

S C I E N T I F I C S T A T U S S U M M A R Y

Foodborne Disease Significance of
Escherichia coli O157:H7
and Other Enterohemorrhagic *E. coli*

A PUBLICATION OF
THE INSTITUTE OF FOOD TECHNOLOGISTS'
EXPERT PANEL ON FOOD SAFETY AND NUTRITION

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This Scientific

Status Summary

addresses the

virulence and

disease

characteristics of

EHEC, their

reservoirs and

sources, survival

and growth, and

disease prevention

strategies

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The unusually virulent enterohemorrhagic strains of *Escherichia coli*, including the O157:H7 serotype, have prompted food microbiologists to rewrite the rule book on food safety. These pathogens are more significant than other well-recognized foodborne pathogens for reasons including the severe consequences of infection that affect all age groups, their low infectious dose, their unusual acid tolerance, and their apparent special but inexplicable association with ruminants that are used for food.

New safety recommendations for destroying enterohemorrhagic *E. coli* (EHEC) include cooking hamburgers thoroughly, incorporating a procedure that kills EHEC in the manufacture of raw fermented sausage, such as salami, and pasteurizing or using an equivalent processing method for apple cider. Public health problems with EHEC are being recognized throughout the world. The need for consumer education on the safe handling of foods has never been more acute.

Historical Perspective

E. coli O157:H7 (designated by its somatic, O, and flagellar, H, antigens) was first recognized as a human pathogen following two hemorrhagic colitis outbreaks in 1982 (Riley et al., 1983). The first outbreak, with 26 cases of which 19 were hospitalized, occurred in Oregon, and the second, with 21 cases and 14 hospitalizations, followed three months later in Michigan. Undercooked hamburgers from the same fast food restaurant chain were identified as the vehicle, and *E. coli* O157:H7 was isolated from patients and a frozen ground beef patty.

Shortly after *E. coli* O157:H7 was determined to be a human pathogen, Karmali et al. (1983) observed that stool samples from children with

hemolytic uremic syndrome (HUS) contained a substance that was toxic to Vero (African green monkey kidney) tissue culture cells. This verocytotoxin was produced by *E. coli* isolates, with O157:H7 the prominent serotype causing infection.

Enterohemorrhagic *E. coli* and Foodborne Illness

E. coli has been used since 1890 as a non-pathogenic indicator of enteric pathogens, such as *Salmonella*. However, as knowledge of enteric diseases increased, investigators began isolating strains of *E. coli* that had acquired virulence characteristics causing pathogenicity to humans or animals. Six classes of diarrheagenic *E. coli* are recognized: enterohemorrhagic (EHEC), enterotoxigenic (ETEC), enteroinvasive (EIEC), enteroaggregative (EaggEC), enteropathogenic (EPEC), and diffusely adherent (DAEC).

• **Definition of EHEC.** EHEC are loosely defined by a combination of the symptoms they produce and the virulence factors they possess (Neill et al., 1994). The disease-defining symptom of EHEC is hemorrhagic colitis (HC), i.e., bloody diarrhea. Not all EHEC infections, however, produce overt blood in the stools. While *E. coli* O157:H7 infections have a high rate of bloody stools, this may not be the case for other EHEC strains.

All EHEC strains produce Shiga toxin 1 (Stx1) and/or Shiga toxin 2 (Stx2), also referred to as verotoxin 1 (VT1) and verotoxin 2 (VT2). The ability to produce Shiga toxin was acquired from a bacteriophage, presumably directly or indirectly from *Shigella*.

The toxin is a 70,000 dalton protein composed of a single A subunit (32 kDal) and five B subunits (7.7 kDal). The B subunits provide tissue specificity by binding to globotriaosylceramide (Gb₃) receptors on the surface of eucaryotic cells. The A subunit has an N-glycosidase that inactivates the 28S ribosome, thus blocking protein synthesis. Endothelial cells high in Gb₃ receptors are the pri-

E. coli O157:H7

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mary target, accounting for the toxin's affinity for the colon and the renal glomeruli, associated with HC and HUS, respectively. The toxin can also indirectly damage cells by releasing cytokines, such as tumor necrosis factor.

Toxin alone, however, is not sufficient to make *E. coli* pathogenic; apparently nonpathogenic, stx-positive isolates are isolated frequently from humans. To be fully pathogenic, EHEC require the presence of other virulence markers. The *eae* chromosomal gene, for example, is ubiquitous among EHEC strains, encoding for an outer membrane protein associated with attachment. Although its role is unclear, the presence of a plasmid-encoded enterohemolysin is characteristic of EHEC.

• Disease Characteristics. Although the symptoms of *E. coli* O157:H7 infections are distinctive, they may be confused at any single phase with other diseases or conditions (Tarr, 1995; Fig. 1). The initial symptoms of HC generally occur 1–2 days after eating contaminated food, though longer periods (3–5 days) have been reported. Symptoms start with mild, nonbloody diarrhea that may be followed by a period of “crampy” abdominal pain and short-lived fever. The initial diarrhea increases in intensity during the next 24–48 hr to a 4- to 10-day phase of overtly bloody diarrhea accompanied by severe abdominal pain and moderate dehydration.

Several life-threatening complications may occur in HC patients; HUS is the most common. The onset of HUS is approximately a week after the onset of gastrointestinal symptoms. Characteristic symptoms are pallor, intravascular destruction of red blood cells (microangiopathic hemolytic anemia), depressed platelet counts (thrombocytopenia), lack of urine formation (oligo-anuria), swelling (edema), and acute renal failure. HUS occurs most often in children under the age of 10. Approximately half of HUS patients require dialysis, and the mortality rate is 3–5%.

Other HUS-associated complications may include seizures, coma, stroke, colonic perforation, pancreatitis, and hypertension. Approximately 15% of cases

lead to early development of chronic kidney failure. Insulin-dependent diabetes may also persist in HUS patients. A small number of HUS cases may recur (Siegler et al., 1993).

A second complication associated with *E. coli* O157:H7 is thrombotic thrombocytopenic purpura. This condition resembles HUS except that it generally causes less renal damage; has significant neurological involvement, e.g., central nervous system deterioration, seizures, and strokes; and is restricted primarily to adults (Boyce et al., 1995).

• Serotypes included in EHEC. Since 1982, *E. coli* O157:H7 has been implicated in outbreaks worldwide and is the primary cause of HC and HUS in the United States, Canada, Great Britain, and regions in Europe. The pathogen is likely responsible for 85–95% of HUS cases (Griffin, 1995). This has led to classification of EHEC largely on the basis of characteristics of serotype O157:H7. Other EHEC serotypes, however, are reported occasionally in these regions, and in other geographical areas non-O157:H7 serotypes predominate. For example, serotypes O111:H– (H– indicates nonmotile) and O157:H– are more common in Australia (Goldwater and Bettelheim, 1995), and a high incidence of sorbitol-positive (capable of fermenting sorbitol-containing culture medium) O157:H– in central Europe has been reported (Bitzan et al., 1993). Other serotypes of EHEC strains are O4:H–, O11:H–, O26:H11, O45:H2, O103:H2, O104:H21, O111:H8, and O145:H– (Beutin et al., 1994; Tzipori et

al., 1988).

• Foods Implicated in EHEC Outbreaks. Ground beef has been the food most often associated with outbreaks in the United States (Griffin and Tauxe, 1991). Large outbreaks include the 1992–93 outbreak that affected more

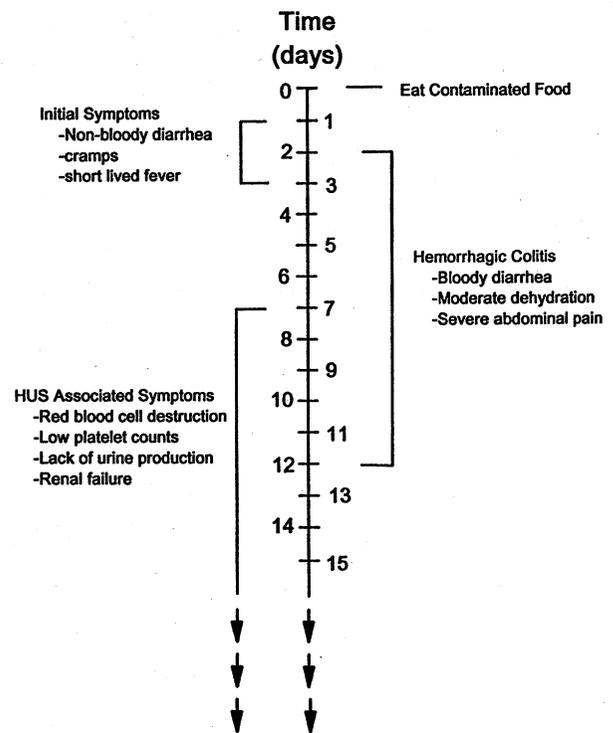


Fig. 1—Symptoms and time course of *Escherichia coli* O157:H7 infection (hemorrhagic colitis) and its primary complications (hemolytic uremic syndrome, HUS)

than 500 individuals in the western United States (CDC, 1993). A significant portion of HC infections are sporadic, i.e., not associated with outbreaks. Ground beef also has been implicated as a risk factor in those sporadic infections (Le Saux et al., 1993; Pai et al., 1988).

Dry-cured salami was associated with an *E. coli* O157:H7 outbreak in the western United States, demonstrating that low levels of this organism can survive in acidic, fermented meats and cause illness (Tilden et al., 1996). An Australian outbreak of HC and HUS resulted from consumption of contaminated

mettwurst (CDC, 1995). EHEC isolates of serotypes O111:H- and O157:H- were isolated from both patients and product (Paton et al., 1996). *E. coli* isolates capable of producing one or more Shiga toxins can be isolated readily from meat, poultry, and seafoods (Samadpour et al., 1994); however, most do not possess the other virulence determinants associated with fully pathogenic EHEC.

Other foods have been associated with EHEC outbreaks worldwide (Table 1). Unpasteurized apple juice and cider have received considerable attention due to local and multistate outbreaks (Besser et al., 1993). A 1980 outbreak of HUS involving fresh apple juice is now suspected of being caused by EHEC (Steele et al., 1982). Sources are not identified in a substantial portion of EHEC cases, but a nonspecific association has often been made with the consumption of food in restaurants (Waters et al., 1994). This may be attributed in part to secondary person-to-person (Griffin and Tauxe, 1991) or animal-to-person (Wilson et al., 1996) spread of EHEC. For example, *E. coli* O157:H7 is similar to *Shigella* in its association with day-care centers, which are often foci for infections (Belongia et al., 1993). The largest reported *E. coli* O157:H7 outbreak, which caused thousands of illnesses, occurred in Japan in 1996. This outbreak and a second one a year later were associated with radish sprouts. Alfalfa sprouts were also implicated in a recent outbreak in the U.S.

The infectious dose (2–2,000 cells) associated with foodborne *E. coli* O157:H7 outbreaks has been consistently low—a characteristic associated with the organism's acid tolerance. It has been suggested that outbreak-associated strains of *E. coli* O157:H7 may have enhanced acid tolerance (Buchanan and Edelson, 1996). The inability of *E. coli* O157:H7 to ferment sorbitol, however, is not associated with its virulence (Frata-mico et al., 1993).

Reservoirs and Sources of *E. coli* O157:H7

Several reservoirs and sources of *E. coli* O157:H7 have been identified:

Cattle. The association of *E. coli* O157:H7 with undercooked ground beef and raw milk led to investigations of the role of cattle as a reservoir of the pathogen. Several surveys of fecal shedding of *E. coli* O157:H7 produced the following general observations:

- Young animals tend to carry *E. coli*

O157:H7 more frequently than adults (Zhao et al., 1995).

- Prevalence of fecal excretion varies substantially among positive herds (Hancock et al., 1994; Zhao et al., 1995).

- Reported incidence among cattle varies widely, in part because of differences in sensitivity of procedures used for detecting *E. coli* O157:H7.

- Results of two major U.S. surveys indicated that 31 (3.2%) of 965 dairy calves (Zhao et al., 1995) and 191 (1.6%) of 11,881 feedlot cattle were positive for *E. coli* O157:H7. An additional 0.4% of feedlot cattle were positive for *E. coli* O157:H- (USDA/APHIS, 1995).

- *E. coli* O157:H7 levels in calf feces range from $<10^2$ CFU/g to 10^5 CFU/g (Zhao et al., 1995).

- Fecal shedding of *E. coli* O157:H7 frequently is intermittent and of short duration, i.e., several weeks to months (Brown et al., 1997; Cray and Moon, 1995).

- Strains of *E. coli* O157:H7 with indistinguishable pulsed field gel electrophoresis (PFGE) genomic DNA profiles can be isolated from calves in different states or farms (Faith et al., 1996; Meng et al., 1995).

- More than one strain of *E. coli* O157:H7 can be isolated from feces of the same animal or different animals within the same herd (Faith et al., 1996; Meng et al., 1995).

Calves have been experimentally infected with *E. coli* O157:H7 (Brown et al., 1997; Cray and Moon, 1995); results revealed that:

- *E. coli* O157:H7 is not pathogenic to calves; inoculation with 10^{10} CFU did not induce significant clinical disease.

- The numbers of *E. coli* O157:H7 shed in feces decreased dramatically during the first 14 days postinoculation (e.g., from 10^4 to 10^6 CFU/g after 48 hr to $5-10^2$ CFU/g at 14 days).

- *E. coli* O157:H7 is confined to the gastrointestinal tract, with the forestomachs (rumen, omasum, and reticulum) and distal sites (distal ileum, proximal

cecum, spiral colon, and descending colon) being the principal sites of localization.

- Fasting increases the levels of *E. coli* O157:H7 shed in the feces of some animals, but not in most.

- *E. coli* O157:H7 did not form attaching and effacing lesions and did not colonize mucosal surfaces.

Oral inoculation of calves and steers with 10^{10} *E. coli* O157:H7 induced prompt and sustained increases in serum antibodies to the O157 antigenic lipopolysaccharide and to a lesser extent to Stx1 (Johnson et al., 1996). The serological responses, however, do not correlate with elimination of carriage by cattle or protection of calves against reinfection by the same strain. The ability of *E. coli* O157:H7 to persist in and reinfest cattle that have a strong immune response is likely to contribute to the introduction

and persistence of infection in herds.

Deer. Recent *E. coli* O157:H7 investigations have established that deer are a source of the pathogen and that transmission of the pathogen may occur between deer and cattle (Keene et al., 1997; Rice et al., 1995). For example, in a recent outbreak involving contaminated venison jerky, *E. coli* O157:H7 with the same distinctive PFGE profile were isolated from the human

cases, leftover jerky, uncooked meat from the same deer, a saw used to cut up the carcass, and fragments of the deer hide. Deer and cattle fecal samples obtained from a ranch in Texas had the same Shiga toxin-producing *E. coli* O157:H7 isolate (Rice et al., 1995).

Sheep. Sheep have also been identified as a reservoir of *E. coli* O157:H7 (Kudva et al., 1996). A six-month study of healthy ewes revealed that fecal shedding of the pathogen was transient and seasonal, with 31% of sheep positive in June, 5.7% positive in August, and none in November. The sheep showed no signs

Table 1 Foods or food handling practices implicated or suspected of being associated with *Escherichia coli* O157:H7 outbreaks

Undercooked ground beef
Raw milk
Unpasteurized apple juice/cider
Dry cured salami
Lettuce
Produce from manure-fertilized garden
Handling potatoes
Radish sprouts, alfalfa sprouts
Yogurt
Sandwiches
Water

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of disease throughout the study. Animals perorally administered 10^9 *E. coli* O157:H7 fecally shed the bacteria for up to 92 days. A shedding sheep passed *E. coli* O157:H7 to a non-dosed pen mate. Diet influenced fecal shedding of *E. coli* O157:H7; sheep apparently negative for *E. coli* O157:H7 began to shed the bacteria when the animals were removed from confinement and their feed was changed from alfalfa pellets to sage brush and bunchgrass (Kudva et al., 1995).

Water. Drinking and recreational waters have been vehicles of several outbreaks of *E. coli* O157:H7 infection (Doyle et al., 1997). A large outbreak of 243 cases including four deaths was associated with contamination of municipal water in Cabool, Mo., from December 1989 to January 1990 (Swerdlow et al., 1992). Two large water mains, broken because of extreme cold weather, and new in-ground water meters may have contributed to the outbreak. In 1991, 21 cases of *E. coli* O157:H7 infection were traced to swimming at a lakeside park in Portland, Ore. (Keene et al., 1994). Bath-ers, including many toddlers not yet toilet trained, ingested fecally contaminated lake water. Water has been suggested as a vehicle of transmitting *E. coli* O157:H7 among cattle (Faith et al., 1996).

Factors Affecting Survival and Growth

Like all bacteria, the survival and growth of *E. coli* O157:H7 in foods are dependent on the interaction of various intrinsic and extrinsic factors such as temperature, pH, and water activity.

• **Temperature.** EHEC strains respond to temperature in the same manner as non-EHEC strains, with the exception of isolates of serotype O157:H7. *E. coli* are differentiated from other *Enterobacteriaceae* (family of Gram-negative, catalase-positive, oxidase-negative, facultatively anaerobic rods) on the basis of their ability to grow and produce gas in EC (*E. coli*) broth at 44.5°C. Many O157:H7 isolates, however, do not grow well, if at all, above 44°C (Doyle and Schoeni, 1984). Palumbo et al. (1995) found that the upper temperature for *E. coli* O157:H7 growth was culture medi-

um-dependent; all strains grew in BHI (brain heart infusion) broth at 45°C, but six of sixteen strains did not grow in EC broth.

The minimum growth temperature for *E. coli* O157:H7 under otherwise optimal conditions is approximately 8–10°C (Buchanan and Bagi, 1994; Rajkowski and Marmer, 1995). The effect of temperature on the growth rates of both *E. coli* biotype 1 and EHEC has been determined, and mathematical models have been developed to describe how temperature interacts with pH, water activity, and sodium nitrite to affect growth kinetics (Buchanan and Bagi, 1994; Gill and Phillips, 1985; Sutherland et al., 1995).

• **pH.** Growth rates are similar at pH values between 5.5 and 7.5, but decline rapidly at lower pH values (Buchanan and Klawitter, 1992). The minimum pH for *E. coli* growth is 4.0–4.5 (Buchanan and Bagi, 1994). This is dependent on the interaction of pH with other growth parameters; for example, additional stresses raise the minimum pH for growth. The type of acid (e.g., organic vs inorganic) and acid concentration influence the effect of pH on *E. coli* growth. Abdul-Raouf et al. (1993) reported that in beef slurries, the relative inhibitory activity of organic acids on *E. coli* O157:H7 was acetic > lactic ≥ citric.

When the pH falls below the minimum for growth, *E. coli* O157:H7 populations decline over time. The pathogen's survival in acidic foods is particularly important, since several outbreaks have been associated with low levels of *E. coli* O157:H7 surviving in acidic foods, such as fermented sausages, apple cider, and apple juice. The pathogen has been shown experimentally to survive for several weeks to months in a variety of acidic foods, including mayonnaise (Zhao and Doyle, 1994), sausages (Clavero and Beuchat, 1996), apple cider (Zhao et al., 1993), and Cheddar cheese (Reitsma and Henning, 1996). Survival in these foods is extended greatly when stored at refrigeration temperature. For example, the pathogen survived in apple cider for only 2–3 days at 25°C, compared to 10–31 days at 8°C (Zhao et al., 1993).

EHEC strains can have a high degree of acid tolerance, surviving virtually unchanged during 2- to 7-hr exposures at pH 2.5 and 37°C (Benjamin and Datta, 1995; Buchanan and Edelson, 1996). Acid-sensitive EHEC strains, however, have also been identified. Conversely, acid-tolerant, non-enterohemorrhagic strains have also been identified, so this is not a characteristic unique to pathogenic isolates.

Acid tolerance in *E. coli* is a complex phenomenon, both growth phase-dependent and inducible. *E. coli* cells in the stationary phase of growth are substantially more acid tolerant than cells in the exponential phase. This increased tolerance is associated with expression of genes regulated by the *rpoS* sigma factor operon (Cheville et al., 1996; Rowbury, 1995; Small et al., 1994). Lin et al. (1996) examined three mechanisms of acid resistance, i.e., oxidative, arginine-dependent, and glutamate-dependent, and found that all three contribute to the microorganism's overall acid tolerance.

Induction of acid tolerance in *E. coli* can enhance its survival in acidic foods (Cheville et al., 1996; Leyer et al., 1995). An acid tolerant state can persist for extended periods (≥ 28 days) if the cells are stored at refrigeration temperature. The induction of acid tolerance can also enhance the organism's ability to survive other stresses. Recent studies have indicated that induction of acid tolerance also increases the microorganism's resistance to heating, radiation, and antimicrobials (Rowbury, 1995). *E. coli* also possesses an inducible alkali tolerance response (Rowbury et al., 1996).

• **Water Activity.** Studies on the effect of water activity on the survival and growth of *E. coli* O157:H7 focused primarily on the effect of sodium chloride, though, presumably, *E. coli* O157:H7 behaves similarly to other *E. coli*. Buchanan and Bagi (1994) developed a mathematical model for the effects and interactions of NaCl concentration (0.5–5.0%) with temperature, pH, and NaNO₂ on the growth kinetics of *E. coli* O157:H7. They compared the effects of mannitol, sorbitol, and sucrose as humectants and concluded that while humectant differences

occur at limiting a_w values, differences among humectants were minimal at a_w 0.98 (Buchanan and Bagi, 1997). Growing *E. coli* at elevated levels of NaCl induces *rpoS* expression with associated increases in thermotolerance and H_2O_2 resistance (Hengge-Aronis et al., 1993). *E. coli* O157:H7 can survive for many weeks when desiccated, particularly at refrigeration temperature (Bagi and Buchanan, 1993).

- **Antimicrobials.** *E. coli* O157:H7 does not appear to have any increased resistance to antimicrobial food additives.

Disease Prevention

E. coli O157:H7 represents unique challenges to preventing foodborne disease. Its low infectious dose in combination with the disease severity means that successful prevention strategies must focus on reducing or eliminating the presence of the microorganism, rather than on preventing pathogen growth, as is done in more traditional approaches. This focus is particularly important for raw products that may not be thoroughly cooked before consumption (e.g., ground beef) or ready-to-eat products that do not receive a definitive treatment that assures elimination of *E. coli* O157:H7 (e.g., fermented sausages, apple cider).

- **HACCP.** The Hazard Analysis and Critical Control Point (HACCP) system continues to be the most effective means for systematically developing food safety protocols that can reduce the risk of EHEC infections. EHEC, however, pose some unique problems when developing and implementing HACCP plans. For example, the low incidence of *E. coli* O157:H7 in foods makes direct microbiological testing for the pathogen as a means of verifying the effectiveness of a HACCP program of limited benefit. In such instances, verification based on microbiological analysis would have to depend on the use of an appropriate indicator organism that could provide a measure of how well a process controls factors associated with risk of *E. coli* O157:H7 contamination.

Most desirable is a process that includes a step lethal to the pathogen. This reduces the critical control points to assuring the effectiveness of that step and preventing subsequent cross contamination. For products that depend on non-thermal interventions to assure product safety (e.g., fermented meats), validation that the integrated process can achieve

the desired level of inactivation may be a necessary part of the hazard analysis phase of HACCP implementation.

HACCP plans that do not include a step that kills pathogens are more complex, since the focus is on risk reduction instead of risk elimination. Typically, there is one or more critical control points associated with steps that either reduce the likelihood that the pathogen has gained access to the product or actively reduce (but not eliminate) the levels that may be present.

Since such processes cannot assure complete absence of the pathogen, there will also be critical control points associated with preventing pathogen growth. For example, the generic HACCP plan for beef slaughter and fabrication developed by the National Advisory Committee on Microbiological Criteria for Foods (NACMCF, 1993) included *E. coli* O157:H7 as a hazard. The HACCP plan listed skinning, post-skinning rinsing/bactericidal spray, evisceration, final bactericidal rinse, chilling, and maintenance of refrigeration as likely critical control points. In addition to these specific activities associated with slaughter, the committee identified factors associated with animal production practices and with the distribution, marketing, and consumption of the final products that would have to be considered in a farm-to-table HACCP plan.

- **Farms.** An important component of HACCP application in animal production is reducing the carriage of *E. coli* O157:H7 by animals. Two approaches that have potential are competitive exclusion and vaccination.

Competitive exclusion involves the use of microbial cultures that out-compete pathogens from colonizing specific niches. This approach uses defined bacterial cultures that can greatly reduce colonization of *Campylobacter jejuni* in poultry (Schoeni and Doyle, 1992).

Vaccination involves exposing an animal to an attenuated pathogen or an antigen of a virulent microorganism to produce immunity. However, traditional vaccination approaches are not likely to be successful with *E. coli* O157:H7. Recent observations showed that *E. coli* O157:H7 does not form attaching and effacing lesions or colonize mucosal surfaces of the gastrointestinal tract (Brown et al., 1997; Cray and Moon, 1995), and cattle exposed to *E. coli* O157:H7 are not protected from reinfection (Johnson et al., 1996). Hence, innovative approaches

will be needed for vaccines to be effective.

- **Slaughterhouse.** Like other *E. coli*, it is assumed that the ultimate source of *E. coli* O157:H7 on carcasses is fecal contamination during animal production and slaughter operations. Fecal contamination is associated primarily with contamination of the carcass during hide removal and spreading of contamination to other carcasses by equipment and workers' hands (Dickson and Anderson, 1992).

Traditional trimming procedures can reduce *E. coli* O157:H7 levels on areas of the carcass with visible fecal contamination (Hardin et al., 1995). Various alternatives to trimming have been investigated for the removal of enteric pathogens. Recent studies with *E. coli* O157:H7 suggest that rinsing of carcass surfaces with solutions of organic acids may have limited effectiveness. Spray chilling with 1–2% acetic acid only produced a 1-log cycle (tenfold) reduction of *E. coli* O157:H7 on lean tissue; a slightly greater effect was observed on fat tissue (Dickson, 1991). Holding the meat for 24 hr indicated only a small residual effect on lean, but a substantial effect on fat tissue. Several investigators observed differences in the effectiveness of acid treatments between lean and fat tissue and among different portions of the carcasses (Cutter and Siragusa, 1994; Fratamico et al., 1996; Hardin et al., 1995).

Investigators found that acid rinses had little effect on eliminating *E. coli* O157:H7 from the surface of beef tissues (Brackett et al., 1994; Fratamico et al., 1996), possibly due to difficulty in removing *E. coli* O157:H7 from beef surfaces previously chilled (Hardin et al., 1995).

Previsceration washing decreased the subsequent attachment of *E. coli* O157:H7 to beef carcasses (Dickson, 1995). Trisodium phosphate has been evaluated as a sanitizing agent for carcass surfaces and equipment. Its overall effectiveness, due to its high pH, was similar to that achieved with organic acids (Fratamico et al., 1996). Trisodium phosphate can increase the removal of *E. coli* O157:H7 from equipment surfaces (Somers et al., 1994).

The actual fate of *E. coli* O157:H7 cells that have been removed from carcass surfaces by rinses with sanitizing agents is still unclear. Model system studies on the microorganism's ability to survive acids and other agents at non-

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lethal temperatures indicate that the exposure times associated with carcass sanitizing are too short to achieve any significant direct inactivation and suggest that the primary effect is the physical removal of the microorganisms.

The use of steam to briefly heat carcass surfaces to temperatures sufficient to inactivate *E. coli* O157:H7 while maintaining the raw character of the animal tissue is a new method for reducing the presence of enteric pathogens on meats and poultry. Steam vacuum systems are used for spot removal, and steam pasteurization cabinets are used for whole carcass treatments. The steam vacuum system is reportedly capable of achieving a 5-log cycle (100,000-fold) reduction of *E. coli* O157:H7 on inoculated beef surfaces (Dorsa et al., 1996).

• **Food Processing.** *E. coli* O157:H7 can be controlled readily through traditional thermal processing techniques; however, the organism's low infectious dose requires that processing be sufficient to assure a low probability of the pathogen's surviving. Dairy pasteurization processes designed to kill *Coxiella burnetii* should be sufficient to eliminate *E. coli* O157:H7. Similarly, pasteurization would be expected to control *E. coli* O157:H7 in fruit juices.

D-values (decimal reduction time, or the time required to destroy 90% of the population) have been determined at a number of different temperatures in various ground meat and poultry products (Ahmed et al., 1995; Doyle and Schoeni, 1984; Line et al., 1991). For example, reported $D_{60^\circ\text{C}}$ values for serotype O157:H7 in ground products range from 0.4 to 0.8 min.

D-values vary to some degree among ground products; however, thermal resistance is more strongly influenced by fat content, i.e., the higher the fat content, the greater the thermal resistance. Splittstoesser et al. (1995) estimated that the $D_{60^\circ\text{C}}$ for *E. coli* O157:H7 in apple juice (pH 4.0) was 0.4 min. Prior heat shock increases thermal resistance, and anaerobic incubation increases recovery of heated cells (Murano and Pierson, 1993). Cells held at refrigeration are more sensitive than cells heated directly

Table 2 Recommendations to reduce the risk of acquiring an *Escherichia coli* O157:H7 infection

1. Cook ground beef and venison thoroughly (minimum 160°F) before eating.
2. Drink only pasteurized milk and apple juice.
3. Wash fresh fruits and vegetables thoroughly before eating.
4. Wash hands thoroughly after handling animals, particularly cattle, deer, goats, or dogs.
5. Wash hands thoroughly after changing diapers or after providing care to children or adults suffering from a diarrheal disease.
6. Do not use fresh manure from ruminants to fertilize vegetables or fruits.
7. Avoid swimming in lakes or ponds used by cattle and drinking surface water that has not been properly treated to eliminate pathogens.

from the frozen state (Jackson et al., 1995). Elevated pH values (pH 10–11) can enhance the thermal destruction of *E. coli* O157:H7 (Teo et al., 1996).

Outbreaks associated with raw milk have prompted investigations into the fate of *E. coli* O157:H7 in dairy products. The pathogen persisted during the manufacture of cottage cheese (Arocha et al., 1992) and Cheddar cheese (Reitsma and Henning, 1996) made from inoculated milk. The organism is inactivated readily by pasteurization of milk, and levels declined during aging of the Cheddar cheese.

Alternative technologies to thermal processing that could eliminate or control *E. coli* O157:H7 while maintaining the raw character of foods are currently being investigated. One that has potential, particularly for meat and poultry products, is ionizing radiation. The pathogen is relatively radiation sensitive, and radiation pasteurization doses of 1.5–3.0 kGy appear to be sufficient to eliminate it at the levels that they are

likely to occur at in ground beef (Clavero et al., 1994; Thayer and Boyd, 1993). Radiation inactivation is temperature dependent; higher doses are required when ground beef is irradiated at frozen temperatures. There appears to be little data on the ability of irradiation to control *E. coli* O157:H7 on fruits and vegetables, though recent studies (Buchanan et al., unpublished data) indicate that low-dose irradiation of apple cider is effective.

While food processing research with *E. coli* O157:H7 has concentrated on products of animal origin, an increasing number of outbreaks have involved fruits and vegetables. *E. coli* O157:H7 strains have been shown to grow on many vegetables if stored at temperatures that support growth (Abdul-Raouf et al., 1993). Modified-atmosphere packaging, used extensively with produce, does not prevent the growth of *E. coli* O157:H7 (Abdul-Raouf et al., 1993; Hao and Brackett, 1993).

• **Home and Foodservice.** Food handling and preparation practices can contribute to *E. coli* O157:H7 infections and conversely play an important part in their prevention. Undercooking has been an important contributing factor in *E. coli* O157:H7 outbreaks associated with ground beef. Adherence to good food handling practices recommended for foodservice and home preparation represent the last line of defense for assuring prevention of *E. coli* O157:H7 infection (Table 2). Infected food handlers could potentially serve as foci for *E. coli* O157:H7 infections, particularly during the first 48 hr of an infection when symptoms are still relatively mild or in those individuals who do not have overtly bloody stools. In particular, adequate cooking temperatures and times, prevention of cross contamination between raw and cooked foods, and appropriate refrigerated storage are key factors for reducing the risks associated with *E. coli* O157:H7.

Summary

It is apparent that microbiologists, molecular biologists, and food scientists have made great strides in understanding *E. coli* O157:H7 and related EHEC and

developing means for controlling them in foods. It is also evident, however, that there are major scientific questions that must be answered before we will be able to fully assess and manage public health concerns associated with their food-borne transmission. Addressing these questions will require the continued effort and support of basic and applied scientists from a variety of disciplines.

On a broader front, a key lesson dramatically reinforced by the emergence of *E. coli* O157:H7 is that both the macroscopic and microscopic worlds change continually. We cannot take for granted that foods and food practices that have been traditionally safe will remain that way in the future. Continued vigilance and the ability to rapidly mobilize research capabilities must be an integral part of food safety programs if we are going to minimize the impact of new foodborne microbial threats to human health.

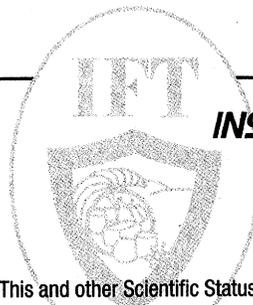
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Escherichia coli O157:H7— Harbinger of Change in Food Safety and Tradition in the Industrialized World

The industrialized world has been forced to change many traditions in the way that we enjoy, handle, and think about our food supply. One small pathogen, *Escherichia coli* O157:H7, has had a tremendous influence on the United States government, industry, and general public. During the last two weeks of August this year, it resulted in one of the largest food recalls in history and captured the attention of the national news media. The pathogen has singlehandedly reopened the public debate on the use of irradiation to reduce pathogens in food.

The Scientific Status Summary, "Foodborne Disease Significance of *Escherichia coli* O157:H7 and Other Enterohemorrhagic *E. coli*" (pages 69–76) prepared for IFT's Expert Panel on Food Safety and Nutrition by Robert L. Buchanan and Michael P. Doyle, describes the low infectious dose, severe disease consequences, and acid tolerance that have allowed *E. coli* O157:H7 to emerge as a threat in unexpected new places.

Not too long ago, calls for stricter government standards were met with the often-repeated response that microorganisms are ubiquitous and the consumer has the responsibility to handle and prepare food carefully. The consumer still has that responsibility, but it is shared more equitably by others in the farm-to-table chain of production, marketing, and preparation.

The 1993 Western states outbreak of *E. coli* O157:H7 attributed to undercooked hamburgers served at a fast-food chain was the turning point. The outbreak sickened hundreds and was responsible for four deaths. It certainly was not the first outbreak of foodborne illness that the United States had experienced, but it was a particularly difficult one for the public to accept; children were affected, and the food source was the traditional American hamburger.

A number of things have changed since then:

USDA. The U.S. Department of Agriculture declared *E. coli* O157:H7 an adulterant in raw ground beef in 1994; initiated a monitoring program for *E. coli* O157:H7 in raw ground beef in 1994 (testing confirmed that contamination occurs sporadically and at low levels); mandated safe food handling labels for raw

meat and poultry products in 1994; finalized the Hazard Analysis and Critical Control Point (HACCP) Systems/Pathogen Reduction regulation, requiring many companies to address enterohemorrhagic *E. coli* (EHEC) when identifying hazards in their HACCP plans (1996); created the Emergency Response Division for outbreak investigations and tracebacks in 1996; with the Food and Drug Administration (FDA), increased retail time/temperature cooking requirements for ground beef (Food Code); increased efforts to educate consumers to use a thermometer and cook hamburger to an internal temperature of 160°F; and proposed new legislation that would give USDA mandatory recall authority in public health emergencies, such as those involving EHEC.

Food Science. The traditional barriers of time/temperature and acid must be reevaluated. Many of the pathogen's attributes, addressed so well in the status summary by Buchanan and Doyle, are defying food safety conventions.

Public Health. The 1996 outbreak surveillance data from the Centers for Disease Control and Prevention (CDC) indicate that there were only a few clusters of illness associated with contaminated beef. There has been a shift to other sources of contaminated foods, such as alfalfa sprouts, unpasteurized apple juice, and lettuce. The traditional roadside apple cider stand will certainly be affected, and the health benefits of fresh juice and sprouts must now be weighed against the possibility of EHEC infection.

In 1995, CDC, USDA, and FDA initiated the FoodNet active site surveillance program. Preliminary data for 1996 linked sporadic illness to hamburger prepared at home. These data are consistent with the recent outbreak in Colorado, where backyard barbecues and picnics around July 4 resulted in a recognized cluster of illnesses.

Increasingly, epidemics are recognized by another component of FoodNet, molecular fingerprinting. The USDA Outbreak Support Laboratory (Athens, Ga.) was able to fingerprint and match hamburger isolates to the human isolates from Colorado. This information and the subsequent traceback and recall of frozen beef patties may have averted a larger outbreak.

Farming. There is a growing recognition that fruits and vegetables are raw agricultural products that are ready-to-eat and must be treated accordingly. The tradition of recycling manure for vegetable gardening has caused *E. coli* O157:H7 illness; the pathogen can survive longer than the traditional 60-day holding period. Harvesters of produce for raw consumption might well be thought of as food handlers. The produce industry has responded with guidelines for increased worker sanitation as well as training.

International. *E. coli* O157:H7 disease is not unique to the United States. In 1996 alone, the largest reported outbreak, linked to radish sprouts, occurred among school children in Japan. An outbreak in Scotland resulted in the death of 19 people, predominantly pensioners receiving meals catered by the local butcher. In Canada, the traditional school day trip to a dairy farm is associated with EHEC infections. In May 1997, a World Health Organization (Geneva, Switzerland) expert consultation on EHEC prevention and control recommended preventive approaches such as HACCP, applied from farm to table.

What Does the Future Hold? *E. coli* O157:H7 has been a driving force for change in many areas and has shaken tradition; but it remains an enigma. Where did it come from? What happened in 1982? Now that it is here, can it be eradicated? ●