000. The effect of different grain diets on fecal shedding of *Escherichia coli* O157:H7 by steers. J Food Prot 63:1467–1474.
77. Byrne CM, Erol I, Call JE, Kaspar CW, Buege DR, Hiemke CJ, Fedorka-Cray PJ, Benson AK, Wallace FM, and Luchansky JB. 2003. Characterization of *Escherichia coli* O157:H7 from downer and healthy dairy cattle in the upper Midwest region of the United States. Appl Environ Microbiol 69:4683–4688.
78. Callaway TR, Anderson RC, Edrington TS, Genovese KJ, Bischoff KM, Poole TL, Jung YS, Harvey RB, and Nisbet DJ. 2004. What are we doing about *Escherichia coli* O157:H7 in cattle? J. Anim Sci 82:E93–E99.
79. Cannon M, Thomas H, Sellers W, Bates M, Blake P, Stetler H, Toomey K, Fowler J, Halford S, Young G, Hall S, Erwin P, Boaz V, and Swinger G. 1996. Outbreak of *Escherichia coli* O157:H7 infection—Georgia and Tennessee, June 1995. Morbid Mortal Weekly Rep 45:249–251.
80. Caprioli A, Morabito S, Brugere H, and Oswald E. 2005. Enterohaemorrhagic *Escherichia coli*: emerging issues on virulence and modes of transmission. Vet Res 36:289–311.
81. Carter AO, Borczyk AA, Carlson JA, Harvey B, Hockin JC, Karmali MA, Krishnan C, Korn DA, and Lior H. 1987. A severe outbreak of *Escherichia coli* O157:H7—associated hemorrhagic colitis in a nursing home. N Engl J Med 317:1496–1500.
82. Centers for Disease Control. 1996. Surveillance for Foodborne Disease Outbreaks—United States, 1988–1992. Morbid Mortal Weekly Rep 45:1–67.
83. Centers for Disease Control. 2000. Surveillance for foodborne-disease outbreaks—United States, 1993–1997. Morbid Mortal Weekly Rep 49:1–62.
84. Centers for Disease Control. 2006. FoodNet Surveillance Report for 2004 (Final Report). 203 p. www.cdc.gov/foodnet/annual/2004/Report.pdf
85. Centers for Disease Control. 2006. National antimicrobial resistance monitoring system for enteric bacteria (NARMS): 2003 human isolates final report. 67 p. www.cdc.gov/narms/annual/2003/NARMS2003AnnualReport.pdf
86. Centers for Disease Control. 2006. Preliminary FoodNet data on the incidence of infection with pathogens commonly transmitted through food—10 states, United States, 2005. Morbid Mortal Weekly Rep 55:392–395.
87. Centers for Disease Control. 2006. Provisional cases of selected notifiable diseases. Morbid Mortal Weekly Rep 55:1083.
88. Centers for Disease Control. 2006. Summary of notifiable diseases, United States 1993–2004. www.cdc.gov/mmwr/summary.html
89. Chalmers RM, Salmon RL, Willshaw GA, Cheasty T, Looker N, Davies I, and Wray C. 1997. Vero-cytotoxin-producing *Escherichia coli* O157 in a farmer handling horses. Lancet 349:1816.
90. Chapman PA. 2000. Sources of *Escherichia coli* O157 and experiences over the past 15 years in Sheffield, UK. J Appl Microbiol 88:51S–60S.
91. Chapman PA and Ackroyd HJ. 1997. Farmed deer as a potential source of verocytotoxin-producing *Escherichia coli* O157. Vet Rec 141:314–315.
92. Chapman PA, Cornell J, and Green C. 2000. Infection with verocytotoxin-producing *Escherichia coli* O157 during a visit to an inner city open farm. Epidemiol Infect 125:531–536.
93. Chapman PA, Malo ATC, Ellin M, Ashton R, and Harkin MA. 2001. *Escherichia coli* O157 in cattle and sheep at slaughter, on beef and lamb carcasses and in raw beef and lamb products in South Yorkshire, UK. Int J Food Microbiol 64:139–150.
94. Chapman PA, Siddons CA, Malo ATC, and Harkin MA. 1996. Lamb products as a potential source of *E. coli* O157. Vet Rec 139:427–428.
95. Chapman PA, Siddons CA, Malo ATC, and Harkin MA. 1997. A 1-year study of *Escherichia coli* O157 in cattle, sheep, pigs and poultry. Epidemiol Infect 119:245–250.
96. Chapman PA, Siddons CA, Manning J, and Cheetham C. 1997. An outbreak of infection due to verocytotoxin-producing *Escherichia coli* O157 in four families—the influence of laboratory methods on the outcome of the investigation. Epidemiol Infect 119:113–119.
97. Charatan F. 1999. New York outbreak of *E. coli* poisoning affects 1000 and kills two. Brit Med J 319:873.
98. Childs KD, Simpson CA, Warren-Serna W, Bellenger G, Centrella B, Bowling RA, Ruby J, Stefanek J, Vote DJ, Choat T, Scanga JA, Sofos JN, Smith GC, and Belk KE. 2006. Molecular characterization of *Escherichia coli* O157:H7 hide contamination routes: Feedlot to harvest. J Food Prot 69:1240–1247.
99. Cho S, Bender JB, Diez-Gonzalez F, Fossler CP, Hedberg CW, Kaneene JB, Ruegg PL, Warnick LD, and Wells SJ. 2006. Prevalence and characterization of *Escherichia coli* O157 isolates from Minnesota dairy farms and county fairs. J Food Prot 69:252–259.
100. Cieslak PR, Noble SJ, Maxson DJ, Empey LC, Ravenholt O, Legarza G, Tuttle J, Doyle MP, Barrett TJ, Wells JG, Mcnamara AM, and Griffin PM. 1997. Hamburger-associated *Escherichia coli* O157:H7 infection in Las Vegas—a hidden epidemic. Am J Publ Health 87:176–180.
101. Cimolai N, Basalyga S, Mah DG, Morrison BJ, and Carter JE. 1994. A continuing assessment of risk factors for the development of *Escherichia coli* O157:H7-associated hemolytic uremic syndrome. Clin Nephrol 42:85–89.
102. Cizek A, Alexa P, Literak I, Hamrik J, Novak P, and Smola J. 1999. Shiga toxin-producing *Escherichia coli* O157 in feedlot cattle and Norwegian rats from a large-scale farm. Lett Appl Microbiol 28:435–439.
103. Cizek A, Literak I, and Scheer P. 2000. Survival of *Escherichia coli* O157 in faeces of experimentally infected rats and domestic pigeons. Lett Appl Microbiol 31:349–352.
104. Clark A, Morton S, Wright P, Corkish J, Bolton FJ, and Russell J. 1997. A community outbreak of Vero cytotoxin producing *Escherichia coli* O157 infection linked to a small farm dairy. Commun Dis Rep Rev 7:R206–R211.
105. Cody SH, Glynn MK, Farrar JA, Cairns KL, Griffin PM, Kobayashi J, Fyfe M, Hoffman R, King AS, Lewis JH, Swaminathan B, Bryant RG, and Vugia DJ. 1999. An outbreak of *Escherichia coli* O157:H7 infection from unpasteurized commercial apple juice. Ann Int Med 130:202–209.
106. Combs BG, Wise RP, Tribe IG, Mwanri L, and Raupach JCA. 2003. Investigation of two clusters if shiga toxin-producing *Escherichia coli* cases in South Australia. Commun Dis Intelligence 27:517–519.
107. Como-Sabetti K, Reagan S, Allaire S, Parrott K, Simonds CM, Hrabowy S, Ritter B, W. Hall, Altamirano J, Martin R, Downes F, Jennings G, Barrie R, Dorman MF, Keon N, Kucab M, Alshab A, Robinsondunn B, Dietrich S, Moshur L, Reese L, Smith J, Wilcox K, Tilden J, Wojtala G, and et al. 1997. Outbreaks of *Escherichia coli* O157:H7 infection associated with eating alfalfa sprouts—Michigan and Virginia, June–July 1997. Morbid Mortal Weekly Rep 46:741–744.
108. Cornick NA and Helgerson AF. 2004. Transmission and infectious dose of *Escherichia coli* O157:H7 in swine. Appl Environ Microbiol 70:5331–5335.
109. Cowden JM, Ahmed S, Donaghy M, and Riley A. 2001. Epidemiological investigation of the Central Scotland

outbreak of *Escherichia coli* O157 infection, November to December 1996. Epidemiol Infect 126:335–341.
110. Crampin M, Willshaw G, Hancock R, Djuretic T, Elstob C, Rouse A, Cheasty T, and Stuart J. 1999. Outbreak of *Escherichia coli* O157 infection associated with a music festival. Eur J Clin Microbiol Infect Dis 18:286–288.
111. Cransberg K, Vandenkerkhof JHCT, Banffer JRJ, Stijnen C, Wernars K, Vandekar NCAJ, Nauta J, and Wolff ED. 1996. Four cases of hemolytic uremic syndrome—source contaminated swimming water. Clin Nephrol 46:45–49.
112. Craun GF, Calderon RL, and Craun MF. 2005. Outbreaks associated with recreational water in the United States. Int J Environ Health Res 15:243–262.
113. Crump JA, Sulka AC, Langer AJ, Schaben C, Crielly AS, Gage R, Baysinger M, Moll M, Withers G, Toney DM, Hunter SB, Hoekstra RM, Wong SK, Griffin PM, and Van Gilder TJ. 2002. An outbreak of *Escherichia coli* O157:H7 infections among visitors to a dairy farm. N Engl J Med 347:555–560.
114. Dargatz D.A., S. J. Wells, L. A. Thomas, D. D. Hancock, and L. P. Garber. 1997. Factors associated with the presence of *Escherichia coli* O157 in feces of feedlot cattle. J Food Prot 60:466–470.
115. David ST, MacDougall L, Louie K, McIntyre L, Paccagnella AM, Schleicher S, and Hamade A. 2004. Petting zoo-associated *Escherichia coli* O157:H7- secondary transmission, asymptomatic infection, and prolonged shedding in the classroom. Can Commun Dis Rep 30(20):173–180.
116. Davies M, Engel J, Griffin D, Ginzl D, Hopkins R, Blackmore C, Lawaczec E, Nathan L, Levy C, Briggs G, Kioski C, Kreis S, Keen J, Durso L, Schulte J, Fullerton K, Long C, Smith S, Barton C, Gleit C, Joyner M, Montgomery S, Braden C, Goode B, and Chertow D. 2005. Outbreaks of *Escherichia coli* O157:H7 associated with petting zoos—North Carolina, Florida, and Arizona, 2004 and 2005. Morbid Mortal Weekly Rep 54:1277–1280.
117. Davis BS and Brogan RT. 1994. Butchers, burgers, and *E. coli* O157. Commun Dis Environ Health Scotland 28:3–6.
118. Davis BS and Brogan RT. 1995. A widespread community outbreak of *E. coli* O157 infection in Scotland. Public Health 109:381–388.
119. Davis M. 1994. Update—Multistate outbreak of *Escherichia coli* O157:H7 infections from hamburgers—western United States, 1992–1993 Morbid Mortal Weekly Rep 42(14):258–264.
120. Davis MA, Cloud-Hansen KA, Carpenter J, and Hovde CJ. 2005. *Escherichia coli* O157:H7 in environments of culture-positive cattle. Appl Environ Microbiol 71:6816–6822.
121. Deschenes G, Casenave C, Grimont F, Desenclos JC, Benoit S, Collin M, Baron S, Mariani P, Grimont PAD, and Nivet H. 1996. Cluster of cases of haemolytic uraemic syndrome due to unpasteurised cheese. Pediatr Nephrol 10(2):203–205.
122. Dev VJ, Main M, and Gould I. 1991. Waterborne outbreak of *Escherichia coli* O157. Lancet 337:1412.
123. Dipineto L, Santaniello A, Fontanella M, Lagos K, Fioretti A, and Menna LF. 2006. Presence of Shiga toxin-producing *Escherichia coli* O157:H7 in living layer hens. Lett Appl Microbiol 43:293–295.
124. Dontorou A, Papadopolou C, Filioussis G, Apostolou I, Economou V, Kansouzidou A, and Levidiotou S. 2004. Isolation of a rare *Escherichia coli* O157:H7 strain from farm animals in Greece. Comp Immunol Microbiol Infect Dis 27:201–207.
125. Doorduyn Y, de Jager CM, van der Zwaluw, Friesma IHM, Heuvelink AE, de Boer E, Wannet WJB, and van Duynhoven YTHP. 2006. Shiga toxin-producing *Escherichia coli* (STEC) O157 outbreak, The Netherlands, September–October 2005. Euro Surveill Monthly 11(7–8) www.eurosurveillance.org/em/v11n07/1107-223.asp.
126. Dorsa WJ, Cutter CN, and Siragusa GR. 1997. Effects of steam-vacuuming and hot water spray wash on the microflora of refrigerated beef carcass surface tissue inoculated with *Escherichia coli* O157:H7, Listeria innocua, and Clostridium sporogenes. J Food Prot 60:114–119.
127. Duffell E, Espié E, Nichols T, Adak GK, De Valk H, Anderson K, and Stuart JM. 2003. Investigation of an outbreak of *E. coli* O157 infections associated with a trip to France of schoolchildren from Somerset, England. Euro Surveill 8(4):81–86.
128. Dundas S, Todd WTA, Stewart AI, Murdoch PS, Chaudhuri AKR, and Hutchinson SJ. 2001. The central Scotland *Escherichia coli* O157:H7 outbreak: Risk factors for the hemolytic uremic syndrome and death among hospitalized patients. Clin Infect Dis 33:923–931.
129. Dunn JR, Keen JE, Moreland D, and Thompson RA. 2004. Prevalence of *Escherichia coli* O157:H7 in white-tailed deer from Louisiana. J Wildlife Dis 40:361–365.
130. Dunn JR, Keen JE, and Thompson RA. 2004. Prevalence of shiga-toxigenic *Escherichia coli* O157:H7 in adult dairy cattle. J Am Vet Med Assoc 224:1151–1158.
131. Durch J, Ringhand T, Manner K, Barnett M, Proctor M, Davis J, and Boxrud D. 2000. Outbreak of *Escherichia coli* O157:H7 infection associated with eating fresh cheese curds—Wisconsin, June 1998. Morbid Mortal Weekly Rep 49:911–913.
132. Echeverry A, Loneragan GH, Wagner BA, and Brashears MM. 2005. Effect of intensity of fecal pat sampling on estimates of *Escherichia coli* O157 prevalence. Am J Vet Res 66:2023–2027.
133. Ejidokun OO, Walsh A, Barnett J, Hope Y, Ellis S, Sharp MW, Paiba GA, Logan M, Willshaw GA, and Cheasty T. 2006. Human Vero cytotoxigenic *Escherichia coli* (VTEC) O157 infection linked to birds. Epidemiol Infect 134:421–423.
134. Elder RO, Keen JE, Siragusa GR, Barkocy-Gallagher GA, Koohmaraie M, and Laegreid WW. 2000. Correlation of enterohemorrhagic *Escherichia coli* O157 prevalence in feces, hides, and carcasses of beef cattle during processing. Proc Nat Acad Sci USA 97:2999–3003.
135. Espié E, Vaillant V, Mariani-Kurkdjian P, Grimont F, Martin-Schaller R, De Valk H, and Vernozy-Rozand C. 2006. *Escherichia coli* O157 outbreak associated with fresh unpasteurized goats' cheese. Epidemiol Infect 134:143–146.
136. Fegan N, Higgs G, Vanderlinde P, and Desmarchelier P. 2005. An investigation of *Escherichia coli* O157 contamination of cattle during slaughter at an abattoir. J Food Prot 68:451–457.
137. Feldman KA, Mohle-Boetani JC, Ward J, Furst K, Abbott SL, Ferrero DV, Olsen A, and Werner SB. 2002. A cluster of *Escherichia coli* O157: Nonmotile infections associated with recreational exposure to lake water. Pub Health Rep 117:380–385.
138. Ferguson DD, Scheftel J, Cronquist A, Smith K, Woo-Ming A, Anderson E, Knutsen J, De AK, and Gershman K. 2005. Temporally distinct *Escherichia coli* O157 outbreaks associated with alfalfa sprouts linked to a common seed source—Colorado and Minnesota, 2003. Epidemiol Infect 133:439–447.
139. Fischer JR, Zhao T, Doyle MP, Goldberg MR, Brown CA, Sewell CT, Kavanaugh DM, and Bauman CD. 2001. Experimental and field studies of *Escherichia coli* O157:H7 in white-tailed deer. Appl Environ Microbiol 67:1218–1224.
140. Fisher I, Meakins S, and Enter-net participants. 2006. Surveillance of enteric pathogens in Europe and beyond: Enter-net annual report for 2004. Eurosurveillance 11.
141. Fremaux B, Delignette-Muller ML, Prigent-Combaret C, Gleizal A, and Vernozy-Rozand C. 2007. Growth and survival of non-O157:H7 shiga-toxin-producing *Escherichia coli* in cow manure. J Appl Microbiol 102(1):89–99.

Corresponding author: M. Ellin Doyle, Ph.D., medoyle@wisc.edu
http://fri.wisc.edu/docs/pdf/FRIBrief_EcoliO157H7humanillness.pdf
Food Research Institute, UW–Madison, August/October 2006
Funded in part by the American Meat Institute Foundation

142. French mult-agency outbreak investigation team. 2005. Outbreak of *E. coli* O157:H7 infections associated with a brand of beefburgers in France. Euro Surveill 10(11):E051103.1 www.eurosurveillance.org/ew/2005/051103.asp#1
143. Friedman MS, Roels T, Koehler JE, Feldman L, Bibb WF, and Blake P. 1999. *Escherichia coli* O157:H7 outbreak associated with an improperly chlorinated swimming pool. Clin Infect Dis 29:298–303.
144. Frost B, Chaos C, Ladaga L, Day W, Tenney M, Mcwilliams D, Barrett E, Branch L, Jenkins S, Linn M, Turf E, Woolard D, Miller GB, Henderson S, Campbell B, Mismas M, Dvorak J, Patel D, Peery D, Morano J, and Campbell K. 1995. *Escherichia coli* O157:H7 outbreak at a summer camp—Virginia, 1994. Morbid Mortal Weekly Rep 44:419–421.
145. Fukushima H, Hashizume T, Morita Y, Tanaka J, Azuma K, Mizumoto Y, Kaneno M, Matsu-ura MO, Konma K, and Kitani T. 1999. Clinical experiences in Sakai City Hospital during the massive outbreak of enterohemorrhagic *Escherichia coli* O157 infections in Sakai City, 1996. Pediatr Int 41:213–217.
146. Fukushima H, Hoshina K, and Gomyoda M. 1999. Long-term survival of shiga toxin-producing *Escherichia coli* O26, O111, and O157 in cattle feces. Appl Environ Microbiol 65:5177–5181.
147. Gage R, Crielly A, Baysinger M, Chernak E, Herbert G, Johnson-Entsuah A, Fraser G, Rinehardt C, Solomon M, Withers G, Berman R, Moll M, Rankin J, Carroll J, Ettinger M, Henderson S, Mismas M, Patel D, Reed T, Smith E, Wozniak J, Toney D, Pearson J, Hofmann J, Grendon J, and Kobayashi J. 2001. Outbreaks of *Escherichia coli* O157:H7 infections among children associated with farm visits—Pennsylvania and Washington, 2000. Morbid Mortal Weekly Rep 50:293–297.
148. Galanis E, Longmore K, Hasselback P, Swann D, Ellis A, and Panaro L. 2003. Investigation of an *E. coli* O157:H7 outbreak in Brooks, Alberta, June–July 2002. Can Commun Dis Rep 29(3)2–28.
149. Garber LP, Wells SJ, Hancock DD, Doyle MP, Tuttle J, Shere JA, and Zhao T. 1995. Risk factors for fecal shedding of *Escherichia coli* O157:H7 in dairy calves. J Am Vet Med Assoc 207:46–49.
150. Garcia A and Fox JG. 2003. The rabbit as a new reservoir host of enterohemorrhagic *Escherichia coli*. Emerg Infect Dis 9:1592–1597.
151. Gerner-Smidt P, Kincaid J, Kubota K, Hise K, Hunter SB, Fair MA, Norton D, Woo-Ming A, Kurzynski T, Sotir MJ, Head M, Holt K, and Swaminathan B. 2005. Molecular surveillance of Shiga toxigenic *Escherichia coli* O157 by PulseNet USA. J Food Prot 68:1926–1931.
152. Gilbert M, Srour L, Paccagnella A, MacDougall L, Fung J, Nelson E, and Fyfe M. 2005. An outbreak of *Escherichia coli* O157:H7 associated withg a children's water spray park and identified by two rounds of pulsed-field gel electrophoresis testing. Can Commun Dis Rep 31(12):133–140.
153. Goh S, Newman C, Knowles M, Bolton FJ, Hollyoak V, Richards S, Daley P, Counter D, Smith HR, and Keppie N. 2002. *E. coli* O157 phage type 21/28 outbreak in North Cumbria associated with pasteurized milk. Epidemiol Infect 129:451–457.
154. Goldwater PN and Bettelheim KA. 1995. Hemolytic uremic syndrome due to shiga-like toxin producing *Escherichia coli* O48:H21 in South Australia. Emerg Inf Dis 1(4):132–133.
155. Goldwater PN and Bettelheim KA. 1998. New perspectives on the role of *Escherichia coli* O157:H7 and other enterohemorrhagic *E. coli* serotypes in human disease. J Med Microbiol 47:1039–1045.
156. Gourmelon M, Montet MP, Lozach S, Le Mennec C, Pommepuy M, Beutin L, and Vernozy-Rozand C. 2006. First isolation of Shiga toxin 1d producing *Escherichia coli* variant strains in shellfish from coastal areas in France. J Appl Microbiol 100:85–97.
157. Greenquist MA, Drouillard JS, Sargeant JM, Depenbusch BE, Shi XR, Lechtenberg KF, and Nagaraja TG. 2005. Comparison of rectoanal mucosal swab cultures and fecal cultures for determining prevalence of *Escherichia coli* O157:H7 in feedlot cattle. Appl Environ Microbiol 71:6431–6433.
158. Grif K, Orth D, Lederer I, Berghold C, Roedl S, Mache CJ, Dierich MP, and Wurzner R. 2005. Importance of environmental transmission in cases of EHEC O157 causing hemolytic uremic syndrome. Eur J Clin Microbiol Infect Dis 24:268–271.
159. Grossmann K, Weniger B, Baljer G, Brenig B, and Wieler LH. 2005. Racing, ornamental and city pigeons carry Shiga toxin producing *Escherichia coli* (STEC) with different Shiga toxin subtypes, urging further analysis of their epidemiological role in the spread of STEC. Berlin Munch Tierarzt Wochenschr 118:456–463.
160. Gupta A, Hunter SB, Bidol SA, Dietrich S, Kincaid J, Salehi E, Nicholson L, Genese CA, Todd-Weinstein S, Marengo L, Kimura AC, and Brooks JT. 2004. *Escherichia coli* O157 cluster evaluation. Emerg Infect Dis 10:1856–1858.
161. Hancock DD, Besser TE, Rice DH, Ebel ED, Herriott DE, and Carpenter LV. 1998. Multiple sources of *Escherichia coli* O157 in feedlots and dairy farms in the northwestern USA. Prev Vet Med 35:11–19.
162. Harakudo Y, Konuma H, Iwaki M, Kasuga F, Sugitakonishi Y, Ito Y, and Kumagai S. 1997. Potential hazard of radish sprouts as a vehicle of *Escherichia coli* O157:H7. J Food Prot 60:1125–1127.
163. Hardnett FP, Hoekstra RM, Kennedy M, Charles L, and Angulo FJ. 2004. Epidemiologic issues in study design and data analysis related to FoodNet activities. Clin Infect Dis 38:S121–S126.
164. Harrison S and Kinra S. 2004. Outbreak of *Escherichia coli* O157 associated with a busy bathing beach. Commun Dis Public Health 7:47–50.
165. Haus-Cheymol R, Espie E, Che D, Vaillant V, DeValk H, and Desenclos JC. 2006. Association between indicators of cattle density and incidence of paediatric haemolytic-uraemic syndrome (HUS) in children under 15 years of age in France between 1996 and 2001: an ecological study. Epidemiol Infect 134:712–718.
166. Heuvelink AE, Van Heerwaarden C, Zwartkruis-Nahuis JTM, Van Oosterom R, Edink K, Van Duynhoven YTHP, and De Boer E. 2002. *Escherichia coli* O157 infection associated with a petting zoo. Epidemiol Infect 129:295–302.
167. Heuvelink AE, Vandenbiggelaar FLAM, Deboer E, Herbes RG, Melchers WJG, Huisintveld JHJ, and Monnens LAH. 1998. Isolation and characterization of verocytotoxin-producing *Escherichia coli* O157 strains from Dutch cattle and sheep. J Clin Microbiol 36:878–882.
168. Heuvelink AE, Zwartkruis-Nahuis JTM, van den Biggelaar FLAM, van Leeuwen WJ, and de Boer E. 1999. Isolation and characterization of verocytotoxin-producing *Escherichia coli* O157 from slaughter pigs and poultry. Int J Food Microbiol 52:67–75.
169. Hilborn ED, Mermin JH, Mshar PA, Hadler JL, Voetsch A, Wojtkunski C, Swartz M, Mshar R, Lambert-Fair MA, Farrar JA, Glynn MK, and Slutsker L. 1999. A multistate outbreak of *Escherichia coli* O157:H7 infections associated with consumption of mesclun lettuce. Arch Intern Med 159:1758–1764.
170. Hilborn ED, Mshar PA, Fiorentino TR, Dembek ZF, Barrett TJ, Howard RT, and Cartter ML. 2000. An outbreak of

Escherichia coli O157:H7 infections and haemolytic uraemic syndrome associated with consumption of unpasteurized apple cider. Epidemiol Infect 124:31–36.
171. Hildebrand JM, Maguire HC, Holliman RE, and Kangesu E. 1996. An outbreak of *Escherichia coli* O157 infection linked to paddling pools. Commun Dis Rep Rev 6:R33–R36.
172. Honish L, Predy G, Hislop N, Chui L, Kowalewska-Grochowska K, Trottier L, Kreplin C, and Zazulak I. 2005. An outbreak of *E. coli* O157:H7 hemorrhagic colitis associated with unpasteurized gouda cheese. Can J Pub Health 96:182–184.
173. Howatt G. 2006. Origin of beef linked to Longville death is still mystery. Star Tribune. www.startribune.com/462/v-print/story/632284.html.
174. Howie H, Mukerjee A, Cowden J, Leith J, and Reid T. 2003. Investigation of an outbreak of *Escherichia coli* O157 infection caused by environmental exposure at a scout camp. Epidemiol Infect 131:1063–1069.
175. Hrudey SE, Payment P, Huck PM, Gillham RW, and Hrudey EJ. 2003. A fatal waterborne disease epidemic in Walkerton, Ontario: comparison with other waterborne outbreaks in the developed world. Water Sci Technol 47:7–14.
176. Hussein HS and Bollinger LM. 2005. Prevalence of Shiga toxin-producing *Escherichia coli* in beef. Meat Sci 71:676–689.
177. Hussein HS and Bollinger LM. 2005. Prevalence of Shiga toxin-producing *Escherichia coli* in beef cattle. J Food Prot 68:2224–2241.
178. Hussein HS and Sakuma T. 2005. Invited review: Prevalence of Shiga toxin-producing *Escherichia coli* in dairy cattle and their products. J Dairy Sci 88:450–465.
179. Hussein HS, Thran BH, and Glimp HA. 2003. Verotoxin-producing *Escherichia coli* in sheep grazing an irrigated pasture or arid rangeland forages. Exp Biol Med 228:358–364.
180. Ihekweazu c, Barlow M, Roberts S, Christensen H, Guttridge B, Lewis D, and Paynter S. 2006. Outbreak of *E. coli* O157 infection in the south west of the UK: risks from streams crossing seaside beaches. Euro Surveill 11(4):128–130.
181. Ikeda K, Ida O, Kimoto K, Takatorige T, Nakanishi N, and Tatara K. 2000. Predictors for the development of haemolytic uraemic syndrome with *Escherichia coli* O157:H7 infections: with focus on the day of illness. Epidemiol Infect 124:343–349.
182. Illinois Dept. of Health. 1999. Petersburg *E. coli* outbreak traced to beef. www.idph.state.il.us/public/press99/ecolinews.htm
183. Islam M, Doyle MP, Phatak SC, Millner P, and Jiang XP. 2005. Survival of *Escherichia coli* O157:H7 in soil and on carrots and onions grown in fields treated with contaminated manure composts or irrigation water. Food Microbiol 22:63–70.
184. Jackson LA, Keene WE, McAnulty JM, Alexander ER, Diermayer M, Davis MA, Hedberg K, Boase J, Barrett TJ, Samadpour M, and Fleming DW. 2000. Where's the beef? The role of cross-contamination in 4 chain restaurant-associated outbreaks of *Escherichia coli* O157:H7 in the Pacific northwest. Arch Intern Med 160:2380–2385.
185. Jackson SG, Goodbrand RB, Johnson RP, Odorico VG, Alves D, Rahn K, Wilson JB, Welch MK, and Khakhria R. 1998. *Escherichia coli* O157:H7 diarrhoea associated with well water and infected cattle on an Ontario farm. Epidemiol Infect 120:17–20.
186. Janisiewicz WJ, Conway WS, Brown MW, Sapers GM, Fratamico P, and Buchanan RL. 1999. Fate of *Escherichia coli* O157:H7 on fresh-cut apple tissue and its potential for transmission by fruit flies. Appl Environ Microbiol 65:1–5.
187. Jay MT, Garrett V, Mohle-Boetani JC, Barros M, Farrar JA, Rios R, Abbott S, Sowadsky R, Komatsu K, Mandrell R, Sobel J, and Werner SB. 2004. A multistate outbreak of *Escherichia coli* O157:H7 infection linked to consumption of beef tacos at a fast-food restaurant chain. Clin Infect Dis 39:1–7.
188. Jensen C, Ethelberg S, Gervelmeyer A, Nielsen EM, Olsen KEP, and Molbak K. 2006. First general outbreak of verocytotoxin-producing *Escherichia coli* O157 in Denmark. Euro Surveill 11(2):55–58.
189. Johannessen GS, Bengtsson GB, Heier BT, Bredholt S, Wasteson Y, and Rorvik LM. 2005. Potential uptake of *Escherichia coli* O157:H7 from organic manure into crisphead lettuce. Appl Environ Microbiol 71:2221–2225.
190. Johannessen GS, Froseth RB, Solemdal L, Jarp J, Wasteson Y, and Rorvik LM. 2004. Influence of bovine manure as fertilizer on the bacteriological quality of organic Iceberg lettuce. J Appl Microbiol 96:787–794.
191. Johnsen G, Wasteson Y, Heir E, Berget OI, and Herikstad H. 2001. *Escherichia coli* O157 H7 in faeces from cattle, sheep and pigs in the southwest part of Norway during 1998 and 1999. Int J Food Microbiol 65:193–200.
192. Johnson RP, Clarke RC, Wilson JB, Read SC, Rahn K, Renwick SA, Sandhu KA, Alves D, Karmali MA, Lior H, McEwen SA, Spika JS, and Gyles CL. 1996. Growing concerns and recent outbreaks involving non-O157:H7 serotypes of verotoxigenic *Escherichia coli*. J Food Prot 59:1112–1122.
193. Jones IG and Roworth M. 1996. An outbreak of *Escherichia coli* O157 and campylobacteriosis associated with contamination of a drinking water supply. Public Health 110:277–282.
194. Kang SJ, Ryu SJ, Chae JS, Eo SK, Woo GJ, and Lee JH. 2004. Occurrence and characteristics of enterohemorrhagic *Escherichia coli* O157 in calves associated with diarrhoea. Vet Microbiol 98:323–328.
195. Karch H, Russmann H, Schmidt H, Schwarzkopf A, and Heesemann J. 1995. Long-term shedding and clonal turnover of enterohemorrhagic *Escherichia coli* O157 in diarrheal diseases. J Clin Microbiol 33:1602–1605.
196. Karmali MA. 2005. Use of comparative genomics as a tool to assess the clinical and public health significance of emerging Shiga toxin-producing *Escherichia coli* serotypes. Meat Sci 71:62–71.
197. Kassenborg HD, Hedberg CW, Hoekstra M, Evans MC, Chin AE, Marcus R, Vugia DJ, Smith K, Ahuja SD, Slutsker L, and Griffin PM. 2004. Farm visits and undercooked hamburgers as major risk factors for sporadic *Escherichia coli* O157:H7 infection: Data from a case-control study in 5 FoodNet sites. Clin Infect Dis 38:S271–S278.
198. Kaufmann M, Zweifel C, Blanco M, Blanco JE, Blanco J, Beutin L, and Stephan R. 2006. *Escherichia coli* O157 and non-O157 Shiga toxin-producing *Escherichia coli* in fecal samples of finished pigs at slaughter in Switzerland. J Food Prot 69:260–266.
199. Keen JE, Wittum JE, Dunn JR, Bono JL, and Durso LM. 2006. Shiga-toxigenic *Escherichia coli* O157 in agricultural fair livestock, United States. Emerg Infect Dis 12:780–786.
200. Keene WE, Hedberg K, Herriott DE./Hancock DD, McKay RW, Barrett TJ, and Fleming DW. 1997. Prolonged outbreak of *Escherichia coli* O157:H7 infections caused by commercially distributed raw milk. J Infect Dis 176:815–818.
201. Keene WE, McAnulty JM, Hoesly FC, Williams LP Jr, Hedberg K, Oxman GL, Barrett TJ, Pfaller MA, and Fleming DW. 1994. A swimming-associated outbreak of hemorrhagic colitis caused by *Escherichia coli* O157:H7 and Shigella sonnei. N Engl J Med 331:579–584.
202. Keene WE, Sazie E, Kok J, Rice DH, Hancock DD, Balan VK, Zhao T, and Doyle MP. 1997. An outbreak of *Escherichia coli* O157:H7 infections traced to jerky made from deer meat. J Am Med Assoc 277:1229–1231.

203. Kijima-Tanaka M, Ishihara K, Kojima A, Morioka A, Nagata R, Kawanishi M, Nakazawa M, Tamura Y, and Takahashi T. 2005. A national surveillance of Shiga toxin-producing *Escherichia coli* in food-producing animals in Japan. J Vet Med Ser B 52:230–237.
204. Kobayashi M, Sasaki T, Saito N, Tamura K, Suzuki K, Watanabe H, and Agui N. 1999. Houseflies: Not simple mechanical vectors of enterohemorrhagic *Escherichia coli* O157:H7. Am J Trop Med Hyg 61:625–629.
205. Kohli HS, Chaudhuri AK, Todd WT, Mitchell AA, and Liddell KG. 1994. A severe outbreak of *E. coli* O157 in two psychogeriatric wards. J Pub Health Med 16:11–15.
206. Krishnan C, Fitzgerald VA, Dakin SJ, and Behme RJ. 1987. Laboratory investigation of outbreak of hemorrhagic colitis caused by *Escherichia coli* O157:H7. J Clin Microbiol 25:1043–1047.
207. Kudaka J, Asato R, Itokazu K, Nakamura M, Taira K, Kuniyosi H, Kinjo Y, Terajima J, Watanabe H, Swaminathan B, Braden CR, and Dunn JR. 2005. *Escherichia coli* O157:H7 infections associated with ground beef from a U.S. military installation—Okinawa, Japan, February 2004. Morbid Mortal Weekly Rep 54:40–42.
208. Kudva IT, Hatfield PG, and Hovde CJ. 1995. Effect of diet on the shedding of *Escherichia coli* O157:H7 in a sheep model. Appl Environ Microbiol 61:1363–1370.
209. Kudva IT, Hatfield PG, and Hovde CJ. 1997. Characterization of *Escherichia coli* O157:H7 and other shiga toxin-producing *E. coli* serotypes isolated from sheep. J Clin Microbiol 35:892–899.
210. La Ragione RM, Ahmed NMY, Best A, Clifford D, Weyer U, Cooley WA, Johnson L, Pearson GR, and Woodward MJ. 2005. Colonization of 8-week-old conventionally reared goats by *Escherichia coli* O157:H7 after oral inoculation. J Med Microbiol 54:485–492.
211. Lahti E, Hirvela-Koski V, and Honkanen-Buzalski T. 2001. Occurrence of *Escherichia coli* O157 in reindeer (Rangifer tarandus). Vet Rec 148:633–634.
212. Laine ES, Scheftel JM, Boxrud DJ, Vought KJ, Danila RN, Elfering KM, and Smith KE. 2005. Outbreak of *Escherichia coli* O157:H7 infections associated with nonintact blade-tenderized frozen steaks sold by door-to-door vendors. J Food Prot 68:1198–1202.
213. Law D. 2000. The history and evolution of *Escherichia coli* O157 and other Shiga toxin-producing *E. coli*. World J Microbiol Biotechnol 16:701–709.
214. Leclercq A and Mahillon J. 2003. Farmed rabbits and ducks as vectors for VTEC O157:H7. Vet Rec 152:723–724.
215. Lee SH, Levy DA, Craun GF, Beach MJ, and Calderon RL. 2002. Surveillance for waterborne disease outbreaks—United States, 1999–2000. Morbid Mortal Weekly Rep 519SS08):1–28.
216. LeJeune JT, Besser TE, and Hancock DD. 2001. Cattle water troughs as reservoirs of *Escherichia coli* O157. Appl Environ Microbiol 67:3053–3057.
217. LeJeune JT, Besser TE, Rice DH, Berg JL, Stilborn RP, and Hancock DD. 2004. Longitudinal study of fecal shedding of *Escherichia coli* O157:H7 in feedlot cattle: Predominance and persistence of specific clonal types despite massive cattle population turnover. Appl Environ Microbiol 70:377–384.
218. Lemunier M, Francou C, Rousseaux S, Houot S, Dantigny P, Piveteau P, and Guzzo J. 2005. Long-term survival of pathogenic and sanitation indicator bacteria in experimental biowaste composts. Appl Environ Microbiol 71:5779–5786.
219. Lerman Y, Cohen D, Gluck A, Ohad E, and Sechter I. 1992. A cluster of cases of *Escherichia coli* O157 infection in a day-care center in a communal settlement (kibbutz) in Israel. J Clin Microbiol 30:520–521.
220. Levy DA, Bens MS, Craun GF, Calderon RL, and Herwaldt BL. 1998. Surveillance for waterborne disease outbreaks—United States, 1995–1996. Morbid Mortal Weekly Rep 47(SS05). 80 p.
221. Licence K, Oates KR, Synge BA, and Reid TMS. 2001. An outbreak of *E. coli* O157 infection with evidence of spread from animals to man through contamination of a private water supply. Epidemiol Infect 126:135–138.
222. Liptakova A, Siegfried L, Rosocha J, Podracka L, Bogyiova E, and Kotulova D. 2004. A family outbreak of haemolytic uraemic syndrome and haemorrhagic colitis caused by verocytotoxigenic *Escherichia coli* O157 from unpasteurised cow's milk in Slovakia. Clin Microbiol Infect 10:576–578.
223. Low JC, McKendrick IJ, McKechnie C, Fenlon D, Naylor SW, Currie C, Smith DGE, Allison L, and Gally DL. 2005. Rectal carriage of enterohaemorrhagic *Escherichia coli* O157 in slaughtered cattle. Appl Environ Microbiol 71:93–97.
224. Ludwig K, Ruder H, Bitzan M, Zimmermann S, and Karch H. 1997. Outbreak of *Escherichia coli* O157:H7 infection in a large family. Eur J Clin Microbiol Infect Dis 16:238–241.
225. MacDonald C, Drew J, Carlson R, Dzogan S, Tataryn S, Macdonald A, Ali A, Amhed R, Easy R, Clark C, and Rodgers F. 2000. Outbreak of *Escherichia coli* O157:H7 leading to the recall of retail ground beef—Winnipeg, Manitoba, May 1999. Can Commun Dis Rep 26(13)109–111.
226. MacDonald DM, Fyfe M, Paccagnella A, Trinidad A, Louie K, and Patrick D. 2004. *Escherichia coli* O157:H7 outbreak linked to salami, British Columbia, Canada, 1999. Epidemiol Infect 132:283–289.
227. MacRae M, Hamilton C, Strachan NJC, Wright S, and Ogden ID. 2005. The detection of Cryptosporidium parvum and *Escherichia coli* O157 in UK bivalve shellfish. J Microbiol Meth 60:395–401.
228. Mannix M. 2005. Large *E. coli* O157 outbreak in Ireland, October–November 2005. Euro Surveill 10(12):E051222.3 www.eurosurveillance.org/ew/2005/051222.asp#3
229. Marsh J, MacLeod AF, Hanson M F, Emmanuel FX, Frost JA, and Thomas A. 1992. A restaurant-associated outbreak of *E. coli* O157 infection. J Pub Health Med 14:78–83.
230. Martin ML, Shipman LD, Wells JG, Potter ME, Hedberg K, Wachsmuth IK, Tauxe RV, Davis JP, Arnoldi J, and Tilleli J. 1986. Isolation of *Escherichia coli* O157:H7 from dairy cattle associated with two cases of haemolytic uraemic syndrome. Lancet 2(8514):1043.
231. Maruzumi M, Morita M, Matsuoka Y, Uekawa A, Nakamura T, and Fuji K. 2005. Mass food poisoning caused by beef offal contaminated by *Escherichia coli* O157. Jap J Infect Dis 58:397.
232. Matthews L, Low JC, Gally DL, Pearce MC, Mellor DJ, Heesterbeek JAP, Chase-Topping M, Naylor SW, Shaw DJ, Reid SWJ, Gunn GJ, and Woolhouse MEJ. 2006. Heterogeneous shedding of *Escherichia coli* O157 in cattle and its implications for control. Proc Nat Acad Sci USA 103:547–552.
233. Matthews L, McKendrick IJ, Ternent H, Gunn GJ, Synge B, and Woolhouse MEJ. 2006. Super-shedding cattle and the transmission dynamics of *Escherichia coli* O157. Epidemiol Infect 134:131–142.
234. McCall B, Strain D, Hills S, Heymer M, Bates J, Murphy D, Kelly R, and Price D. 1996. An outbreak of *Escherichia coli* O157 infection on the Gold Coast. Commun Dis Intelligence 20:236–239.
235. McCarthy TA, Barrett NL, Hadler JL, Salsbury B, Howard RT, Dingman DW, Brinkman CD, Bibb WF, and Cartter ML. 2001. Hemolytic-uremic syndrome and *Escherichia coli* O121 at a Lake in Connecticut, 1999. Pediatrics 108: NIL_9-NIL_15.
236. McCluskey BJ, Rice DH, Hancock DD, Hovde CJ, Besser TE, Gray S, and Johnson RP. 1999. Prevalence of

Escherichia coli O157 and other Shiga-toxin-producing *E. coli* in lambs at slaughter. J Vet Diagn Invest 11:563–565.

237. McDonnell RJ, Rampling A, Crook S, Cockcroft PM, Wilshaw GA, Cheasty T, and Stuart J. 1997. An outbreak of Vero cytotoxin producing *Escherichia coli* O157 infection associated with takeaway sandwiches. Commun Dis Rep Rev 7:R201–R205.
238. McDonough S, Heer F, and Shireley L. 1991. Foodborne outbreak of gastroenteritis caused by *Escherichia coli* O157:H7—North Dakota, 1990. Morbid Mortal Weekly Rep 40:265–267.
239. McIntyre L, Fung J, Paccagnella A, Issac-Renton J, Rockwell F, Emerson B, and Preston T. 2002. *Escherichia coli* O157 outbreak associated with the ingestion of unpasteurized goat's milk in British Columbia, 2001. Can Commun Dis Rep 28(1):6–8.
240. Mellmann A, Bielaszewska M, Zimmerhackl LB, Prager R, Harmsen D, Tschape H, and Karch H. 2005. Enterohemorrhagic *Escherichia coli* in human infection: In vivo evolution of a bacterial pathogen. Clin Infect Dis 41:785–792.
241. Michino H, Araki K, Minami S, Takaya S, Sakai N, Miyazaki M, Ono A, and Yanagawa H. 1999. Massive outbreak of *Escherichia coli* O157:H7 infection in schoolchildren in Sakai City, Japan, associated with consumption of white radish sprouts. Am J Epidemiol 150:787–796.
242. Milne LM, Plom A, Strudley I, Pritchard GC, Crooks R, Hall M, Duckworth G, Seng C, Susman MD, Kearney J, Wiggins RJ, Moulsdale M, Cheasty T, and Willshaw GA. 1999. *Escherichia coli* O157 incident associated with a farm open to members of the public. Commun Dis Public Health 2:22–26.
243. Misselwitz J, Karch H, Bielazewska M, John U, Ringelmann F, Ronnefarth G, and Patzer L. 2003. Cluster of hemolytic-uremic syndrome caused by Shiga toxin-producing *Escherichia coli* O26:H11. Pediatr Infect Dis J 22:349–354.
244. Mohle-Boetani JC, Farrar JA, Werner SB, Minassian D, Bryant R, Abbott S, Slutsker L, and Vugia DJ. 2001. *Escherichia coli* O157 and Salmonella infections associated with sprouts in California, 1996–1998. Ann Int Med 135:239–247.
245. Moore K, Damrow T, Abbott DO, and Jankowski S. 1995. Outbreak of acute gastroenteritis attributable to *Escherichia coli* serotype O104:H21—Helena, Montana, 1994. Morbid Mortal Weekly Rep 44:501–503.
246. Morabito S, Dell'Omo G, Agrimi U, Schmidt H, Karch H, Cheasty T, and Caprioli A. 2001. Detection and characterization of Shiga toxin-producing *Escherichia coli* in feral pigeons. Vet Microbiol 82:275–283.
247. Morgan AK, Piispanen, Humphreys, and Murphy D. 2005. A cluster of cases of haemolytic uraemic syndrome in north Queensland associated with a novel Shiga-like toxin-producing *Escherichia coli*. Commun Dis Intelligence 29:191–193.
248. Morgan D, Newman CP, Hutchinson DN, Walker AM, Rowe B, and Majid F. 1993. Verotoxin producing *Escherichia coli* O157 infections associated with consumption of yoghurt. Epidemiol Infect 111:181–187.
249. Moriya K, Fujibayashi T, Yoshihara T, Matsuda A, Sumi N, Umezaki N, Kurahashi H, Agui N, Wada A, and Watanabe H. 1999. Verotoxin-producing *Escherichia coli* O157:H7 carried by the housefly in Japan. Med Vet Entomol 13:214–216.
250. Mudgett CC, Ruden R, and Austin CC. 1998. A beach-associated outbreak of *Escherichia coli* O157:H7. J Environ Health 60:7–13.
251. Mukherjee A, Cho S, Scheftel J, Jawahir S, Smith K, and Diez-Gonzalez. 2006. Soil survival of *Escherichia coli* O157:H7 acquired by a child from garden soil recently fertilized with cattle manure. J Appl Microbiol 101:429–436.
252. Murphy J. 2005. Minnesota finds *E. coli* in lettuce bags. www.producenews.com/storydetail.cfm?ID=5449.
253. Nagano H, Hirochi T, Fujita K, Wakamori Y, Takeshi K, and Yano S. 2004. Phenotypic and genotypic characterization of beta-D-glucuronidase-positive Shiga toxin-producing *Escherichia coli* O157:H7 isolates from deer. J Med Microbiol 53:1037–1043.
254. Naugle AL, Holt KG, Levine P, and Eckel R. 2005. Food safety and inspection service regulatory testing program for *Escherichia coli* O157:H17 in raw ground beef. J Food Prot 68:462–468.
255. Naylor SW, Low JC, Besser TE, Mahajan A, Gunn GJ, Pearce MC, McKendrick IJ, Smith DG, and Gally DL. 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. Infect Immun 71:1505–1512.
256. Nielsen EM and Scheutz F. 2002. Characterisation of *Escherichia coli* O157 isolates from Danish cattle and human patients by genotyping and presence and variants of virulence genes. Vet Microbiol 88:259–273.
257. Notario R, Fain JC, Prado V, Rios M, Borda N, and Gambande T. 2000. Animal reservoir and genotipic characterization of enterohemorrhagic *Escherichia coli* (EHEC) in Argentina. Rev Med Chile 128:1335–1341.
258. Novello A. 1999. Outbreak of *Escherichia coli* O157:H7 and Campylobacter among attendees of the Washington County Fair-New York, 1999. Morbid Mortal Weekly Rep 48:803–805.
259. Novotna R, Alexa P, Hamrik J, Madanat A, Smola J, and Cizek A. 2005. Isolation and characterization Shiga toxin-producing *Escherichia coli* from sheep and goats in Jordan with evidence of multiresistant serotype O157:H7. Vet Medicina 50:111–118.
260. NY State Dept Health. 2000. Health Commissioner releases *E. coli* outbreak report. www.health.state.ny.us/press/releases/2000/ecoli.htm
261. O'Brien SJ, Gillespie IA, Sivanesan MA, Elson R, Hughes C, and Adak GK. 2006. Publication bias in foodborne outbreaks of infectious intestinal disease and its implications foe evidence-based food policy. England and Wales 1992–2003. Epidemiol Infect 134:667–674.
262. O'Brien SJ, Murdoch PS, Riley AH, King I, Barr M, Murdoch S, Greig A, Main R, Reilly WJ, and Thomson-Carter FM. 2001. A foodborne outbreak of Vero cytotoxin-producing *Escherichia coli* O157:H-phage type 8 in hospital. J Hosp Infect 49:167–172.
263. O'Flynn G, Ross RP, Fitzgerald GF, and Coffey A. 2004. Evaluation of a cocktail of three bacteriophages for biocontrol of *Escherichia coli* O157:H7. Appl Environ Microbiol 70:3417–3424.
264. Ogden ID, Hepburn NF, MacRae M, Strachan NJC, Fenlon DR, Rusbridge SM, and Pennington TH. 2002. Long-term survival of *Escherichia coli* O157 on pasture following an outbreak associated with sheep at a scout camp. Lett Appl Microbiol 34:100–104.
265. Ogden ID, MacRae M, and Strachan NJC. 2005. Concentration and prevalence of *Escherichia coli* O157 in sheep faeces at pasture in Scotland. J Appl Microbiol 98:646–651.
266. Olsen SJ, Miller G, Breuer T, Kennedy M, Higgins C, Walford J, McKee G, Fox K, Bibb W, and Mead P. 2002. A waterborne outbreak of *Escherichia coli* O157:H7 infections and hemolytic uremic syndrome: Implications for rural water systems. Emerg Infect Dis 8:370–375.
267. Omisakin F, MacRae M, Ogden ID, and Strachan NJC. 2003. Concentration and prevalence of *Escherichia coli*

O157 in cattle feces at slaughter. Appl Environ Microbiol 69:2444–2447.
268. Orr P, Lorencz B, Brown R, Kielly R, Tan B, Holton D, Clugstone H, Lugtig L, Pim C, Macdonald S, Hammond G, Moffatt M, Spika J, Manuel D, Winther W, Milley D, Lior H, and Sinuff N. 1994. An outbreak of diarrhea due to verotoxin-producing *Escherichia coli* in the Canadian Northwest Territories. Scan J Infect Dis 26:675–684.
269. Osek J. 2004. Phenotypic and genotypic characterization of *Escherichia coli* O157 strains isolated from humans, cattle and pigs. Vet Medicina 49:317–326.
270. Ostroff SM, Griffin PM, Tauxe RV, Shipman LD, Greene KD, Wells JG, Lewis JH, Blake PA, and Kobayashi JM. 1990. A statewide outbreak of *Escherichia coli* O157:H7 infections in Washington State. Am J Epidemiol 132:239–247.
271. Paton AW, Ratcliff RM, Doyle RM, Seymour-Murray J, Davos D, Lanser JA, and Paton JC. 1996. Molecular microbiological investigation of an outbreak of hemolytic-uremic syndrome caused by dry fermented sausage contaminated with shiga-like toxin-producing *Escherichia coli*. J Clin Microbiol 34:1622–1627.
272. Paunio M, Pebody R, Keskimaki M, Kokki M, Ruutu P, Oinonen S, Vuotari V, Siitonen A, Lahti E, and Leinikki P. 1999. Swimming-associated outbreak of *Escherichia coli* O157: H7. Epidemiol Infect 122:1–5.
273. Pavia AT, Nichols CR, Green DP, Tauxe RV, Mottice S, Greene KD, Wells JG, Siegler RL, Brewer ED, Hannon D, and Blake PA. 1990. Hemolytic-uremic syndrome during an outbreak of *Escherichia coli* O157:H7 infections in institutions for mentally retarded persons: clinical and epidemiologic observations. J Pediatr 116:544–551.
274. Payne CJI, Petrovic M, Roberts RJ, Paul A, Linnane E, Walker M, Kirby D, Burgess A, Smith RMM, Cheasty T, Willshaw G, and Salmon RL. 2003. Vero cytotoxin-producing *Escherichia coli* O157 gastroenteritis in farm visitors, North Wales. Emerg Infect Dis 9:526–530.
275. Pebody RG, Furtado C, Rojas A, McCarthy N, Nylen G, Ruutu P, Leino T, Chalmers R, de Jong B, Donnelly M, Fisher I, Gilham C, Graverson L, Cheasty T, Willshaw G, Navarro M, Salmon R, Leinikki P, Wall P, and Bartlett C. 1999. An international outbreak of Vero cytotoxin-producing *Escherichia coli* O157 infection amongst tourists; a challenge for the European infectious disease surveillance network. Epidemiol Infect 123:217–223.
276. Pedersen K, Clark L, Andelt WF, and Salman MD. 2006. Prevalence of shiga toxin-producing *Escherichia coli* and *Salmonella enterica* in rock pigeons captured in fort Collins, Colorado. J Wildlife Dis 42:46–55.
277. Pennington H. 1998. Factors involved in recent outbreaks of *Escherichia coli* O157:H7 in Scotland and recommendations for its control. J Food Safety 18:383–391.
278. Pennington TH. 2000. VTEC: lessons learned from British outbreaks. J Appl Microbiol 88:90S–98S.
279. Phebus RK, Nutsch AL, Schafer DE, Wilson RC, Riemann MJ, Leising JD, Kastner CL, Wolf JR, and Prasai RK. 1997. Comparison of steam pasteurization and other methods for reduction of pathogens on surfaces of freshly slaughtered beef. J Food Prot 60:476–484.
280. Pichner R, Sander A, Steinruck H, and Gareis M. 2005. Occurrence of Salmonella spp. and shigatoxin-producing *Escherichia coli* (STEC) in horse faeces and horse meat products. Berlin Munch Tierarzt Wochenschr 118:321–325.
281. Pilipcinec E, Tkacikova L, Naas HT, Cabadaj R, and Mikula I. 1999. Isolation of verotoxigenic *Escherichia coli* O157 from poultry. Folia Microbiol 44:455–456.
282. Pinto C. 2006. *E. coli* sends four toddlers to hospital. http://www.hendersonvillestarnews.com/apps/pbcs.dll/article?AID=/20060628/NEWS07/60628016 June 29, 2006.
283. Pollett GL and Warshawsky B. 1999. *E. coli* O157:H7 investigation complete. Middlesex-London Health Unit. www.healthunit.com/index.asp?mode=article&lang=english&articleID=11329&rank=313
284. Potter AA, Klashinsky S, Li Y, Frey E, Townsend H, Rogan D, Erickson G, Hinkley S, Klopfenstein T, Moxley RA, Smith DR, and Finlay BB. 2004. Decreased shedding of *Escherichia coli* O157:H7 by cattle following vaccination with type III secreted proteins. Vaccine 22:362–369.
285. Preston M, Borczyk A, Davidson R, McGeer A, Bertoli J, Harris S, Thususka J, Goldman C, Green K, Low D, Proctor P, Johnson W, and Khakhria R. 1997. Hospital outbreak of *Escherichia coli* O157:H7 associated with a rare phage type—Ontario. Can Commun Dis Rep 23(5):71–73.
286. Pritchard GC, Williamson S, Carson T, Bailey JR, Warner L, Willshaw G, and Cheasty T. 2001. Wild rabbits—novel vector for verocytotoxigenic *Escherichia coli* O157. Vet Rec 149:567.
287. Pritchard GC, Willshaw GA, Bailey JR, Carson T, and Cheasty T. 2000. Verocytotoxin-producing *Escherichia coli* O157 on a farm open to the public: outbreak investigation and longitudinal bacteriological study. Vet Rec 147:259–264.
288. Rabatsky-Ehr T, Dingman D, Marcus R, Howard R, Kinney A, and Mshar P. 2002. Deer meat as the source for a sporadic case of *Escherichia coli* O157:H7 infection, Connecticut. Emerg Infect Dis 8:525–527.
289. Rahn K, Renwick SA, Johnson RP, Wilson JB, Clarke RC, Alves D, McEwen S, Lior H, and Spika J. 1997. Persistence of *Escherichia coli* O157:H7 in dairy cattle and the dairy farm environment. Epidemiol Infect 119:251–259.
290. Rajpura A, Lamden K, Forster S, Clarke S, Cheesbrough J, Gornall S, and Waterworth S. 2003. Large outbreak of infection with *Escherichia coli* O157 PT21/28 in Eccleston, Lancashire, due to cross contamination at a butcher's counter. Commun Disease Public Health 6:279–284.
291. Rangel JM, Sparling PH, Crowe C, Griffin PM, and Swerdlow DL. 2005. Epidemiology of *Escherichia coli* O157:H7 outbreaks, United States, 1982–2002. Emerg Infect Dis 11:603–609.
292. Ravva SV, Sarreal CZ, Duffy B, and Stanker LH. 2006. Survival of *Escherichia coli* O157:H7 in wastewater from dairy lagoons. J Appl Microbiol 101(4):891–902.
293. Reida P, Wolff M, Pohls HW, Kuhlmann W, Lehmacher A, Aleksic S, Karch H, and Bockemuhl J. 1994. An outbreak due to enterohaemorrhagic *Escherichia coli* O157:H7 in a children day care centre characterized by person-to-person transmission and environmental contamination. Int J Med Microbiol Virol Parasitol Infect Dis 281:534–43.
294. Reiss G, Kunz P, Koin D, and Keeffe EB. 2006. *Escherichia coli* O157:H7 infection in nursing homes: Review of literature and report of recent outbreak. J Am Geriatr Soc 54:680–684.
295. Renter DG, Morris JG, Sargeant JM, Hungerford LL, Berezowski J, Ngo T, Williams K, and Acheson DWK. 2005. Prevalence, risk factors, O serogroups, and virulence profiles of Shiga toxin-producing bacteria from cattle production environments. J Food Prot 68:1556–1565.
296. Renter DG, Sargeant JM, Hygnstorm SE, Hoffman JD, and Gillespie JR. 2001. *Escherichia coli* O157:H7 in free-ranging deer in Nebraska. J Wildlife Dis 37:755–760.
297. Renter DG, Sargeant JM, Oberst RD, and Samadpour M. 2003. Diversity, frequency, and persistence of *Escherichia coli* O157 strains from range cattle environments. Appl Environ Microbiol 69:542–547.
298. Rice DH, Hancock DD, and Besser IE. 2003. Faecal culture of wild animals for *Escherichia coli* O157:H7. Vet Rec 152:82–83.
299. Riley LW, Remis RS, Helgerson SD, McGee HB, Wells JG, Davis BR, Hebert RJ, Olcott ES, Johnson LM, Hargrett NT,

Blake PA, and Cohen ML. 1983. Hemorrhagic colitis associated with a rare *Escherichia coli* serotype. N Engl J Med 308:681–685.

300. Roberts CL, Mshar PA, Cartter ML, Hadler JL, Sosin DM, Hayes PS, and Barrett TJ. 1995. The role of heightened surveillance in an outbreak of *Escherichia coli* O157:H7. Epidemiol Infect 115:447–454.
301. Roberts JA, Upton PA, and Azene G. 2000. *Escherichia coli* O157: H7; an economic assessment of an outbreak. J Pub Health Med 22:99–107.
302. Rodrigue DC, Mast EE, Greene KD, Davis JP, Hutchinson MA, Wells JG, Barrett TJ, and Griffin PM. 1995. A university outbreak of *Escherichia coli* O157:H7 infections associated with roast beef and an unusually benign clinical course. J Infect Dis 172:1122–1125.
303. Ryan CA, Tauxe RV, Hosek GW, Wells JG, Stoesz PA, McFadden HW Jr, Smith PW, Wright GF, and Blake PA. 1986. *Escherichia coli* O157:H7 diarrhea in a nursing home: clinical, epidemiological, and pathological findings. J Infect Dis 154:631–638.
304. Safdar N, Said A, Gangnon RE, and Maki DG. 2002. Risk of hemolytic uremic syndrome after antibiotic treatment of *Escherichia coli* O157:H7 enteritis—A meta-analysis. J Am Med Assoc 288:996–1001.
305. Salmon R. 2005. Outbreak of verotoxin producing *E. coli* O157 infections involving over forty schools in south Wales, September, 2005. Euro Surveill 10(10):E051006.1 www.eurosurveillance.org/ew/2005/051006.asp#1
306. Samadpour N, Stewart J, Steingart K, Addy C, Louderback J, McGinn M, Ellington J, and Newman T. 2002. Laboratory investigation of an *E. coli* O157:H7 outbreak associated with swimming in Battle Ground Lake, Vancouver, Washington. J Environ Health 64:16–20.
307. Sargeant JM, Hafer DJ, Gillespie JR, Oberst RD, and Flood SJA. 1999. Prevalence of *Escherichia coli* O157:H7 in white-tailed deer sharing rangeland with cattle. J Am Vet Med Assoc 215:792–794.
308. Sasaki T, Kobayashi M, and Agui N. 2000. Epidemiological potential of excretion and regurgitation by Musca domestica (Diptera: Muscidae) in the dissemination of *Escherichia coli* O157:H7 to food. J Med Entomol 37:945–949.
309. Scaife HR, Cowan D, Finney J, Kinghorn-Perry SF, and Crook B. 2006. Wild rabbits (*Oryctolagus cuniculus*) as potential carriers of verocytotoxin-producing *Escherichia coli*. Vet Rec 159:175–178.
310. Schimmer B, Outbreak Investigation Team. 2006. Outbreak of haemolytic uraemic syndrome in Norway: update. Euro Surveill 11(4):E060406.2 www.eurosurveillance.org/ew/2006/060406.asp#2
311. Schmidt H, Scheef J, Morabito S, Caprioli A, Wieler LH, and Karch A. 2000. A new shiga toxin 2 variant (Stx2f) from *Escherichia coli* isolated from pigeons. Appl Environ Microbiol 66:1205–1208.
312. Schoeni JL and Doyle MP. 1994. Variable colonization of chickens perorally inoculated with *Escherichia coli* O157:H7 and subsequent contamination of eggs. Appl Environ Microbiol 60:2958–2962.
313. Schouten JM, van de Giessen AW, Frankena K, De Jong MCM, and Graat EAM. 2005. *Escherichia coli* O157 prevalence in Dutch poultry, pig finishing and veal herds and risk factors in Dutch veal herds. Prev Vet Med 70:1–15.
314. Shefer AM, Koo D, Werner SB, Mintz ED, Baron R, Wells JG, Barrett TJ, Ginsberg M, Bryant R, Abbott S, and Griffin PM. 1996. Cluster of *Escherichia coli* O157:H7 infections with the hemolytic-uremic syndrome and death in California—a mandate for improved surveillance. West J Med 165:15–19.
315. Sheng H, Knecht HJ, Kudva IT, and Hovde CJ. 2006. Application of bacteriophages to control intestinal *Escherichia coli* O157:H7 levels in ruminants. Appl Environ Microbiol 72:5359–5366.
316. Shere JA, Bartlett KJ, and Kaspar CW. 1998. Longitudinal study of *Escherichia coli* O157:H7 dissemination on four dairy farms in Wisconsin. Appl Environ Microbiol 64:1390–1399.
317. Shere JA, Kaspar CW, Bartlett KJ, Linden SE, Norell B, Francey S, and Schaefer DM. 2002. Shedding of *Escherichia coli* O157:H7 in dairy cattle housed in a confined environment following waterborne inoculation. Appl Environ Microbiol 68:1947–1954.
318. Shillam P, Heltzel D, Beebe J, and Hoffman R. 1997. *Escherichia coli* O157:H7 infections associated with eating a nationally distributed commercial brand of frozen ground beef patties and burgers—Colorado, 1997. Morbid Mortal Weekly Rep 46:777–778.
319. Shillam P, Woo-Ming A, Mascola L, Bagby R, Lohff C, Bidol S, Stobierski MG, Carlson C, Schaefer L, Kightlinger L, Seys S, Kubota K, Mead PS, and Kalluri P. 2002. Multistate outbreak of *Escherichia coli* O157:H7 infections associated with eating ground beef—United States, June–July 2002. Morbid Mortal Weekly Rep 51:637–639.
320. Shukla R, Slack R, George A, Cheasty T, Rowe B, and Scutter J. 1995. *Escherichia coli* O157 infection associated with a farm visitor centre. Commun Dis Rep Rev 5:R86–R90.
321. Smith DJ, Oliver CE, Caton JS, and Anderson RC. 2005. Effect of sodium [36Cl]chlorate dose on total radioactive residues and residues of parent chlorate in beef cattle. J Agr Food Chem 53(18):7352–7360.
322. Smith KE, Stenzel SA, Bender JB, Wagstrom E, Soderlund D, Leano FT, Taylor CM, Belle-Isle PA, and Danila R. 2004. Outbreaks of enteric infections caused by multiple pathogens associated with calves at a farm day camp. Pediatr Infect Dis J 23:1098–1104.
323. Smith-Palmer A, Locking M, Reilly B, and Fisher I. 2005. Cluster of *E. coli* O157 infections in Scottish tourists returning from southwest Turkey, July–August 2005. Euro Surveill 10(8):E050818.2 www.eurosurveillance.org/ew/2005/050818.asp#2
324. Solomon EB, Pang HJ, and Matthews KR. 2003. Persistence of *Escherichia coli* O157:H7 on lettuce plants following spray irrigation with contaminated water. J Food Prot 66:2198–2202.
325. Sproston EL, Macrae M, Ogden ID, Wilson MJ, and Strachan NJC. 2006. Slugs: Potential novel vectors of *Escherichia coli* O157. Appl Environ Microbiol 72:144–149.
326. Stavric S, Buchanan B, and Gleeson TM. 1993. Intestinal colonization of young chicks with *Escherichia coli* O157:H7 and other verotoxin-producing serotypes. J Appl Microbiol 74:557–563.
327. Stevenson J and Hanson S. 1996. Outbreak of *Escherichia coli* O157 phage type 2 infection associated with eating precooked meats. Commun Dis Rep Rev 6:R116–R118.
328. Strachan NJC, Doyle MP, Kasuga F, Rotariu O, and Ogden ID. 2005. Dose response modelling of *Escherichia coli* O157 incorporating data from foodborne and environmental outbreaks. Int J Food Microbiol 103:35–47.
329. Sugiyama A, Iwade Y, Akachi S, Nakano Y, Matsuno Y, Yano T, Yamauchi A, Nakayama O, Sakai H, Yamamoto K, Nagasaka Y, Nakano T, Ihara T, and Kamiya H. 2005. An outbreak of Shigatoxin-producing Eshcherichia coli O157:H7 in a nursery school in Mie Prefecture. Jap J Infect Dis 58:398–400.
330. Sutcliffe P, Picard L, Fortin BA, Malaviarachchi D, Hohenadel J, and O'Donnell B. 2004. *Escherichia coli* O157:H7 outbreak at a summer hockey camp, Sudbury, 2004. Can Commun Dis Rep 30(22):189–194.
331. Swerdlow DL, Woodruff BA, Brady RC, Griffin PM, Tippen S, Donnell HD Jr, Geldreich E, Payne BJ, Meyer A

Jr, Wells JG, and et al. 1992. A waterborne outbreak in Missouri of *Escherichia coli* O157:H7 associated with bloody diarrhea and death. Ann Int Med 117:812–819.
332. Szalanski AL, Owens CB, Mckay T, and Steelman CD. 2004. Detection of Campylobacter and *Escherichia coli* O157:H7 from filth flies by polymerase chain reaction. Med Vet Entomol 18:241–246.
333. Söderström A, Lindberg A, and Andersson Y. 2005. EHEC O157 outbreak in Sweden from locally produced lettuce, August–September 2005. Euro Surveill 10(9):E050922.1 www.eurosurveillance.org/ew/2005/050922.asp#1
334. Tamblyn S, deGrosbois BA, Taylor D, and Stratton J. 1999. An outbreak of *Escherichia coli* O157:H7 infection associated with unpasteurized non-commercial, custom-pressed apple cider—Ontario, 1998. Can Commun Dis Rep 25(13):113–120.
335. Teunis P, Takumi K, and Shinagawa K. 2004. Dose response for infection by *Escherichia coli* O157:H7 from outbreak data. Risk Anal 24:401–407.
336. Thoms B. 1999. Detection of verotoxin-producing *Escherichia coli* in deer's meat. Arch Lebensmittelhyg 50:52–54.
337. Tkalcic S, Zhao T, Harmon BG, Doyle MP, Brown CA, and Zhao P. 2003. Fecal shedding of enterohemorrhagic *Escherichia coli* in weaned calves following treatment with probiotic *Escherichia coli*. J Food Prot 66:1184–1189.
338. Tozzi AE, Niccolini A, Caprioli A, Luzzi I, Montini G, Zacchello G, Gianviti A, Principato F, and Rizzoni G. 1994. A community outbreak of haemolytic-uraemic syndrome in children occurring in a large area of northern italy over a period of several months. Epidemiol Infect 113:209–219.
339. Tserenpuntsag B, Chang HG, Smith PF, and Morse DL. 2005. Hemolytic uremic syndrome risk and *Escherichia coli* O157:H7. Emerg Infect Dis 11:1955–1957.
340. Turney C, Greensmith M, Shipp M, Mordhorst C, Whittingslow C, Brawley L, Koppel D, Bridges E, Davis G, Voss J, Lee R, Jay M, Abbott S, Bryant R, Reilly K, Werner SB, Barrett L, Jackson RJ, Rutherford GW, and Lior H. 1994. *Escherichia coli* O157:H7 outbreak linked to home-cooked hamburger—California, July 1993. Morbid Mortal Weekly Rep 43:213–216.
341. Tuttle J, Gomez T, Doyle MP, Wells JG, Zhao T, Tauxe RV, and Griffin PM. 1999. Lessons from a large outbreak of *Escherichia coli* O157:H7 infections: insights into the infectious dose and method of widespread contamination of hamburger patties. Epidemiol Infect 122:185–192.
342. Upton P and Coia JE. 1994. Outbreak of *Escherichia coli* O157 infection associated with pasteurised milk supply. Lancet 344:1015.
343. Valcour JE, Michel P, McEwen SA, and Wilson JB. 2002. Associations between indicators of livestock farming intensity and incidence of human shiga toxin-producing *Escherichia coli* infection. Emerg Infect Dis 8:252–257.
344. Vanselow BA, Krause DO, and McSweeney CS. 2005. The Shiga toxin-producing *Escherichia coli*, their ruminant hosts, and potential on-farm interventions: a review. Austral J Agr Res 56:219–244.
345. Varma JK, Greene KD, Reller ME, DeLong SM, Trottier J, Nowicki SF, DiOrio M, Koch EM, Bannerman TL, York ST, Lambert-Fair MA, Wells JG, and Mead PS. 2003. An outbreak of *Escherichia coli* O157 infection following exposure to a contaminated building. J Am Med Assoc 290:2709–2712.
346. Vogt RL and Dippold L. 2005. *Escherichia coli* O157:H7 outbreak associated with consumption of ground beef, June–July 2002. Pub Health Rep 120:174–178.
347. Wallace JS, Cheasty T, and Jones K. 1997. Isolation of vero cytotoxin-producing *Escherichia coli* O157 from wild birds. J Appl Microbiol 82:399–404.
348. Warrner M, Kuo K, Williams L, Adam B, Langkop C, Ruden R, Francis B, and Haupt T. 1996. Lake-associated outbreak of *Escherichia coli* O157:H7—Illinois, 1995. Morbid Mortal Weekly Rep 45:437–439.
349. Wasteson Y, Arnemo JM, Johansen BK, Vold L, Mathiesen SD, Olsen MA, Wiig O, and Derocher AE. 1999. Analysis of faecal samples from wild animals for verocytotoxin producing *Escherichia coli* and *E. coli* O157. Vet Rec 144:646–647.
350. Watanabe H, Wada A, Inagaki Y, Itoh K, and Tamura K. 1996. Outbreaks of enterohaemorrhagic *Escherichia coli* O157:H7 infection by two different genotype strains in Japan, 1996. Lancet 348:831–832.
351. Watanabe Y, Ozasa K, Mermin JH, Griffin PM, Masuda K, Imashuku S, and Sawada T. 1999. Factory outbreak of *Escherichia coli* O157:H7 infection in Japan. Emerg Infect Dis 5:424–428.
352. Welinder-Olsson C, Stenqvist K, Badenfors M, Brandberg A, Florén K, Holm M, Holmberg L, Kjellin E, Marild S, Studahl A, and Kaijser B. 2004. EHEC outbreak among staff at a children's hospital—use of PCR for verocytotoxin detection and PFGE for epidemiological investigation. Epidemiol Infect 132:43–49.
353. Wells JG, Davis B R, Wachsmuth IK, Riley LW, Remis RS, Sokolow R, and Morris GK. 1983. Laboratory investigation of hemorrhagic colitis outbreaks associated with a rare *Escherichia coli* serotype. J Clin Microbiol 18:512–520.
354. Werber D, Fruth A, Liesegang A, Littmann M, Buchholz U, Prager R, Karch H, Breuer T, Tschape H, and Ammon A. 2002. A multistate outbreak of Shiga toxin-producing *Escherichia coli* O26:H11 infections in Germany, detected by molecular subtyping surveillance. J Infect Dis 186:419–422.
355. Wick LM, Qi WH, Lacher DW, and Whittam TS. 2005. Evolution of genomic content in the stepwise emergence of *Escherichia coli* O157:H7. J Bacteriol 187:1783–1791.
356. Williams AP, Avery LM, Killham K, and Jones DL. 2005. Persistence of *Escherichia coli* O157 on farm surfaces under different environmental conditions. J Appl Microbiol 98:1075–1083.
357. Williams LD, Hamilton PS, Wilson BW, and Estock MD. 1997. An outbreak of *Escherichia coli* O157:H7 - involving long term shedding and person-to-person transmission in a child care center. J Environ Health 59:9–14.
358. Williams RC, Isaacs S, Decou ML, Richardson EA, Buffett MC, Slinger RW, Brodsky MH, Ciebin BW, Ellis A, and Hockin A. 2000. Illness outbreak associated with *Escherichia coli* O157:H7 in Genoa salami. Can Med Assoc J 162:1409–1413.
359. Willshaw GA, Smith HR, Cheasty T, Wall PG, and Rowe B. 1997. Vero cytotoxin-producing *Escherichia coli* O157 outbreaks in England and Wales, 1995: phenotypic methods and genotypic subtyping. Emerg Infect Dis 3:561–565.
360. Willshaw GA, Thirlwell J, Jones AP, Parry S, Salmon RL, and Hickey M. 1994. Vero cytotoxin-producing *Escherichia coli* O157 in beefburgers linked to an outbreak of diarrhoea, haemorrhagic colitis and haemolytic uraemic syndrome in Britain. Lett Appl Microbiol 19:304–307.
361. Wilson J, Spika J, Clarke R, McEwen S, Johnson R, Rahn K, Renwick S, Karmali M, Lior H, Alves D, Gyles C, and Sandhu K. 1998. Verocytotoxigenic *Escherichia coli* infection in dairy farm families. Can Commun Dis Rep 24(3).
362. Xu JG, Liu QY, Jing HQ, Pang B, Yang JC, Zhao GF, and Li HW. 2003. Isolation of *Escherichia coli* O157:H7 from dung beetles Catharsius molossus. Microbial Immunol 47:45–49.
363. Yamasaki S and Takeda Y. 1997. Enterohemorrhagic *Escherichia coli* O157:H7 episode in Japan with a

perspective on vero toxins (shiga-like toxins). J Toxicol Toxin Rev 16:229–240.

364. Yoder JS, Blackburn BG, Craun GF, Hill V, Levy DA, Chen N, Lee SH, and Calderon RL Beach MJ. 2004. Surveillance for waterborne disease outbreaks associated with recreational water—United States, 2001–2002. Morbid Mortal Weekly Rep 53(SS08):1–22.
365. Younts-Dahl SP, Osborn GD, Galyean ML, Rivera JD, Loneragan GH, and Brashears MM. 2005. Reduction of *Escherichia coli* O157 in finishing beef cattle by various doses of Lactobacillus acidophilus in direct-fed microbials. J Food Protection 68:6–10.
366. Zhang J, Xia S, Shen G, Chen Z, Huang P, Fu B, and Tu G. 2002. A study on acute renal failure after an outbreak of diarrhea in Suixian county, Henan province [Chinese]. Chung-Hua Liu Hsing Ping Hsueh Tsa Chih Chinese J Epidemiol 23:105–107.
367. Zhao T, Doyle MP, Harmon BG, Brown CA, Mueller POE, and Parks AH. 1998. Reduction of carriage of enterohemorrhagic *Escherichia coli* O157:H7 in cattle by inoculation with probiotic bacteria. J Clin Microbiol 36:641–647.
368. Ziese T, Anderson Y, de Jong B, Löfdahl S, and Ramberg M. 1996. Outbreak of *Escherichia coli* O157 in Sweden. Euro Surveill 1(1):2–3.
369. Zweifel C, S. Schumacher, Beutin L, Blanco J, and Stephan R. 2006. Virulence profiles of Shiga toxin 2e-producing *Escherichia coli* isolated from healthy pig at slaughter. Vet Microbiol. 117(2–4):328–332.
370. Archer, John (*personal communication*). Epidemiologist, Wisconsin Division of Public Health, Bureau of Communicable Diseases and Preparedness, Communicable Disease Epidemiology Section. http://dhfs.wisconsin.gov/communicable/Communicable/Contacts.htm
371. Loneragan GH, and Brashears MM. 2005. Pre-harvest interventions to reduce carriage of *E. coli* O157 by harvst-ready feedlot cattle. Meat Sci 71:72–78.
372. Callaway TR; Elder RO; Keen JE; Anderson RC, and Nisbet DJ. 2003. Forage feeding to reduce preharvest *Escherichia coli* populations in cattle, a review. J Dairy Sci 86(3):852–860.
373. Centers for Disease Control. 2006. Ongoing multistate outbreak of *E. coli* O157:H7 infections associated with consumption of fresh spinach – United States, September, 2006. Morbid Mortal Week Rep Dispatch 55:September 26, 2006. http://www.cdc.gov/mmwr/PDF/wk/mm55d926.pdf
374. Food and Drug Administration. 2006. Nationwide *E. coli* O157:H7 outbreak: questions and answers. http://www.cfsan.fda.gov/~dms/spinacqa.html
375. Franz E ; Visser AA; Van Diepeningen AD; Klerks MM; Termorshuizen AJ , and van Bruggen AH. 2007. Quantification of contamination of lettuce by GFP-expressing *Escherichia coli* O157:H7 and *Salmonella enterica* serovar Typhimurium. Food Microbiol 24(1):106–112.
376. Centers for Disease Control. 2006. *E. coli* O157:H7 outbreak from fresh spinach. http://www.cdc.gov/foodborne/ecolispinach/
377. The Associated Press. Wild pigs eyed in tainted spinach probe. New York Times, October 27, 2006. http://www.nytimes.com/aponline/us/AP-Tainted-Spinach.html

Corresponding author: M. Ellin Doyle, Ph.D., medoyle@wisc.edu

Food Research Institute, UW–Madison, August/October 2006
Funded in part by the American Meat Institute Foundation

Appendix 1

Chronological List of Outbreaks of *E. coli* O157:H7

Date	Location	Cases	Deaths	HUS	Vehicle	Ref #
1982 (a)	US: OR, MI	20			beef, ground	(*299*;*353*)
1982 (b)	Canada	31	1	0	beef, ground	(*294*)
1984	US: NE	34	4	1	beef, ground (nursing home)	(*303*)
1985	Canada (nursing home)	73	17	12	meat;person-person	(*81*;*206*)
1986 (a)	US: WA	40	2	1	beef, ground	(*270*)
1986 (b)	US: WI	2	0	2	milk, unpasteurized	(*230*)
1986 (c)	Canada	74		1	milk, unpasteurized	(*178*)
1988 (a)	US: MN	32	0	0	meat patties	(*54*)
1988 (b)	US: WI	61	0	0	beef, roast	(*302*)
1988 (c)	US: UT	51	4	8	beef, ground	(*273*)
1989	Canada	7	1		water, well (nursing home)	(*294*)
1990 (a)	Japan	14	2	14	water, tap	(*5*)
1990 (b)	US: MO	243	4	2	water, well	(*331*)
1990 (c)	UK: Scotland	11	4		person-person;geriatric hospital	(*205*)
1990 (d)	UK	16		4	restaurant food	(*229*)
1990 (e)	UK	4	0	4	water, drinking	(*122*)
1990 (f)	Israel	4	0	0	person-person	(*219*)
1990 (g)	US: ND	70		2	beef, roast	(*238*)
1991 (a)	Canada	521	2	22	beef, ground?;person-person	(*268*)
1991 (b)	US: MA	18	0	0	apple cider	(*59*)
1991 (c)	UK	16	0	5	yogurt	(*248*)
1991 (d)	US: OR	21	0	3	water, lake	(*201*)
1992 (a)	Germany	45	1	3	person-person	(*293*)
1992 (b)	Scotland	6		1	water, pool	(*71*)
1992-3 (c)	US: WA, ID, CA, NV (Jack in Box)	>700	3	37	beef, ground	(*52*;*69*;*100*;*119*;*314*;*341*)
1992-3 (f)	France	4	0	4	cheese, cows' unpasteurized	(*121*)
1992-4	US: OR	14	0	0	milk, unpasteurized	(*200*)
1993 (a)	UK: Wales	8		1	beef, ground	(*360*)
1993 (b)	US: OR, WA,	93	0	0	salad bar ?	(*184*)
1993 (c)	Netherlands	11		4	water, lake	(*111*)
1993 (d)	US: CA	15	0	0	beef, ground	(*340*)
1993 (e)	UK	6	1	3	water, paddling pool	(*171*)
1993 (f)	US: CT	20	0	0	beef, ground	(*300*)
1993 (g)	Germany	6		1	person-person	(*224*)
1994 (a)	US: VA	20	0	1	beef, ground	(*144*)
1994 (b)	US: WA, CA	23	0	2	salami	(*9*)
1994 (c)	Italy	15	1	15	poultry ?	(*338*)
1994 (d)	UK: Scotland	>100	1	9	milk, pasteurized	(*342*)
1994 (e)	US: NY	12	0	2	water, lake	(*4*)
1994 (f)	UK: Scotland	22			beef, ground	(*118*;*278*)
1994 (g)	UK: Scotland	71	2	10	milk, pasteurized	(*301*)
1994 (h)	UK: Scotland	26	0	1	beef, ground	(*117*)
1994 (i)	US: CT	21			supermarket, food?	(*47*)
1994 (j)	UK: England	7		4	animals, farm	(*320*)
1994 (k)	US: WI	26	0	0	sandwiches	(*370*)
1994 (l)	UK: Scotland	22		1	cheese	(*178*)
1995 (a)	US: IL	12		3	water, lake	(*348*)
1995 (b)	US: Mont.	92		1	lettuce, leaf	(*3*)
1995 (c)	UK: Wales	31		2	person-person	(*6*)
1995 (d)	US: GA, TN	10		1	beef, ground	(*79*)
1995 (e)	US: IL	12			water, lake	(*250*)
1995 (f)	US: OR	11	0	0	deer jerky	(*202*)
1995 (g)	UK	8		1	potatoes, person-person	(*96*)
1995 (h)	Canada	21	0	0	lettuce	(*285*)
1995 (j)	UK: Scotland	633	0	2	water, tap	(*193*)
1995 (k)	UK	14	0	2	meats, precooked	(*327*)
1995 (l)	UK	26	0	2	sandwiches, takeaway	(*237*)
1995 (m)	Czech Republic	9		4	milk, goat unpasteurized	(*65*)

1995 (n)	US: WI	27	0	0	water	(*370*)
1995 (o)	UK: England	11	0	4	ham, cooked	(*359*)
1995 (p)	US: MN	33			water, drinking	(*220*)
1995-6 (a)	Sweden	110	0	29	unknown	(*368*)
1995-6 (b)	Germany	28	3	28	sausage	(*12*)
1996 (a)	US and Canada (Odwalla)	70	1	14	apple juice	(*14*;*105*)
1996 (b)	US: CT, IL	61		3	lettuce, mesclun	(*169*)
1996 (c)	US: GA	18		1	water, pool	(*143*)
1996 (d)	Japan, Sakai	12,680	12	121	sprouts, radish (schools)	(*145*;*241*;*350*;*363*)
1996 (e)	US: CT	14	0	4	apple cider	(*170*)
1996 (f)	Japan	47	1	3	sprouts,radish (factory)	(*351*)
1996 (g)	Scotland (Wishaw)	503	20	34	meats, cooked	(*128*;*277*;*278*)
1996 (h)	US: CO	24	0	1	person-person	(*357*)
1996 (i)	Australia	6	0	0	food handler	(*234*)
1996 (j)	US: FL	2			water, well	(*220*)
1996 (k)	UK	3	0	0	milk, unpasteurized	(*15*)
1996 (l)	UK	12	0	1	milk, raw and "pasteurized"	(*104*)
1996-7	Scotland	512	17		meat from a shop	(*109*)
1997	Europe	15	0	3	water, well	(*275*)
1997 (a)	US: CO (Hudson)	15			beef, ground	(*318*)
1997 (c)	US: MI, VA	108	0	4	sprouts, alfalfa	(*70*;*107*)
1997 (d)	UK: England	5		2	animals, farm	(*287*)
1997 (e)	UK	8	0	1	environment (dairy farm field)	(*21*;*110*)
1997 (f)	UK	45	0	0	cakes, cream-filled	(*262*)
1997 (g)	Finland	14			water, lake	(*272*)
1997 (h)	UK	332	0	1	restaurant food	(*17*)
1997 (i)	UK	2	0	0	cheese, cows' unpasteurized	(*23*)
1997 (j)	UK	3	0	2	animals, farm	(*92*;*242*)
1997 (k)	Europe (travel overseas)	10	0	3	water, drinking ?	(*18*)
1997 (l)	UK: Scotland	6			unknown (nursing home)	(*19*)
1997 (m)	UK: Scotland	10			meat, cooked, butcher shop	(*20*)
1997 (n)	UK: Scotland	5		1	unknown:farm animals?	(*16*)
1997 (o)	UK: Scotland	34			unknown; hospital	(*22*)
1997 (p)	US: WA	4			water, well	(*49*)
1997 (q)	US: MO	8			water, lake	(*49*)
1998 (a)	US: WI	69	0	1	cheese curds	(*131; 370*)
1998 (b)	US: WY	157		4	water, tap	(*266*)
1998 (c)	US: CA, NV	8	0		sprouts, alfalfa	(*244*)
1998 (d)	Canada	39	0	2	salami	(*358*)
1998 (e)	Canada	11	0	0	apple cider	(*334*)
1998 (f)	UK (Dorset)	10			water, drinking	(*26*)
1998 (g)	UK	7	0	0	cream, unpasteurized	(*24*)
1998 (h)	UK	1		1	cheese, cows' unpasteurized	(*25*)
1998 (i)	US: WI	47	0	3	fruit salad	(*370*)
1998 (j)	US: WI	13	0	0	beef, ground	(*370*)
1998 (k)	US: GA	26			water park	(*49*)
1998 (l)	US: MN	5			water, lake	(*49*)
1998 (m)	US: IL	3			water, well	(*49*)
1999 (a)	US: NY (Wash. Co. fair)	127	2	14	water, well; 781cases *E. coli* and/or *C. jejuni*	(*258*;*260*)
1999 (b)	UK	114		3	milk, pasteurized	(*153*)
1999 (c)	Canada	143	0	6	salami	(*226*)
1999 (d)	US: CA, NV, AZ	13	0	3	beef, tacos	(*187*)
1999 (e)	US: CT	1			deer meat	(*288*)
1999 (f)	US: WA	37	0	3	water, lake	(*74*;*306*)
1999 (g)	Scotland	6			water, drinking	(*221*)
1999 (h)	UK: Wales	24		3	animals, farm	(*274*)
1999 (i)	Canada	7	0	0	beef, ground	(*225*)
1999 (j)	US: NY	>1,000	2	11	water, well	(*97*)
1999 (k)	UK	3	0	0	cheese, cows' unpasteurized	(*27*)
1999 (l)	UK	9		3	milk?; person-person	(*30*)
1999 (m)	UK	27	0	0	unknown	(*28*)
1999 (n)	UK	5	0	2	unknown-visit to Turkey	(*29*)
1999 (o)	UK	14	1	3	beach	(*31*;*164*)
1999 (p)	US: CA	7	0	0	water, lake	(*137*)

1999 (q)	Canada	159			animals, petting zoo	(*283*)
1999 (r)	Sweden	11	0	0	lettuce	(*352*)
1999 (s)	US: TX	22			water, drinking	(*215*)
1999 (t)	US: IL, KY, MO	329			beef	(*182*)
2000 (a)	US: WA	5	0		animals, farm	(*147*)
2000 (b)	Canada (Walkerton)	2,300	7	27	water,drinking	(*175*)
2000 (c)	UK: Scotland	20			environment (agr. showground)	(*174*)
2000 (d)	UK	6	0	1	milk, unpasteurized (2 outbrk)	(*35*)
2000 (e)	UK	9	0	1	foods, deli	(*36*)
2000 (f)	UK	15	0	3	unknown	(*36*)
2000 (g)	UK	7			person-person	(*33*)
2000 (h)	UK	45	0	0	foods? (prison)	(*34*)
2000 (i)	UK: Scotland	18			animals, farm ?; camp	(*32*)
2000 (j)	US: WI (Sizzler)	788	1	4	beef, unknown foods	(*370*)
2000 (k)	US: OH	48	0	2	water, drinking	(*294*)
2000 (l)	Netherlands	2		1	animals, petting zoo	(*166*)
2000 (m)	US: PA	51	0	8	animals, farm	(*113*)
2000 (n)	US: WI	15	0	3	unknown food	(*370*)
2000 (o)	US: WI	9	0	1	beef, ground	(*370*)
2000 (p)	US: ID	4			water, canal	(*215*)
2000 (q)	US: CA	5			water, stream	(*215*)
2000 (r)	US: UT	102			water, irrigation; also *C. jejuni*	(*215*)
2000 (s)	US: ID	15			water, spring	(*215*)
2000 (t)	US: CT	11			water, lake	(*215*)
2001 (a)	US: OH	23		1	environment (fair building)	(*345*)
2001 (b)	Canada	5	0	2	milk, goat, unpasteurized	(*239*)
2001 (c)	Canada	4	0	0	water, lake	(*75*)
2001 (d)	Austria	2	0	1	milk, unpasteurized	(*11*)
2001 (e)	UK	20		1	person-person	(*38*)
2001 (f)	UK: Scotland	15			water private well	(*37*)
2001 (g)	China	39	32	28	water, animals?(91%>60yrs)	(*366*)
2001 (h)	UK	30	0	2	meat (butcher shop)	(*290*)
2001 (i)	US: MN	4	0	0	animals, farm	(*322*)
2001 (j)	US: WI	55		1	animals, fair	(*370*)
2001 (k)	US: WI	34	0	1	environment (stock pavilion)	(*370*)
2001 (l)	US: MN	20			water, lake	(*364*)
2001 (m)	US: SC	45			water, lake	(*364*)
2001 (n)	US: ME	9			water, wading pool	(*364*)
2002 (a)	US: CO, etc. (ConAgra)	28		5	beef, ground	(*319*;*346*)
2002 (c)	Canada	13	0	2	cheese, cows' unpasteurized	(*172*)
2002 (d)	Canada	17	0	2	person-person;daycare	(*148*)
2002 (e)	Canada	109	2	0	salads	(*67*)
2002 (f)	UK, France	10	0	0	salad, cucumber	(*127*)
2002 (g)	UK: Wales	16	0	0	water, drinking	(*41*)
2002 (h)	UK	16			person-person	(*40*)
2002 (i)	UK	15			water, drinking	(*39*)
2002 (j)	US: WI	54	0	0	beef, ground	(*370*)
2002 (k)	US: WA	76			lettuce, romaine	*Ecoliblog*
2002 (l)	US: KY	2			water, well	(*66*)
2002 (m)	US: OR	60		12	environment, animal dust (fair)	(*57*)
2003 (a)	US: MN, CO	20			sprouts, alfalfa	(*138*)
2003 (b)	US	12		1	beef, steak, blade tenderized	(*212*)
2003 (c)	Canada	45	0	0	animals, petting zoo	(*115*)
2003 (d)	Canada	8	0	3	person-person	(*1*)
2003 (e)	Australia	3	0	0	foods?	(*106*)
2003 (f)	US: CA	32	2	3	spinach (nursing home)	(*294*)
2003?	Slovakia	9		3	milk, unpasteurized	(*222*)
2003-4	Denmark	25	0	0	milk, pasteurized	(*188*)
2004 (a)	Japan: Okinawa	6	0	0	beef, ground	(*207*)
2004 (b)	France	3		2	cheese, goats'	(*135*)
2004 (c)	Canada	34	0	2	beef, ground	(*330*)
2004 (d)	Canada	13	0	1	water park	(*152*)
2004 (e)	UK: Scotland	5	0	2	water, drinking	(*42*)
2004 (f)	Japan: Mie	23		1	person-person	(*329*)
2004 (g)	UK: England	7	0	0	water, stream	(*180*)

2004 (h)	US: NC	108		15	animals, petting zoo	(*116*)
2005 (a)	France	13		13	beef, ground	(*142*)
2005 (b)	UK: Wales	157	1	2	meat, cooked	(*305*)
2005 (c)	UK: Scotland; Turkey	15	0	0	unknown	(*323*)
2005 (d)	Sweden	120	0	7	lettuce	(*333*)
2005 (e)	Ireland	18	0	2	water, drinking; person-person	(*228*)
2005 (f)	UK	79	0		beef	(*44*)
2005 (g)	Japan: Kumamoto	11	0	0	beef, offal	(*231*)
2005 (h)	US: FL	73	0	12	animals, petting zoo	(*116*)
2005 (i)	US: AZ	2			animals, petting zoo	(*116*)
2005 (j)	US: WA	18	0	2	milk, unpasteurized	(*51*)
2005 (k)	Netherlands	32	0	0	beef, steak tartare	(*125*)
2005 (l)	US: MN, WI, OR	25	1		lettuce, packaged (Dole)	(*252*)
2006 (a)	UK: Scotland (Fife)	23	0	4	person-person? (nursery)	(*43*)
2006 (b)	UK: Scotland	3	0	0	meat (butcher shop,Lanarkshire)	(*43*)
2006 (c)	UK: England	4	1	4	unknown (nursery)	(*43*)
2006 (d)	UK: England	25			meat, cooked, butcher shop	(*45*)
2006 (e)	US: TN	10		3	person-person (daycare)	(*282*)
2006 (f)	US: MN	17	2	1	ground beef	(*173*)
2006 (g)	US: 26 states; Canada	204	3	31	spinach	(*373;376*)