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

SCIENTIFIC STATUS SUMMARY

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 verotoxotxin 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 kDa) and five B subunits (7.7 kDa). 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 primary targets for EHEC.


**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 cases may recur (Siegrist 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, 1992–93 outbreak that affected more 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 has been implicated as a risk factor in those sporadic infections (Le Saux et al., 1993; Pai 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...
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 10^6 CFU/g (Zhao et al., 1995).
  - Fecal shedding of E. coli O157:H7 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 10^6 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 10^6 to 10^2 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 foregut (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.

- **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

<table>
<thead>
<tr>
<th>Food</th>
<th>Outbreak Implicated or Suspected of Pathogen Association</th>
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<tr>
<td>Undercooked ground beef</td>
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<tr>
<td>Raw milk</td>
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<tr>
<td>Unpasteurized apple juice/cider</td>
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<td>Dry cured salami</td>
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<td>Lettuce</td>
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<td>Produce from manure-fertilized garden</td>
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<td>Handling potatoes</td>
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<td>Radish sprouts, alfalfa sprouts</td>
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<tr>
<td>Yogurt</td>
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<tr>
<td>Sandwiches</td>
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<tr>
<td>Water</td>
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E. coli O157:H7 (continued)

of disease throughout the study. Animals perorally administered 10⁸ 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 1992. Two large water mains, broken because of extreme cold weather, and new in-ground water meters may have caused EHEC growth in Cabool, Mo., from December 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., 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). Bathers, 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 medium-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-Rauf 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 Beuchat, 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).

EH EC 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 pO5 expression with associated increases in thermotolerance and H$_2$O$_2$ resistance (Henge-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 E. coli O157:H7 infections. E. coli O157:H7 has been isolated in 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 a complex, since the focus is on risk reduction rather than on preventing pathogen growth. 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 (NACMF, 1993) included E. coli O157:H7 as a hazard. The HACCP plan listed skinning, post-skinning rinsing/bactericidal spray, evisceration, final bacterial 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 mucusosal 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. 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).

Preevisceration 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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**SCIENTIFIC STATUS SUMMARY**

**E. coli O157:H7**

Disease Prevention

- **Antimicrobials.**
- **HACCP.**
- **Farms.**
- **Slaughterhouse.**

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**E. coli**

- **O157:H7**

- **SCIENTIFIC STATUS SUMMARY**
**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.

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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 (D’orsa 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 burnetti* 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$_{90°C}$ 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. Splittoots esser et al. (1995) estimated that the D$_{90°C}$ 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 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 (Rettsma 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 is a method for meat and poultry products, which involves 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 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 foodborne 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

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