An Update: *Escherichia coli* O157:H7 in Humans and Cattle

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Centers for Epidemiology and Animal Health
555 South Howes
Fort Collins, Colorado 80521

Contents

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This report updates a previous report titled ***Escherichia coli* O157:H7: Issues and Ramifications**. The **Executive Summary** of the previous report is included in the appendix of this update for easy reference. The full report may be ordered by mailing your request to CEAH, 555 S. Howes, Suite 200, Fort Collins, CO 80521; or e-mailing your request to NAHMS_info@aphis.usda.gov.

The original report described the role of cattle, specifically ground beef, as a source of *Escherichia coli (E. coli)* O157:H7 in food products. It addressed *E. coli* O157:H7 in humans, cattle and human food, and changes in ground beef production and distribution which may impact human exposure. This update focuses on *E. coli* O157:H7 as a continuing source of illness in humans, improvements in diagnosis, testing and reporting, and reports on the prevalence of *E. coli* O157:H7 in the cattle population as well as post-harvest control measures.

Background

The first recognized *E. coli* O157:H7 outbreak occurred in 1982 in Oregon and Michigan and was associated with eating hamburgers from a particular fast-food chain (1). Evidence indicating rare sporadic infection occurred prior to 1982 comes from a retrospective review by the Centers for Disease Control and Prevention (CDC) of over 3,000 *E. coli* serotypes identified from 1973-1983, in which O157:H7 was detected only once in a 1975 isolate from a 50 year old California woman (1). The subsequent occurrence of large outbreaks and the widespread distribution of cases has led to the designation of *E.coli* O157:H7 as a new, emerging pathogen.

The Disease

The disease caused by *E. coli* O157:H7 is hemorrhagic colitis and is characterized by severe cramping (abdominal pain) and diarrhea (watery and/or bloody). Other symptoms may include vomiting and/or low grade fever. The illness lasts an average of 8 days. Treatment for *E. coli* O157:H7 infection is primarily supportive, including management of dehydration and complications such as anemia and renal failure (3). Antimotility agents should not be given and antibiotic treatment does not appear to diminish the severity of illness or prevent the development of hemolytic uremic syndrome (HUS) (3,4,5). Potential explanations for the lack of benefit for antibiotic treatment are 1) elimination of competing bowel flora by the antibiotic giving a competitive advantage to *E. coli* O157:H7, and 2) lysis/death of *E. coli* O157 leading to increased release of verotoxin (3).

The proportion of all cases of diarrhea estimated to be associated with *E. coli* O157:H7 is 0.6% to 2.4%. Of all cases of bloody diarrhea or hemorrhagic colitis, 15% to 36% are estimated

to be caused by *E coli* O157:H7 (3). Serious complications of *E. coli* O157:H7 disease occur in 0 to 15% of cases and are experienced more frequently by the very young and the elderly. These complications are HUS and thrombotic thrombocytopenic purpura (TTP). HUS primarily affects infants and young children and is characterized by renal failure and hemolytic anemia. HUS is the most common cause of acute renal failure in children and the mortality rate is 5% to 10%. TTP primarily affects the elderly, and is characterized by HUS plus two other symptoms, fever and neurologic symptoms (3). TTP has a mortality rate as high as 50%. (6). Other potential complications are unnecessary surgical intervention, coma or seizures, pancreatitis, and diabetes mellitus (5).

The Bacteria

Escherichia coli (E. coli) are a group of bacteria, which inhabit the intestines of all humans and most animals. Most do not cause disease. *E. coli* O157:H7 is a specific serotype (expressing the 0-antigen 157 and the H-antigen 7) of *Escherichia coli*. The *E. coli* O157:H7 serotype belongs to the enterohemorrhagic *E. coli* (EHEC) group(4). EHEC bacteria contain one or more virulence attributes: the ability to produce shiga-like toxin(s) (SLT also known as verotoxins or VT), adherence factor(s) and enterohemolysin. The adherence factor(s) enables the organism to attach to and colonize intestinal mucosal cells (6).The majority of HUS-associated strains contain a 60-MDa plasmid which encodes the production of enterohemolysin. Another virulence marker is the chromosomal eae gene which encodes the production of intimin, an adherence factor (4,7). Use of DNA probes to detect the genes encoding for these virulence factors, particularly for the production of verotoxins, are the most sensitive EHEC testing methods (6).

E. coli O157 strains which are non-motile are designated as *E. coli* O157:H- or O157:NM. They are missing the H antigen which is the flagellar or motility antigen. They usually also produce verotoxin (VT) and cause the same pattern of disease (4). The two serotypes, *E. coli* O157:H7 and O157:H- , are referred to collectively as *E. coli* O157 VT+ in this report.

An important characteristic of *E. coli O157* VT+, which contributes to its public health significance, is its very small infective dose. Research has indicated the infective dose to be as few as 50-100 bacteria, which is much smaller than for most other foodborne pathogens, including Salmonella (2). This means that contamination with only a few organisms, without subsequent bacterial growth, is sufficient to cause disease.

Non O157 Serotypes

The focus of most research has been on the serotype O157:H7 because of its frequent association with human infections worldwide (8). There are, however, over 60 non-O157 verotoxin producing serotypes. Reports from other countries indicate that some non-O157 serotypes are also important causes of HUS disease (7,8). In 1995 the largest community outbreak of HUS in Australia affected 23 people and was attributed primarily to the serotype 0111:NM. Disease was associated with consumption of an uncooked, semi-dry fermented sausage product produced locally (9). The first reported outbreak of a non-O157 EHEC causing

hemorrhagic colitis in the U.S. occurred in Montana in 1994. Infection with *E. coli* O104:H21 was confirmed in eleven cases and illness was associated with consumption of a particular brand of milk (10). Infections with non-O157 serotypes may be under reported because their isolation requires techniques not generally available in clinical laboratories. Most non-O157 serotypes do ferment sorbitol and therefore are not detected by sorbitol-MacConkey medium screening (4). To detect these serotypes in stool specimens, specialized tests which identify the presence of toxin or toxin genes must be employed (7).

E. coli O157:H7 in Humans

Have outbreaks in the U.S. been increasing?

Since the first recognized U.S. outbreak in 1982, monitoring and reporting of *E.coli* O157: H7 has increased, resulting in an increase in reported O157 outbreaks since that time. However, there has not been an increase in the number of reported outbreaks during the most recent three years (1994-1996) and the total number of ill persons per year due to outbreaks in 1994-1996 was 543, 455, and 488 respectively. Twenty nine outbreaks were reported to the Centers for Disease Control and Prevention (CDC) in 1996 (Table 4). During 1994 and 1995, 32 outbreaks were documented *each* year in the U.S. (Tables 2,3). In contrast, 39 outbreaks were documented for the 11 year period 1982-1993 (Table 1). The likely vehicle for infection remains unknown for 28% of the reported outbreaks in 1994-1996. In 1994 and 1995 the number of outbreaks associated with ground beef were 9 (28%) and 10 (31%) respectively, and decreased to 4 (14%) in 1996 (Tables 2,3,4). Other vehicles identified during 1994-1996 were swimming/recreational water, salami, roast beef, lettuce, venison jerky, apple juice/cider, coney dog sauce, punch and fruit salad. The proportion of outbreaks with a person-to-person mode of spread varied and was (7)18% in 1982-1993, (4) 12% in 1994, (3) 9% in 1995, and (9) 31% in 1996. Only (9) 9.7% of the total 93 outbreaks in 1994-1996 reported in the U.S. were associated with restaurants compared with 15% of outbreaks during 1982-1993 (Tables 1,2,3,4). Outbreaks in 1994-1996 followed a similar seasonal pattern as in previous years, peaking in the warmest months of the year.

Some of the increase in reported cases since 1982 is likely due to improved surveillance and reporting. In 1994, the National Center for Infectious Diseases (NCID) at the Centers for Disease Control and Prevention (CDC), in collaboration with the California, Connecticut, Georgia, Minnesota and Oregon State Health Departments, United States Department of Agriculture (USDA) and Food and Drug Administration (FDA), established Emerging Infections Programs (EIPs). The primary foodborne diseases component of the EIP is the Foodborne Diseases Active Surveillance Network (FoodNet). As part of FoodNet, EIP sites conduct active laboratory based surveillance for verotoxin producing *E. coli* as well as other foodborne diseases in these states. In 1996, results from the 5 EIP sites indicate an incidence rate for *E. coli* O157:H7 infection of 3 per 100,000 population with a range of 0.6 for Georgia to 5 for Minnesota (11). Passive surveillance occurs in many other states, with reporting of *E. coli* O157:H7 infections required by 38 states as of January, 1996 (64).

Prevalence of *E. coli* O157:H7 illness in the U.S. may be underestimated for several reasons. One reason is that, because *E. coli* O157:H7 is cultured by using a specialized media, sorbitol MacConkey (SMAC) agar, it is not easily detected on routine stool culture. In 1993, the Council of State and Territorial Epidemiologists recommended that clinical laboratories begin culturing at least all bloody stools specifically for *E. coli* O157:H7. A nationwide survey conducted 18 months after the above recommendation, determined that only 54% of laboratories were actually following the recommendation (12).

Another reason for under diagnosis is that the organism is shed primarily in the early period of illness and is cleared rapidly from the gastrointestinal tract (7). Therefore *E. coli* O157:H7 may not be detected if a stool culture is not done during the early period of illness. In addition, stool cultures may not be sensitive enough to detect small numbers of the organism. Asymptomatic cases and cases with only non-bloody diarrhea may also go undiagnosed (7).

Table 1. *Reported Outbreaks of E. Coli O157:H7 in the U.S., 1982-1993*

No.	State	Month/Year	Setting	Likely vehicle	No. ill
1.	OR	Feb 1982	Community	Ground beef	26
2.	MI	May 1982	Community	Ground beef	21
3.	NE	Sep 1984	Nursing home	Ground beef	34
4.	NC	Sep 1984	Day-care	Person-to-person	36
5.	NC	May 1986	Day-care	Person-to-person	15
6.	WA	Oct 1986	Community	Ground beef Ranch dressing	37
7.	UT	Jun 1987	Custodial Institution	Ground beef Person-to-person	51
8.	WI	May 1988	School	Roast beef	61
9.	MN	Aug 1988	Day-care centers (9)	Person-to-person	38
10.	MN	Oct 1988	School	Precooked ground beef	54
11.	WA	Aug 1989	Restaurant	Unknown	3
12.	MO	Dec 1989	Community	Drinking water	243
13.	ND	Jul 1990	Community	Roast beef	65
14.	MT	Nov 1990	School	School lunch	10
15.	OR	Jul 1991	Community	Swimming water	21
16.	WA	Aug 1991	Picnic	Ground beef	2
17.	MN	Sep 1991	Fair	Ground beef	8
18.	MA	Nov 1991	Community	Apple cider	23

No.	State	Month/Year	Setting	Likely vehicle	No. ill
19.	NY	May 1992	Unknown	Unknown	5
20.	NV	Jun 1992	Day-care	Person-to-person	57
21.	ME	Sep 1992	Home	Vegetable Person-to-person	4
22.	OR	Dec 1992	Community	Raw milk	9
23.	ID	Jan 1993	Restaurant	Ground beef	13
	NV	Jan 1993	Restaurant	Ground beef	58
	CA	Jan 1993	Restaurant	Ground beef	32
	WA	Jan 1993	Restaurant	Ground beef	629
24.	OR	Mar 1993	Restaurant	Mayonnaise	47
25.	ME	Jun 1993	Unknown	Unknown	4
26.	OR	Jun 1993	Home	Raw milk	6
27.	NC	Jul 1993	Day-care	Person-to-person	27
28.	IL	Jul 1993	Community	Unknown	8
29.	NM	Jul 1993	Party	Unknown	4
30.	MA	Jul 1993	Community	Ground beef	10
31.	WA	Jul 1993	Church picnic	Pea salad	16
32.	CA	Jul 1993	Home	Ground beef	10
33.	OR	Aug 1993	Restaurant	Cantaloupe	27
34.	PA	Aug 1993	Community	Ground beef	3
35.	WA	Aug 1993	Restaurant	Salad bar	53
36.	CT	Sep 1993	Club BBQ	Ground beef	23
37.	MT	Sep 1993	Community	Ground beef	8
38.	WA	Oct 1993	Restaurant	Unknown	9
39.	TX	Oct 1993	Unknown	Unknown	13
				Total	1823

Source: Centers for Disease Control and Prevention

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Table 2. Reported Outbreaks of E. Coli O157:H7 in the U.S., 1994 (These data are subject to change)					
No.	**State**	**Month**	**Setting**	**Likely vehicle**	**No. ill**
1.	WA & OR	Jan	Home	Ground beef	21
2.	MN	Feb	Community	Ground beef	8
3.	NE	Apr	Home/camp	Ground beef	24
4.	ND	May	Restaurant	Ground beef	33
5.	CA	May	Home	Ground beef	9
6.	OH	May	Community	Coney dog sauce	10
7.	NY	Jun	Home	Ground beef	19
8.	CT	Jun	Home	Retail foods	21
9.	CT	Jun	Community	Ground beef	2
10.	PA	Jun	Home	Ground beef	4
11.	OH	Jun	Day-care	Person-to-person	8
12 .	VA	Jul	Community	Unknown	7
13.	VA	Jul	Camp	Ground beef	20
14.	OH	Jul	Community	Unknown	5
15.	WI	Jul	Day care	Person-to-person	43
16.	OK	Jul	Restaurant	Unknown	4
17.	HI	Jul	Unknown	Unknown	17
18.	NY	Jul	Day camp	Unknown	5
19.	MI	Jul	Day care	Person-to-Person	13
20.	NJ	Jul	Homes	Unknown	89
21.	NY	Jul	Community	Swimming water	12
22.	TX	Aug	Cafeteria	Salad bar	26
23.	KY	Aug	Market	Unknown	5
24.	FL	Aug	Unknown	Unknown	9
25.	OH	Aug	Day care	Person-to-person	6
26.	MN	Sep	College	Unknown	11
27.	NY	Sep	Oktoberfest	Unknown	37

No.	State	Month	Setting	Likely vehicle	No. ill
28.	WA	Oct	Home	Unknown	7
29.	WI	Oct	Restaurant	Foodhandler	26
30.	WA & CA	Nov	Home	Salami	19
31.	NM	Nov	School	Unknown	20
32.	NY	Jul	Restaurant	Unknown	3
				TOTAL	543

Source: Centers for Disease Control and Prevention

Table 3. Reported Outbreaks of E. Coli O157:H7 in the U.S., 1995
(These data are preliminary and subject to change)

No.	State	Month	Setting	Likely vehicle	No. ill
1.	OR	Mar	Day-care	Person-to-person	4
2.	MN	May	Picnic	Ground beef	2
3.	NC	May	Day care	Person-to-person	33
4.	MN	May	Home	Ground beef	4
5.	SD	Jun	Camp	Ground beef	3
6.	GA &TN	Jun	Restaurant	Ground beef	8
7.	IL	Jun	Lake	Swimming	12
8.	CO	Jun	Day care	Person-to-person	25
9.	WI	Jun	Lake	Swimming	8
10.	MT	Jul	Community	Leaf lettuce	74
11	NY	Jul	Day care	Ground beef	12
12.	NY	Jul	Camp	Unknown	5
13.	CO	Jul	Camp	Ground beef	21
14.	MN	Jul	Lake	Swimming	6
15.	MN	Jul	Lake	Swimming	2
16.	MN	Jul	Camp	Water	9
17.	MA	Jul	Fair	Ground beef	8
18.	ID	Aug	Lake	Swimming	4
19.	WI	Aug	Festival	Ice	27

No.	State	Month	Setting	Likely vehicle	No. ill
20.	CT	Aug	Camp	Unknown	24
21.	MN	Aug	Church	Roast beef	31
22.	ME	Sep	Camp	Lettuce	37
23.	ID	Sep	Restaurant	Lettuce	12
24.	WA	Sep	Home	Ground Beef	2
25.	KS	Oct	Wedding	Punch, Fruit salad	21
26.	OH	Oct	Community	Unknown	11
27.	NY	Oct	Home	Ground beef	2
28.	OR	Nov	Home	Venison jerky	11
29.	VT	Nov	Home	Unknown	3
30.	MN	Nov	Home	Ground beef	5
31.	IL	Nov	Church	Unknown	4
32.	CA	Dec	Prison	Unknown	5
				TOTAL	455

Source: Centers for Disease Control and Prevention

Table 4. Reported Outbreaks of E. coli O157:H7 in the U.S., 1996 (These data are preliminary and subject to change)					
No.	**State**	**Month**	**Setting**	**Likely vehicle**	**No. ill**
1.	TX	Apr	Home	Ground beef	3
2.	CT & IL	May	Home	Lettuce	47
3.	WA	Jun	Pool	Swimming	4
4.	MN	Jun	Lake	Swimming	8
5.	NY	Jun	Restaurant	Unknown	61
6.	MI & OH	Jun	Restaurant	Unknown	10
7.	NH & MA	Jun	Community	Unknown	29
8.	MN	Jun	Day care	Person-to-person	7
9.	OR	Jun	Picnic	Unknown	38
10.	NY	Jun	Nursing home	Person-to-person	5
11.	PA	Jun	Day-care	Person-to-person	3

No.	State	Month	Setting	Likely vehicle	No. ill
12.	NC	Jun	Day care	Person-to-person	2
13.	NV	Jul	Party	Ground beef	2
14.	GA	Jul	Pool	Swimming	18
15.	MO	Jul	Community	Unknown	3
16.	PA	Aug	Party	Ground beef	9
17.	MN	Aug	Day care	Person-to-person	8
18.	MS	Aug	School	Person-to-person	36
19.	MN	Aug	Day care	Person-to-person	63
20.	VT	Sep	Fair/Festival	Unknown	11
21.	RI	Sep	Community	Unknown	5
22.	NY	Sep	Day care	Person-to-person	9
23.	OR	Sep	Restaurant	Ground beef	7
24.	CA, WA, CO	Oct	Community	Apple juice	71
25.	CT	Oct	Home	Apple cider	14
26.	MN	Oct	Day care	Person-to-person	3
27.	WA	Oct	Fair/Festival	Apple cider	6
28.	IL	Nov	Home	Venison	2
29.	OR	Dec	Home	Venison	4
				Total	488

Source: Center for Disease Control and Prevention

Do outbreaks of *E. coli* O157:H7 occur in other countries?

E. coli O157:H7 has been isolated from humans in many places other than the U.S., including South Africa, Australia, Argentina, Canada, Chile, China, Czechoslovakia, France, Germany, India, Ireland, Italy, Japan, and the United Kingdom (4). The majority of reported outbreaks in other countries have occurred in Canada and the United Kingdom (UK) (65). In July/August 1996, a large outbreak occurred in Japan which affected over 9000 people, many of them school children (15). Cases occurred in 40 prefectures and several food sources were implicated, particularly watercress. The U.K. experienced its largest outbreak of *E. coli* O157 infection in November/December 1996. The outbreak occurred in Scotland, with 408 persons reported to have symptoms, 256 confirmed cases and 16 deaths. The vehicle was traced to cooked beef and gravy from a specific butcher shop. (65)

Though beef products are an important vehicle of transmission of *E.coli* O157:H7, what other vehicles have been linked to outbreaks?

In 1994, 12.5% of outbreaks were linked to food sources other than beef products; in 1996 the proportion increased to 21% (Tables 2,3,4). The proportion of outbreaks linked to beef products has decreased, from 28% in 1994 to 14% in 1996 (Tables 2,3,4). Some of these unexpected foodborne vehicles of transmission are acidic foods, salad vegetables, lettuce and venison (Tables 2,3,4). The acidic foods confirmed as sources of outbreaks include unpasteurized apple juice and apple cider, mayonnaise and yogurt (8). Fresh-pressed, unpasteurized, unpreserved apple cider was first identified as a vehicle for *E. coli* O157:H7 in an outbreak in Massachusetts in 1991, although HUS was first linked to apple juice in 1982 (13). In October 1996, two separate outbreaks associated with drinking unpasteurized apple cider occurred, one in Connecticut and the other in the western U.S. The Connecticut outbreak involved 14 cases and was associated with drinking a specific brand of cider (16). The second outbreak involved 66 persons in multiple states in the western U.S. and was associated with drinking a specific brand of unpasteurized apple juice or the brand's juice mixtures containing apple juice (17).

The ability of *E. coli* O157:H7 to survive in low pH and at low temperatures has been documented in several laboratory studies (18,19,20). Miller and Kasper (18) inoculated apple cider with O157:H7 strains which were still detectable after 14-21 days at 4° C. The O157:H7 strains also withstood a pH of 2 and survival was generally greater at 4° C than 25° C. Conner and Kotrola (19) found that *E. coli* O157:H7 survived in acidic conditions (pH >= 4.0) for up to 56 days and survival was affected by type of acidulant and temperature.

Salad vegetables have also been implicated as an outbreak vehicle (Tables 1,2). Populations of viable *E. coli* O157:H7 inoculated onto vegetables declined when vegetables were stored at 5° C and increased on vegetables stored at 12 and 21° C for up to 14 days (21). Dry cured salami was implicated as the vehicle in an outbreak in the state of Washington (23) and venison jerky was reported as the likely vehicle for an outbreak in Oregon in 1995 (74). Consumption of deer steak is being investigated as the cause of *E. coli* O157:H7 illness in two individuals in Illinois in early 1997 (24).

Raw milk can be a vehicle of transmission for E. coli O157:H7 but confirmed outbreaks have been few. The presumed mechanism of contamination is fecal contamination during milking. Two outbreaks associated with raw milk have been documented by the CDC (Table 1), one in 1992 with 9 cases and the other in 1993 with 6 cases. Both outbreaks occurred in Oregon and were traced to two specific dairies which were licensed to sell raw milk (76). The estimated number of raw milk drinkers in the U.S is only 1 to 2 percent (76). This small population at risk may partly explain the small number of outbreaks due to raw milk consumption. Pasteurization kills E. coli O157:H7 and is an effective way to prevent milkborne transmission (14).

Both drinking water and recreational water have been linked to outbreaks (24,25). The only known outbreak in the U.S. associated with drinking water occurred in 1989 in Missouri. An unchlorinated municipal water source and deficiencies in the water distribution system were

implicated as the probable source of contamination (25,26). Outbreaks associated with freshwater swimming/recreational areas have been more frequent. During 1982-1994, only 2 (2.8%) outbreaks associated with swimming water were identified (Tables 1,2). During 1995-1996, however, there were 8 (13%) associated with swimming water (Tables 3,4).

The importance of person-to-person spread should not be overlooked. During 1994-1995 in the U.S., person-to-person spread was identified as the likely vehicle in 7 (11%) outbreaks (Tables 2,3). In 1996 there were 9 (31%) outbreaks attributed to person-to-person spread. The most frequent setting for person-to-person spread is a day-care facility, but person-to-person spread has occurred in other institutional settings such as nursing homes and mental health facilities, and is common among family members (27). A small, recent outbreak involving 5 cases of *E. coli* O157:H7 in Florida involved 2 cousins and 3 siblings. The 2 cousins contracted *E. coli* O157:H7 during international travel and, upon return to the U.S., had contact with the three siblings who became affected (28). Person-to-person transmission from asymptomatic cases also occurs (2).

What are the risk factors for human cases of *E. coli* O157:H7?

E. coli O157:H7 infections occur in all age groups, with the highest incidence rate in children less than 5 years old. In 1987, the first year that Washington State required reporting of *E. coli* O157:H7 infection, the highest age-specific incidence rate was among children younger than 5 years (6.1 cases per 100,000 population per year) and lowest for adults 50-59 years of age (0.5 cases per 100,000) (29). Higher incidence of infection in young children may in some part be due to the greater likelihood of their being brought to medical attention. Variation of incidence rates by age may also be related to variation of exposure to the agent by age. The demographic profile of people exposed to a specific vehicle (such as swimming water) affects the demographic profile of outbreak cases. The young and the elderly are more often affected by the serious complications of *E. coli* O157:H7 infection, HUS and TTP. Consequently, the young and the elderly have the highest morbidity and mortality rates from *E. coli* O157:H7 infection (3).

The most commonly identified risk factor in case-control studies of sporadic *E. coli* O157:H7 illness was consumption of undercooked ground beef. Other risk factors identified were consumption of ground beef in a non-commercial setting such as a picnic or “special event”, drinking of well water, swimming, handling animal feces, close contact with a person with diarrhea, and failure to wash one’s hands after handling raw ground beef (2). In 1992-1993 the Food and Drug Administration sponsored a national telephone survey of 1,620 respondents at least 18 years of age, to assess the prevalence of selected self-reported food consumption and preparation behaviors associated with increased risks of food-borne illness. Consumption of undercooked hamburger was reported by 23% of respondents, and 25% of respondents reported that after cutting raw meat or chicken, they use the cutting board again without cleaning it. These results indicate that unsafe food preparation and consumption behaviors are common in the U.S. (33).

Reymond et al. (30) used assays to detect antibodies to *E. coli* O157 lipopolysaccharide (LPS) and verotoxin 1 (VT1) to determine and compare exposure of dairy farm residents in

southern Ontario, Canada and in urban residents of Toronto, Canada. The frequency of O157 LPS antibodies was significantly higher in dairy farm residents (12.5%) than in urban residents (4.7%). The difference between the groups was even greater for VT1 neutralizing antibodies, with detection in 42% of dairy farm residents and only 7.7% in urban residents. These findings indicate that dairy farm residents are at higher risk for *E. coli* O157 VT+ exposure.

A case-control study was conducted in an Inuit community in northern Canada to evaluate risk factors for childhood HUS and gastroenteritis during an epidemic of *E. coli* O157:H7 infection in 1991. Results of the study indicated that in the 7 days before the onset of gastrointestinal symptoms, children with HUS and those with uncomplicated gastroenteritis were 9 times more likely to have been exposed to a family member with diarrhea than were the healthy control subjects (27). This study illustrates the importance of person-to-person transmission.

What are the latest testing methods?

It is difficult to detect *E. coli* O157:H7 in raw meats and food due to much higher levels of other sorbitol non-fermenting bacteria. Isolation of the organism from food or stool specimens involves first enrichment in a selective broth and then plating onto sorbitol MacConkey agar with additives (3,4). Biochemical and serological confirmation tests are then done. Immunomagnetic beads coated with specific O157 antibody can be used to enhance isolation (32). Also, enzyme linked immunosorbent assay (ELISA) methods are used to isolate and identify suspect colonies (31). Another method to detect *E. coli* O157:H7 is to look for verotoxin or verotoxin genes. Immunoassay methods are available to identify the presence of verotoxins(3). Testing for verotoxin can also be done using toxin specific antibodies and genes using DNA probes (3,36). Testing for verotoxin can identify verotoxin producing serotypes other than O157:H7.

Identification of *E. coli* O157:H7 can also be done rapidly, specifically and sensitively using DNA based polymerase chain reaction (PCR) methods. One multiplex PCR method amplifies simultaneously three different DNA sequences of E. coli O157:H7: a specific fragment of the eae gene, conserved sequences of verotoxins I and II, and a fragment of the 60-MDa plasmid (66) . Since this test detects other virulence markers besides verotoxin, it is more specific than tests which only identify verotoxin genes. PCR methods however are affected by many laboratory variables and are less reproducible between laboratories than other methods, and are often less sensitive than direct culture.

Molecular methods for interstrain differentiation of *E. coli* O157:H7 have been developed. These methods are useful in distinguishing between outbreak related and unrelated isolates. The most commonly used DNA fingerprinting tests are based on restriction fragment length polymorphism (RFLP) methodology where restriction enzymes are used to cut genomic DNA into fragments that are separated by agarose gel electrophoresis. A pattern or “fingerprint” is resolved for particular bacterial strains. Several RFLP methods have been developed, one uses pulsed-field gel electrophoresis (PFGE)(34), others use conventional gel electrophoresis (35).

E. coli O157:H7 in Cattle

What is the animal and herd prevalence of *E. coli* O157 VT+ in cattle?

Individual animal prevalence estimates have risen slightly since the first studies of cattle in the U.S. in 1991 indicated a prevalence of approximately 0%-3%. This may be due to improved sensitivity of diagnostic tests used in more recent studies and may not represent a real change. Within-herd prevalence for herds identified through tracebacks from human O157 cases, or herds that were previously identified as positive ranged from 1.3% to 9.5% (Table 5). In herds which were not tracebacks or previously identified as positive, within-herd prevalence was slightly lower, and ranged from 0% to 6.1%. Results from recent studies which incorporated a longitudinal design of repeat sampling of herds over time, indicate that herd prevalence is greater than previously thought, ranging from 22% to 100% (Table 5). The *E. coli* O157 VT+ organism is widely distributed geographically across the U.S., both in dairy and feedlot cattle populations and prevalence tends to be higher in the warmer months (37-39).

E. coli O157 VT+ causes no disease in cattle and has been found in all age groups. Hancock et al (37) found the prevalence of O157 to be higher in weaned heifers (1.8%) than in preweaned calves (0.9%) or adults (0.4%). Gut colonization is transient, with a median shedding duration of less than 30 days (67).

Table 5. *Escherichia coli* O157 Verotoxin Positive (VT+) Healthy Cattle Sampling Studies

Period/ Site	Number & Type of Animal/Premises	Animal Prevalence	Herd Prevalence	Comments	Reference
1986 WI	226 dairy cattle 2 premises	2.2%	100%	O157:H7 Both premises were tracebacks*	Wells, et al. 1991 (40)
	428 dairy cattle 11 premises	1.2%	27.3%		
	46 dairy cattle 1 stockyard	2.2%	NA	Stockyard was a traceback*	
1987 WA/OR	539 dairy cattle 11 premises	1.3%	55.5%	O157:H7 All premises were tracebacks*	Wells, et al. 1991 (40)
	27 dairy heifers and calves 1 packing house	0	NA	Packing house was a traceback*	
1987 England	207 cattle abattoir	1.0%		O157 VT+ Fecal samples from randomly selected cattle	Chapman, et al. 1989 (41)
1988 Canada	665 cattle	0-1.50%		O157:H7 Fecal samples from healthy cattle submitted to lab, 1985-1991	Clarke et al. 1991 (42)

Period/ Site	Number & Type of Animal/Premises	Animal Prevalence	Herd Prevalence	Comments	Reference
1988 Canada	200 beef cattle 200 cull dairy cows 200 veal calves at slaughter	1.5% 0.5% 0		O157:H7 Fecal samples from randomly selected animals over a 10-week period	Clarke, et al. 1988 (43)
1990 Germany	212 bulls 47 cows slaughterhouse	0.9% 0		O157:H7 Fecal samples taken at slaughter	Montenegro et al., 1990 (44)
1991 WA	3570 dairy cattle 60 premises 1412 pastured beef cows 25 premises 600 feeder cattle 5 feedlots	0.3% 0.7% 0.3%	8.3% 16.0% 40.0%	O157:H7 All dairy positives found from Jun-Sept.	Hancock et al. 1994 (45)
1991-1992 U.S. 28 states	6894 dairy calves 1068 premises	0.4%	1.8%	O157:H7 National Study randomly selected operations	NAHMS, 1994 (46)
1993	171 calves 24 hours old to weaning 132 calves weaning to 4 months old 14 case herds-11 states 399 calves 24 hours old to weaning 263 calves weaning to 4 months old 50 control herds-14 states	2.9% 5.3% 1.5% 4.9%	50% 22%	Follow-up to above study: Sample of positive & negative herds from original sample retested	Zhao, et al. 1995 (47) Garber, et al. 1995 (48)
1992 England	1055 cattle abattoir	3.6%		O157 VT+ Rectal swabs taken immediately after slaughter* origin of positive cattle geographically diverse	Chapman, et al. 1992 (73)
1992-1993 Canada	886 dairy cows 592 calves under 3 months of age 80 premises	0.45% 1.7%	10.0%	O157:H7 Identical isolates in humans support cattle-human transmission	Renwick et al., 1993 (72)
1993 Spain	112 calves	1.79%		O157 VT+ Sample of healthy calves from small family farms	Blanco, et al. 1993 (50)
1993 England	105 dairy cattle	9.5%		O157 VT+ Rectal swabs, dairy was a traceback from contaminated milk*	Chapman, et al. 1993 (49)

Period/ Site	Number & Type of Animal/Premises	Animal Prevalence	Herd Prevalence	Comments	Reference
1993-1994 WA	5148 dairy cattle follow up of 5 positive herds 3763 dairy cattle follow up of 8 negative herds	1.9% 0.2%	80.0% 50.0%	O157 VT+ Samples collected monthly for 13 months, highest prevalence occurred in summer	Hancock, et al. 1997 (37)
1994 WA/CA/WI	304 calves< 10 days old slaughterhouses	0		O157:H7 Rectal swabs from calves brought to slaughter	Martin, et al. 1994 (51)
1994 U.S. 13 states	11,881 samples 100 feedlots	1.61% of fecal swab samples from pen floor positive	63.0%	O157:H7 Feedlots randomly selected	NAHMS, 1995 (52) Hancock, et al. 1997, (39)
1994 WI	560 weaned dairy calves < 4 months 70 premises 517 cattle of various ages (Follow up - 5 positive farms and 7 negative farms)	1.8% 3.7%	7.1% 50.0%	O157:H7 Prevalence survey Follow-up Study: Sample of positive & negative herds from original sample retested	Faith, et al. 1996 (53)
1995 WA, OR, ID	6 dairy premises 1097 fecal samples 6 feedlots 1046 fecal samples	1.1%-4.4% 1.5%-6.1%	100%	O157 VT+ Each premise sampled 3 times over a 2-3 month period, 60 fecal pat samples collected on each visit	Hancock, et al. 1997 (71)
1995 WA, OR, ID	heifers 36 dairy premises 12,664 fecal samples	0.5%-2.0%	75%	O157 VT+ ~60 fecal samples collected monthly from each premise for 6 months, tendency for herds to maintain relatively low or high prevalence status	Hancock, et al. 1997 (38)
1995 WA, OR, ID	205 cull cows 19 herds, sampled at farm 103 cull cows 15 herds, sampled at slaughter 89 cull cows (subset of above), sampled at both farm and slaughter	3.4% 3.9% 7.9% positive at either farm, slaughter, or both		O157VT+ Fecal samples from total of 219 cattle, sampled either on farm, at slaughter, or both	Rice, et al. 1997 (70)
1995 NY	1602 ambulatory cull dairy cows 67 non-ambulatory cull cows sampled at slaughter	0.9% 3.0%		O157:H7 fecal samples	Wheeler, Rossiter 1996 (77)

*Premises or abattoir believed to be potential sources of O157, based on traceback from human O157 cases

What are the risk factors for *E. coli* O157 VT+ shedding in cattle?

Based on studies conducted to date, *E. coli* O157 VT+ shedding in cattle is multifactorial (Table 6). No specific factor stands out as the major risk factor for shedding. Multiple studies have identified several dietary factors as either positively or negatively associated with shedding (Table 6). Cray et al. (54) found that 4 calves fasted for 48 hours prior to oral inoculation with *E. coli* O157:H7, shed the organism for a significantly longer time period compared to 4 nonfasted controls. Dietary stress and feed type may alter *E. coli* shedding through their effects on the ruminal environment. These factors alter the concentration of volatile fatty acids and pH in the rumen, which in turn influence bacterial growth (55,2).

Another area of current research is the role of environmental factors such as *E. coli* VT+ contaminated water and feed (53,56) on the epidemiology of *E. coli* O157 VT+ shedding on the farm. Hancock et al. sampled trough water and associated biofilms, and cattle feed on 6 dairies and 6 feedlots in the Pacific Northwest (71). *E. coli* O157 was found in 4 trough water samples and 6 water trough biofilm samples. *E. coli* O157 was not found in any feed samples. Faith et al. (53) also identified animal drinking water as a source of *E. coli* O157:H7 in their study of dairy farms in Wisconsin. The effect of manure handling practices such as application of manure to cattle forage crops, and cattle housing, were examined as risk factors for E. coli O157 shedding in 36 dairy herds in the Pacific Northwest (38). Application of manure to cattle forage crops or pasture was not associated with the prevalence of E. coli O157.

Table 6. Association of *E. coli* O157 with Selected Management Practices[*]

Management Practice	Subgroup	Association with O157
Small herd size (45)	Dairy farms	pos
Use of computerized feeders (45)	Dairy farms	pos
Irrigation of pastures with manure slurry (45,38)	Dairy farms	pos, none
Feeding of whole cottonseed (45,48)	Dairy heifers and cows	neg
Feeding of milk replacer (48)	Dairy calves	neg
Feeding of ionophores (48,68)	Dairy calves	none, pos
Grouping of calves prior to weaning (48)	Dairy calves	pos
Sharing of unwashed feeding utensils among calves (48)	Dairy calves	pos
Feeding of oats in starter ration (48)	Dairy calves	pos
Feeding of grain during first week of life (48)	Dairy calves	pos
Feeding of clover as first forage (48)	Dairy calves	neg
Gradual weaning (68)	Dairy calves	neg
Feeding of corn silage (68)	Dairy heifers	pos
Increasing # of bulls/steers on farm (68)	Dairy farms	pos
Pens $\geq$ 85% beef type heifers (69)	Feedlot	neg
Feeding of barley (69)	Feedlot	pos
Feeding of soybean meal (69)	Feedlot	neg
On feed < 20 days (69)	Feedlot	pos
Entry weight $\geq$ 700 pounds (69)	Feedlot	neg

[*] Many other management factors have been tested for association with O157; only those listed were found to have statistical significance at $p \leq 0.10$.

pos = positive association, i.e., management practice is associated with increased O157 prevalence
neg = negative association, i.e., management practice is associated with decreased O157 prevalence
none = no association

What animals other than cattle shed *E. coli* O157:H7?

Sheep have recently been documented to naturally shed *E. coli* O157:H7. Fecal samples were taken from 35 free-ranging sheep from a flock in Idaho over a 6- month period (June-November) and tested using methods sensitive for *E. coli* O157:H7. Incidence of *E. coli* O157:H7 was seasonal and varied from 11 (31%) positive of 35 samples obtained in June, 2 (5.6%) positive of 35 samples obtained in August, and none (0%) positive in November (57). Researchers in Britain surveyed 700 sheep at slaughter (100 a month for 7 months) and isolated *E. coli* O157:H7 from rectal swabs of 18 (2.6%) (58). Bulaga et al. determined a 0.4% prevalence of fecal shedding of *E. coli* O157:H7 in 499 adult sheep at a livestock market in New Jersey. The two positive sheep were part of a group of 253 sheep which had traveled greater than 12 hours to market. No positive samples were detected in the 246 sheep which had traveled less than 3 hours to the market. Though the sample size is small, this study supports the postulated effect of transport distance on shedding prevalence (74). In a study of market lambs at a midwestern slaughter establishment, McCluskey et al. determined a 0.9% prevalence of *Escherichia coli* O157:H7 in 882 lambs (78). This study examined production system, transit time and time held prior to slaughter, as possible risk factors for fecal shedding. Of the 882 lambs, 56% were from commercial feedlots and 7 (88%) of the 8 *E. coli* O157:H7 positive lambs originated in commercial feedlots. The prevalence of *E.coli* O157:H7 shedding was 0.68% among lambs with a total farm to slaughter time equal to or greater than 18 hours and was 0.23% among lambs with a total farm to slaughter time of less than 18 hours.

A study was conducted in the Pacific Northwest to examine the occurrence of non-bovine sources of *E. coli* O157:H7 on the farm. All domestic and wild animals on 6 dairies and 6 feedlots were sampled. Samples that were culture positive for *E. coli* O157:H7 included 1 horse, 2 dogs, 1 pooled bird sample and 2 pooled fly trap samples. All rodent samples were negative. Farms with positive non-bovine samples also had positive cattle samples (71). In addition, 2 deer samples from 538 samples from deer and /or elk which shared ranges with cattle in Oregon, South Dakota, Texas, and Washington were positive (56). The above studies indicate that other species can harbor *E. coli* O157:H7.

The 1995 National Animal Health Monitoring System swine study included testing of fecal samples from 4,229 swine from 152 randomly selected pork operations in the 16 top swine-producing states. None of the samples were positive for *E. coli* O157:H7. Based on this result and the statistical design of the study, it was estimated that if *E. coli* O157:H7 does exist in hogs, it is shed by less than 0.07% of the population. (written communication, Dr. Eric Bush, USDA, APHIS).

E. coli O157:H7 Prevention

What control mechanisms are currently used during beef processing?

New controls are being enacted to ensure an increasingly safe food supply. One or more new interventions (since 1994) applied during the slaughtering process to reduce microbial contamination are used on at least 80% of beef carcasses produced in the U.S.(79). These new interventions include pre-evisceration washing (80), steam vacuuming to remove visible external contamination (82), organic acid washing (81), and steam pasteurization (83). Steam pasteurization is a patented process used just prior to carcasses entering the cooler. Carcasses are placed into a special chamber which is slightly over atmospheric pressure, and steam condenses on the carcass at 200 degrees Fahrenheit. Other antimicrobial agents which can be used on carcasses are trisodium phosphate and chlorinated water (82).

The Food Safety and Inspection Service (FSIS) of the USDA has recently established new requirements for all meat and poultry plants to improve food safety. All slaughter and processing plants in the U.S. will be required to adopt a system of process controls known as Hazard Analysis and Critical Control Points (HACCP). Adoption of the HACCP program will be phased in over a three year period beginning January 1998. Large plants are required to have their HACCP system in place by January 26, 1998, medium plants by January 25, 1999 and small plants by January 25, 2000. The new program also requires the plant, beginning January 27, 1997, to conduct microbial testing for generic *E.coli* as an indicator of fecal contamination. Fecal contamination is the primary pathway for contamination of meat and poultry with harmful bacteria (59).

From October 1992 to September 1993, FSIS conducted the Nationwide Beef Microbiological Baseline Data Collection Program (61). The program's goal was to provide a national microbial profile of steer and heifer carcasses from fed cattle which included the number and types of bacteria recovered. Tissue samples representing approximately 2,100 steer or heifer carcasses were collected from federally inspected establishments that slaughtered an average of approximately 40 or more fed cattle per week. *E. coli* O157:H7 was recovered from 0.2% of 2,081 carcasses. Cow and bull carcasses were tested as part of the above program from December 1993 to November 1994. Tissue samples from 2,112 cow or bull carcasses were collected from establishments operating under Federal inspection. These establishments are responsible for approximately 99% of all cows and bulls slaughtered, and 18% of total beef animals slaughtered under Federal inspection. *Escherichia coli* O157:H7 was not recovered from any of the 2,112 carcasses.

The Nationwide Federal Plant Raw Ground Beef Microbiological Survey (60) was conducted from August 1993 to March 1994. The purpose of this FSIS survey was to estimate the national prevalence and levels of bacteria of public health concern in raw ground beef as currently produced in federal plants. One randomly selected pound of ground beef was requested from each of 789 randomly selected plants operating under Federal inspection. Over a 7-month period approximately 600 samples were received. Microbiological analyses were performed on a

25 gram portion of the original sample. *E. coli* O157:H7 was not recovered from any of the 563 samples analyzed.

FSIS commenced an ongoing microbiological testing program for *E. coli* O157:H7 in raw ground beef in October 1994 (62). This program is intended to stimulate industry actions to reduce the presence of *E. coli* O157:H7 in raw ground beef prepared at federally inspected establishments and at the retail level. The program's goal is to test 5,000 samples of ground beef, from federally inspected plants and from manufacturers of ground beef products in retail stores, for the presence of *E. coli* O157:H7. In 1995, 5291 samples of raw ground beef were tested for this program and 3 (0.06%) positives were found. In 1996, 5326 samples were tested and 4 (0.08%) positives were found (84). Confirmed positive results lead to regulatory action, since E. coli O157 on raw ground beef is considered an adulterant.

Though these programs will help to improve processing safety, they cannot provide a guarantee of an individual product's safety. The most important preventive measures involve thorough cooking of beef and hygienic food handling by the consumer to prevent cross-contamination in the home.

What should consumers do to prevent *E. coli* O157:H7 infection?

The following preventive measures are recommended by the Centers for Disease Control and Prevention (63):

- Cook all ground beef or hamburger thoroughly until the meat is gray or brown throughout and juices run clear.
- When dining in restaurants, send undercooked hamburger back for further cooking.
- Avoid raw, unpasteurized milk and milk products.
- To reduce person-to-person transmission, make sure that infected persons, especially children, wash their hands frequently with soap.
- Make sure your drinking water has been treated with adequate levels of chlorine or other effective disinfectants.

Conclusion/Summary

The number of *E. coli* O157 outbreaks reported in the U.S. has not increased during the last three years (1994-1996). Ground beef continues to be an important foodborne vehicle of transmission for outbreaks, though novel vehicles such as unpasteurized apple juice, salad vegetables, deer meat and recreational/swimming water are increasingly recognized. Cross-contamination of non-bovine food sources at the farm, post-harvest and during food preparation, contamination of swimming water and person-to-person transmission are important factors leading to outbreaks.

Prevalence of *E. coli* O157:H7 is widespread geographically in both dairy and beef cattle. Results of recent studies which incorporated repeat sampling of herds over time, indicate that herd prevalence can be as high as 100%. The individual animal prevalence remains low and

tends to range from 0.5%-5.0% in both dairy and beef cattle. Shedding of the organism in cattle is transient and multifactorial. Recent research has focused on farm management and environmental factors which may influence *E. coli* O157 VT+ shedding, as well as identification of other species besides bovines which may shed *E. coli* O157 VT+. Efforts by USDA, universities and production and processing industries will continue to assure a safer food supply.

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Escherichia coli O157:H7

Issues and Ramifications

Executive Summary

March 1994

USDA:APHIS:VS
Centers for Epidemiology and Animal Health
555 South Howes
Fort Collins, Colorado 80521

To obtain a copy of the complete report entitled "*Escherichia coli* O157:H7 -- Issues and Ramifications" please contact:

Centers for Epidemiology and Animal Health
USDA:APHIS:VS
555 South Howes, Suite 200
Fort Collins, Colorado 80521

Area Code (303) 490-7812

Executive Summary

Escherichia coli O157:H7 -- Issues and Ramifications

This document summarizes findings presented in the report entitled "*Escherichia coli* O157:H7 -- Issues and Ramifications[1]." The primary purpose of that report is to help define the role of cattle as a source of *E. coli* O157:H7 in food products. Although different modes of transmission from cattle to humans are discussed in the report, it concentrates on the vehicle most frequently implicated in human disease outbreaks, ground beef. This summary is divided into four sections: (1) Why the interest in *E. coli* O157:H7?, (2) What is known about *E. coli* O157:H7 in cattle?, (3) Do production and consumption patterns for ground beef offer any additional insight into *E. coli* O157:H7?, and (4) Future directions.

Why the interest in *E. coli* O157:H7?

Escherichia coli O157:H7 (O157) was first identified as a human pathogen capable of causing foodborne illness in 1982. However, the public was generally unaware of the existence of 0157 until a decade later. In late 1992, an outbreak associated with the consumption of undercooked hamburgers began in Washington state. The more than 600 illnesses and the subsequent deaths of 4 children were publicized throughout the country. In addition, evidence suggesting that the frequency of O157 illness in humans is increasing has heightened concern. Of the 32 outbreaks reported in the U.S. from 1982 through 1993, 13 occurred in 1993.

Human illness associated with O157 is infrequent in comparison to illness associated with some other foodborne pathogens such as *Salmonella*. However, the range in severity of clinical illness and the potential for debilitating complications and death makes O157 a noteworthy food safety issue. The abdominal cramping and bloody diarrhea typical of O157-associated disease result from toxin production and subsequent destruction of the mucosal lining of the colon. In most patients, the disease is self-limiting. However, a small percentage of O157 cases progress to hemolytic uremic syndrome (HUS) and/or thrombotic thrombocytopenic purpura (TTP). The elderly and children less than 5 years old are at highest risk of developing these complications. Such cases may result in kidney failure or death.

Although not definitively established, it is believed that O157 inhabits the lower intestine of cattle and is shed in the feces. Human infection with O157 occurs primarily through ingestion of food contaminated with fecal material. Another recognized source of infection is O157-contaminated water. Human-to-human and calf-to-human transmission have also been documented.

Although O157 is one of many serotypes of a common and ubiquitous bacteria, a unique characteristic of O157 is the organism's hardiness. It can survive for extended periods in water, meat stored at subfreezing temperatures, acidic environments, and soil. The organism is, however, destroyed by thorough cooking or pasteurization.

A variety of foods have been implicated in O157-associated illnesses. Of the 24 outbreaks associated with foods, 17, or 71 percent, have been linked to bovine products. Contaminated ground beef was associated with 12 of the outbreaks, raw milk and roast beef with 2 each, and 1

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[1] References are not given in this summary document but may be found in the complete version of "*Escherichia coli* O157:H7 -- Issues and Ramifications."

with hot dogs containing beef. Cross-contamination of other foods, including apple cider, vegetables, and mayonnaise, by manure or meat products has been confirmed or is suspected in the seven other foodborne outbreaks.

Although not directly linked to human illness, several other meat and poultry products have been sampled for O157. In addition to beef, the organism has been isolated from veal kidneys, poultry, pork, and lamb. However, cross-contamination of these meat products is considered likely. To date, farm-level testing in the U.S. has concentrated on cattle. As a result, the status of O157 in other food animal species is not known.

There is no definitive evidence of a geographic pattern of human O157 cases. However, a 2-year study concluded that a significantly higher percentage of stool samples were O157-positive from hospitals in the northern and western U.S. than in the southern part of the country.

Both O157 sporadic cases and outbreaks have a definite seasonal pattern. The four largest studies in the U.S. have revealed that at least 67 percent of sporadic cases occurred between May and September, with a peak in July and August. Of all U.S. outbreaks associated with O157, 88 percent have occurred from May through November.

At least 16 countries on 6 continents have documented human cases or bovine isolates of O157, indicating the widespread presence of the organism. Outside of the U.S., most occurrences of O157 illness have involved sporadic cases; only Canada and the United Kingdom have reported outbreaks. As in the U.S., cases have generally peaked in the summer and fall months.

What is known about *E. coli* O157:H7 in cattle?

The epidemiologic link between human O157-associated illness and products of bovine origin has raised many questions concerning the occurrence of the organism in the cattle population. Beyond the observation that O157 is not known to cause clinical disease in cattle under natural conditions, little is known about the on-farm ecology of the organism. Analysis of O157 on-farm studies indicates that virtually all types and breeds of cattle should be viewed as potential sources of O157 contamination. Changes in various management practices which may have allowed or enhanced the ability of the O157 organism to inhabit the gastrointestinal tract of cattle are under investigation. At present, no definitive cause and effect relationships have been established.

The only nationwide on-farm study completed to date focused solely on preweaned dairy heifers (National Dairy Heifer Evaluation Project, NDHEP). Other studies, primarily in Washington state, have looked at adult dairy and beef cattle, as well as dairy calves. All studies found relatively low percentages of cattle shedding O157 (animal prevalence), generally less than 1.0 percent. In the one study which has looked at beef premises, the prevalence of shedding among adult beef cattle was slightly higher than has been found among adult dairy cattle. In all studies, dairy heifers and calves generally had a higher prevalence of O157 shedding than did adult dairy cattle.

The prevalence of herds with O157 (herd prevalence) has generally been higher than the overall animal prevalence of O157. To date, in studies of premises not associated with O157 tracebacks, 27 (2.4%) of 1,139 dairies and 4 (16.0%) of 25 beef premises have been culture-positive for O157. However, research suggests that the greater the number of animals sampled on a premises, the greater the likelihood of finding that premises positive for O157. Because there have not yet been many studies that sampled more than a few animals per premises, it is probable that true herd prevalence in the U.S. is much higher than has been found to date. In addition, the NDHEP found no geographic patterns or regional differences in herd prevalence or overall animal prevalence.

Most of the initial prevalence studies have been based on one-time fecal sampling. Consequently, little is known concerning the carrier status of individual animals. Preliminary evidence does, however, suggest that cattle transiently or sporadically shed O157 in their feces and that the excretion period ranges from hours to weeks. These observations are important in that on-farm sampling of individual animals may not be an accurate reflection of the shedding status of animals entering the slaughter facility.

Evaluation of seasonal patterns in the detection of O157-positive animals is inconclusive. One Washington state study found the isolation rate of O157 to be highest during the summer months, reaching a peak in September with 13 positives per 1,000 samples. This seasonal pattern was observed in both years of the study. These results are of interest in light of the seasonal pattern evident in human O157-associated illness. In contrast, no seasonal pattern could be established from the NDHEP, which had a much larger sample size and in which roughly equal numbers of dairy calves were sampled during each calendar month. The conflicting results may be attributable to differences in age, since the Washington study included adult cattle whereas the NDHEP did not. No evidence was found of significant O157 transmission between dairy cattle in the NDHEP. Positive and negative herds were compared with respect to calf contact with older cattle and time spent by calves in maternity pens. Prevalence of O157 among preweaned dairy calves having contact with older heifers was similar to that of calves having no contact. No significant difference in herd prevalence was identified between herds that did and those that did not permit contact among calves and older animals. The length of time calves remained in the maternity area was likewise not shown to affect the prevalence of O157.

Various management and feeding practices are being examined for possible links to the presence of O157. Several practices have been found to have either a positive or negative association with the presence of O157 (Table 1). Whether or not these associations are relevant to the colonization of cattle with O157, or if cattle are even truly colonized by O157, is not yet known.

Feeding subtherapeutic levels of antibiotics to cattle to improve feed conversion and rates of weight gain is a management practice that has raised concerns. No evidence exists to suggest that O157 has acquired resistance to antibiotics. In fact, the opposite is true; most O157 organisms are susceptible to a variety of antibiotics. In addition, the use of antibiotics in cattle feed has been reduced since 1985. Current estimates indicate that only about 10 percent of all feed produced for beef cattle in the U.S. is formulated to contain antibiotics.

There is speculation that the use of ionophores, a class of antibiotics which is currently fed to certain types of cattle, may have allowed or enhanced the ability of O157 to become established as part of the intestinal microflora of cattle. The approval and subsequent adoption of ionophores for feedlot diets of cattle in the mid- to late-1970's roughly coincides with the identification of O157 as a foodborne human pathogen. Ionophore products are currently reported to be used in the diets of more than 90 percent of feedlot and farm-fed cattle and in less than 50 percent of replacement heifers and beef and dairy calves. Ionophores have been shown to inhibit gram-positive organisms in the rumen and, therefore, may allow the increased proliferation of gram-negative organisms such as *E. coli*. One study has reported that dairy farms feeding ionophores in grain had a higher O157 prevalence in calves than did farms not feeding ionophores. However, a follow-up study found no such association.

Other management practices can result in increased levels of stress in cattle. Weaning, abrupt changes in dietary composition, fasting, shipping, disease, or changes in immunologic status can predispose animals to shifts in the normal microflora of the gastrointestinal tract. It has been suggested that these shifts may result in increased numbers and/or increased shedding of O157 in cattle.

Dietary stress may be an especially important factor. The first notable dietary stress in an animal's life is weaning. One study of dairy calves revealed that the prevalence of O157 in postweaned calves was three times higher than among preweaned calves. Studies in nonbovine species have shown increased numbers of *E. coli* organisms in the intestinal tract post-weaning. *E. coli* numbers have also been shown to increase in the gastrointestinal tracts of adult animals and birds following starvation or abrupt dietary changes. Cattle are usually held off feed in the hours prior to slaughter.

Transportation provides another source of stress for livestock and may be a critical factor prior to slaughter. There is some indication, based on a recent survey of packers, that transport distances to slaughter are greater for cows and bulls than they are for fed steers and heifers and have increased over the past 10 years. Whether greater transport distance leads to increased stress is not known, but greater time in transport and holding has been shown to increase rates of infection of cattle with organisms such as *Salmonella*.

Although there has been speculation that mastitic cows may be a primary source of O157 contamination, no evidence exists to single out this particular subgroup of the cattle population. No O157 was identified in more than 500 cases of coliform mastitis in 2 separate 1993 studies conducted in California and Pennsylvania. In addition, patterns in the recorded cases of clinical mastitis identified at slaughter do not correspond to trends in outbreaks and sporadic cases of human O157-associated illness. Neither the number nor the rate of mastitic cows at slaughter increased between 1983 and 1992.

Similarly, no evidence has been presented which argues for focusing on nonambulatory cows (downer cows) as a major source of O157. The hypothesis that possible increased antibiotic usage in nonambulatory cattle could help to select for O157 or allow O157 to more readily colonize such animals does not seem highly plausible since O157 is itself susceptible to most antibiotics. However, increased stress as a result of the downer condition may increase the likelihood of shedding O157 if it were present. A current Food Safety and Inspection Service study of nonambulatory cattle should help define any relationship between O157 and such animals.

Do production and consumption patterns for ground beef offer any additional insight into *E. coli* O157:H7?

Contaminated ground beef has been the most frequently identified vehicle for O157 in human disease outbreaks. The introduction of O157 may occur at any point along the entire production to consumption continuum. Therefore, changes in the continuum over the past decade need to be identified and examined for potential impacts on ground beef contamination or increased human exposure to O157-contaminated ground beef.

Relative proportions of different types of cattle slaughtered in the U.S. have changed little since 1980. Steers and heifers accounted for approximately 80 percent of cattle slaughtered, cows 18 percent, and bulls 2 percent. Calf slaughter was minimal when compared with cattle slaughter and meat from calves generally does not go into ground beef. Production for all types of cattle continued to concentrate geographically into fewer and larger herds, particularly in dairy and cattle feeding operations.

Marketing of all types of cattle for slaughter has changed somewhat over the same time period. Currently on a national basis, greater percentages of cattle are being sold directly to packing establishments rather than being marketed indirectly through public markets. In 1980, 88 percent of steers and heifers and 35 percent of cows were sold directly, but by 1990 those figures were 94 and 40 percent, respectively.

Slaughter facilities have become larger and more concentrated geographically, particularly in the Great Plains region. In 1992, 90 percent of all fed steers and heifers were slaughtered in only 33 plants, as compared to 90 plants in 1983. In 1992, 90 percent of cows were slaughtered in 68 plants, down from 152 plants in 1983.

Once cattle have been slaughtered, ground beef production flows through a variety of processing and distribution channels (Figure 1). Ground beef is produced directly in some slaughter plants from varying combinations of cuts and trimmings produced in-house, purchased trimmings, and domestic and imported boneless manufacturing-grade beef (BMB). Ground beef is also produced by grinders and retailers who purchase carcasses, boxed beef, bulk trimmings, and/or coarse ground trimmings from slaughter plants, other grinders, and/or distributors. There are currently 2,965 grinders in the U.S., of which less than 900 slaughter cattle. In 1992, there were 30,700 supermarkets with in-house meat departments.

The sale of fed beef by packers in the form of boxed beef rather than carcasses has steadily increased over the past 20 years and has had an impact on the production and distribution of ground beef. Boxed beef is sold as vacuum-packaged primal and subprimal cuts from which much of the bone and excess fat has been removed. This has meant that more trimmings from fed cattle are produced centrally at the slaughter plant rather than locally at the grinder or retail level.

The percentages of ground beef derived from individual types of cattle can be estimated as a national average for a given time period. In 1980, steers and heifers accounted for 56 percent of domestic raw product going into ground beef, cows for 36 percent, and bulls for 8 percent. By 1992, these percentages had changed only slightly to 58 percent steers and heifers, 34 percent cows, and 8 percent bulls. Boneless manufacturing beef imports also remained stable over the last decade, comprising approximately 15 percent of the total U.S. ground beef supply.

Although the proportion of cattle types slaughtered varies regionally, ground beef formulation does not. The formulation of ground beef is based largely on fat content. Lean meat from cows and bulls and lean and fat trimmings from fed steers and heifers can be shipped to various locations and then mixed to produce the final ground beef product.

The composition of ground beef in terms of the sources of raw product (lean and fat) appears to be independent of the production and distribution channel through which it passes. Any given pound or patty of ground beef can contain any combination of domestic cow meat, domestic fed beef, and/or imported BMB, regardless of the channel through which it was produced.

Per capita ground beef consumption (net disappearance) has increased since 1980 but is still below mid-1970 levels. Both the proportion of people that consumed ground beef in the form of hamburgers and the amount consumed increased in most age groups, including those at highest risk for O157-related illness, young children and the elderly. There was a corresponding increase in food expenditures outside of the home during the same time period. In 1992, fast food hamburgers accounted for about 47 percent of fast food sales, or 15 percent of all hotel, restaurant, and institution (HRI) sales.

Ground beef consumed in HRI settings, especially fast food establishments, is purchased primarily from grinders in the form of patties. Retail sources of ground beef are more evenly distributed among cow packers, fed beef packers, grinders, and trimmings produced in-house. This information along with the apparent increased consumption of hamburgers in HRI settings appears to indicate that a greater proportion of ground beef is now flowing through the channel from grinders to HRI's than during the early 1980's.

Future directions

- Would a geographic pattern in the number of O157 cases in humans tell something about O157 prevalence in cattle?

It is unlikely that any geographic pattern of human disease would reflect a geographic variation in the source of the O157-contaminated ground beef. In many cases the location of consumption of ground beef is not related to the original location of the sources of that ground beef nor to the potential sources of O157 contamination. Cattle that go into ground beef production may be moved great distances in the hours prior to slaughter, lean and fat trimmings may be shipped some distance prior to final grinding and mixing, and the final product may in turn be widely distributed.

- How can we explain the seasonality of human cases and outbreaks associated with ground beef?

The seasonality of cases and outbreaks associated with ground beef might be a reflection of any one or a combination of factors. First, there may be greater shedding of O157 by cattle during warmer months of the year, which may lead to increased contamination of ground beef during these months. Second, consumption of ground beef is higher during warmer months (summer barbecues, picnics, etc.). Third, there may be a greater likelihood of temperature abuse and/or less thorough cooking of ground beef during these months.

- Is there a particular channel in the ground beef production continuum that is associated with an increased risk of O157 contamination?

Ground beef intended for both retail and HRI can pass through various channels which may include a number of different steps. Although additional handling creates more opportunities for cross-contamination, no one channel can be singled out at this time as posing a greater risk.

- Should the goal be to eradicate O157 on the farm?

It does not currently appear feasible to target on-farm eradication of O157 for the following reasons: the lack of knowledge about the ecology of O157, the widespread geographic distribution of the organism, the fact that O157 has been found in both beef and dairy cattle, and the difficulty of identifying infected animals because of the likelihood of sporadic shedding and the absence of clinical disease. Since the risk of O157 illness cannot be eliminated at this time, it must be managed.

- How can the risk of O157 illness best be managed?

A general approach to manage the risk of O157 illness attributable to ground beef is: (1) to reduce the level of O157 on the farm, and (2) to better understand different channels of the ground beef production system and use this knowledge to identify critical points at which intervention would be most effective. To gain a better understanding of the system, specific questions that need to be addressed include: (a) how does the number of steps involved in the production of ground beef affect the risk of contamination?, (b) how does the risk change as ground beef moves through the system?, and (c) what is the volume of ground beef that flows through the various channels? If it is possible to identify one or two points along the continuum that can be associated with an increased risk of O157 contamination, then research can be focused on those specific channels.

- Where should attention be focused?

Attention should be focused on what occurs just prior to slaughter. Because shedding of O157 may be sporadic, cattle that test O157-negative on the farm may test positive just prior to slaughter. This is especially plausible in light of the many stress factors to which cattle are subjected between leaving the farm or feedlot and slaughter. Although it is not known if cattle that are not shedding O157 at the time of slaughter can be a source for ground beef contamination, animals which are shedding can be a factor in such contamination. Thus, individual cattle should be followed and sampled at various points after leaving the farm. Sampling at the auction barn, feedlot, after unloading at the slaughter plant, and immediately before slaughter may provide valuable information about shedding patterns. The cleanliness of animals entering the slaughter facility is also an important consideration. Contamination of the hide and haircoat with mud and feces may provide O157 with an additional mode of entry into the slaughter facility via either culture-positive or culture-negative animals.

- What other types of preharvest research should be recommended?

Research should concentrate on the ecology of O157 in the gastrointestinal tract of ruminants, specifically to assess the effects of stressors such as dietary changes and movement of animals. The ecology of O157 in the farm environment also needs further research. Since previous studies of management factors, such as the use of ionophores, have not been definitive, further work is needed to address the effects of management factors on the prevalence of O157. Competitive exclusion, the administration of protective intestinal microorganisms known as probiotics, should also be evaluated as an intervention strategy. Probiotics can protect poultry from colonization by human enteropathogens, including O157. Results of studies on the use of probiotics in cattle have been variable. None of the currently available probiotic feed supplements for cattle marketed in the U.S. has met the regulatory requirements for demonstration of prophylactic or therapeutic claims.

- What about postharvest research?

Emphasis should be placed on identifying and monitoring where and how contamination occurs. The Hazard Analysis and Critical Control Point (HACCP) system should continue to be developed and implemented as a preventative food safety assurance system. HACCP principles should be applied not only at slaughter and grinding facilities but also at other points along the continuum including shipment between locations and storage. The intent would be to ensure that a product leaving a certain phase of production or location is as safe or safer than when it entered.

- What about tracebacks?

Tracebacks have been proposed as an important component of a food safety agenda. In the case of O157, tracebacks could provide valuable information about on-farm factors and production processes associated with the organism, as well as about the ecology of O157. However, from an immediate disease prevention perspective, tracebacks would currently be of uncertain value. Not enough is known about the ecology of O157 in cattle to implement prudent, on-farm measures to prevent future contamination. Tracebacks involving ground beef would be especially difficult to carry out to the farm level with a high degree of precision. Even given a highly dependable system of individual animal identification, the complexity of production and distribution channels for ground beef tends to make the determination of individual animal contributions to any given pound of product a difficult process.

Table 1. Association of *E. coli* O157:H7 with Selected Management Practices*

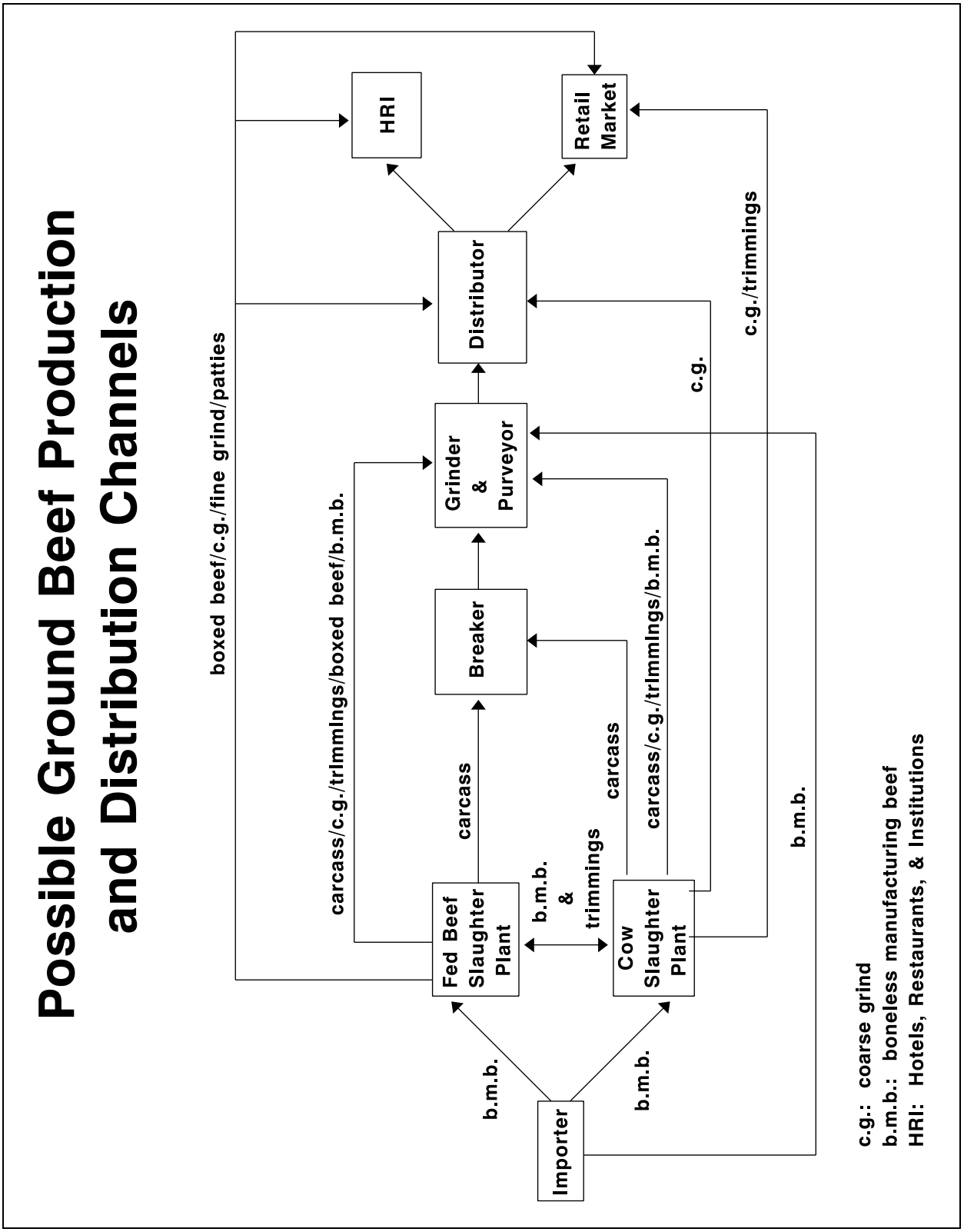
Management Practice	Subgroup	Association with O157
Small herd size[a,b]	Dairy farms	pos, none
Use of computerized feeders[a]	Dairy farms	pos
Irrigation of pastures with manure slurry[a]	Dairy farms	pos
Feeding of whole cottonseed[a,c]	Dairy heifers and cows	neg, neg
Feeding of milk replacer[b,c]	Dairy calves	neg, none
Feeding of ionophores[b,c]	Dairy calves	pos, none
Grouping of calves prior to weaning[b,c]	Dairy calves	none, pos
Sharing of unwashed feeding utensils among calves[c]	Dairy calves	pos
Feeding of oats in starter ration[c]	Dairy calves	pos
Feeding of grain during first week of life[b,c]	Dairy calves	none, pos
Feeding of clover as first forage[c]	Dairy calves	neg

* Many other management factors have been tested for association with O157; only those listed were found to have statistical significance at $p \leq 0.10$.

pos = positive association, i.e., management practice is associated with increased O157 prevalence
neg = negative association, i.e., management practice is associated with decreased O157 prevalence
none = no association

[a] Hancock et al., 1994
[b] Hancock et al., 1993b
[c] Garber et al., 1994

Figure 1


Possible Ground Beef Production and Distribution Channels
HRI
Retail Market
Distributor
Grinder & Purveyor
Breaker
Fed Beef Slaughter Plant
Cow Slaughter Plant
Importer
boxed beef/c.g./fine grind/patties
carcass/c.g./trimmings/boxed beef/b.m.b.
carcass
b.m.b. & trimmings
carcass
carcass/c.g./trimmings/b.m.b.
c.g.
c.g./trimmings
b.m.b.
b.m.b.
b.m.b.
c.g.: coarse grind
b.m.b.: boneless manufacturing beef
HRI: Hotels, Restaurants, & Institutions