

Public Health Significance of Verotoxin-Producing *Escherichia coli* O157:H7

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Abstract

Escherichia coli O157:H7 is an emerging bacterial zoonotic pathogen, which causes significant morbidity as well as mortality in developing and developed nations of the world. Specific *E. coli* strains, which are pathogens to humans include Enterotoxigenic, Enteropathogenic (EPEC), Diffusely Adherent (DAEC), Enteroaggregative (EAEC), Enteroinvasive and Verotoxigenic (VTEC are also known as Shigatoxigenic, STEC or Enterohemorrhagic *E. coli*). Recently, verotoxin expressing EAEC have been identified as a significant public health concern. They cause a wide range of illnesses, from mild diarrhea to haemorrhagic colitis with severe abdominal pain and bloody diarrhea. Illness is usually self-limiting and resolves after about eight days. *Escherichia coli* O157:H7 is responsible for 20 % of foodborne outbreaks. Ruminants have been identified as the major reservoir of *E. coli* O157:H7, with cattle as the most important source of human infections. In addition, other ruminants such as sheep, goats, and deer also harbor these bacteria. Birds are thought to be likely transport hosts for *E. coli* O157:H7. In addition, flies and beetles, including houseflies and filth flies of several species and dung beetles may also act as possible transport hosts. Several routes for human infection with *E. coli* O157:H7 are direct contact, person-to-person spread, contaminated food, and contaminated water. A number of foods, after meat, milk, cheese, juices, salads, vegetables and fruits, etc. can serve as vehicle for *E. coli* O157:H7. Additional work on risk factors, fecal carriage of organism in other food animals, agent host interaction and simple and low-cost diagnostic kit should be undertaken.

Keywords: Diarrhea; Emerging Pathogen; *Escherichia coli* O157:H7; Public Health; Reservoirs; Transmission

Introduction

Escherichia coli O157:H7 is considered one of the most serious of known foodborne pathogens of worldwide significance. It can cause life threatening infections, particularly in children, elderly and immunocompromised patients [1]. The infections by *Escherichia coli* O157:H7 have been reported of increasing frequency from all parts of the world in the form of food poisoning outbreaks. Because of the severity of these illnesses and the apparent low infective dose (< 10 cells), *Escherichia coli* O157:H7 is considered one of the most serious of known foodborne pathogens [2].

Specific *E. coli* strains may be pathogens, which are divided into intestinal pathogens causing diarrhea and extra intestinal *E. coli* causing a variety of infections in both humans and animals including urinary tract infections (UTI), meningitis and septicemia. There are six recognized enteric pathotypes of *E. coli*, Enterotoxigenic, Enteropathogenic (EPEC), Diffusely Adherent (DAEC), Enteroaggregative (EAEC), Enteroinvasive and Verotoxigenic (VTEC are also known as Shigatoxigenic, STEC or Enterohemorrhagic *E. coli*). VTEC are distinguished from other *E. coli* pathotypes by the possession of the genes for one or more verotoxins [3]. In addition, some VTEC strains carry a virulence factor common to the EPEC pathotype, the locus of enterocyte effacement (LEE) [3]. It is mentioned that verotoxin expressing EAEC are recognized as an important public health issue [4].

Hemolytic uremic syndrome (HUS) is the most worrisome complication of Enterohemorrhagic *Escherichia coli* (EHEC) infections and is characterized by the triad of acute renal failure, microangiopathic, hemolytic anemia, and thrombocytopenia, with a fatality rate between 2% and 7% [5].

Verotoxigenic *E. coli* (VTEC) are so-called because of their ability to produce one or both of two verotoxins (VT1 and VT2). They cause a wide range of illnesses, from mild diarrhoea to haemorrhagic colitis with severe abdominal pain and bloody diarrhoea. Illness is usually self-limiting and resolves after about eight days [6].

It is well known that some species of *Escherichia coli* produce verotoxin (shiga toxin) and those that do are mainly hosted by cattle and sheep, and the gastrointestinal system flora of some domestic animals. Despite the fact that *Escherichia coli* as a commensal organism, bacteria can be found in intestinal microflora of a variety of animals including man, not all the strains are harmless, and some can cause debilitating and sometimes fatal diseases in humans as well as mammals and birds [7]. The present communication is an attempt to delineate to the public health significance of verotoxin producing *Escherichia coli* O157: H7.

Public health importance

E. coli O157:H7 has been isolated from ill people around the world. It tends to be reported more often from more developed countries but this may be an artifact caused by the paucity of sophisticated diagnostic laboratories in developing countries. Food Net data indicate that *E. coli* O157:H7 causes significantly more cases of sporadic infections than cases linked to an outbreak [8]. For example, in 2004, only 9% of 402 confirmed cases of infection with *E. coli* O157:H7 were associated with outbreaks. Sporadic infections appear to be associated with some of the same factors that cause outbreaks: undercooked hamburgers and exposure to farms and cattle. Some sporadic infections are also associated with use of immunosuppressive medications and dining at table service restaurants [9].

Vehicles of infection, suspected or confirmed, have been identified for most outbreaks. During the 1980s most outbreaks of *E. coli* O157:H7 were associated with inadequately cooked hamburgers and unpasteurized milk [10,11]. Some later outbreaks have been traced to other dairy products such as cheese and yogurt [12]. Increasingly, contaminated water has been reported as a source of human infection. This includes drinking water sources contaminated with animal feces and also contaminated lake and pool water used for swimming and playing [13].

A large outbreak in Japan, affecting more than 12,000 persons, was associated with contaminated radish sprouts [14], and other outbreaks have been associated with contaminated fruit juices, melon, and salad greens [15]. More recently a number of outbreaks have occurred among children visiting farms and petting zoos where they come into direct contact with animals carrying *E. coli* O157:H7 and their environment [16].

Outbreaks at country fairs may also result from airborne dispersion of bacteria in buildings used to show animals [17,18]. Finally, direct person-to person infection occurs particularly among children and their caregivers, such as in day care facilities and also within families [19].

Despite more than 100 *E. coli* serotypes identified as responsible for verotoxin production in cattle, they usually do not lead to disease in animals; however, they have been an important cause of foodborne illness worldwide [20].

Reservoirs of *E. coli* O157:H7

Understanding the epidemiology of this organism requires knowledge of where these bacteria live and grow in nature (their reservoir) and of how humans come into contacts them. Ruminants have been identified as the major reservoir of *E. coli* O157:H7, with cattle as the most important source of human infections. Other ruminants known to harbor these bacteria include sheep, goats, and deer. Shigatoxi-genic (STEC) bacteria are occasionally isolated from other animals but it is believed that the bacteria are present as transients and that

the animals acquired these bacteria from meat, foods, or water contaminated by fecal material from ruminants [10]. STEC bacteria usually do not cause illness in animals with a few exceptions such as diarrhea in calves [21].

Transport Hosts

Birds are thought to be possible transport hosts for *E. coli* O157:H7. Some wild birds harbor these bacteria, and pigeons, for example, might spread these bacteria around a farm environment. *E. coli* O157:H7 has been isolated from gulls [22], a rook (relative of crows) [23], and pigeons [24].

Flies and beetles, including houseflies and filth flies of several species [25,26] and dung beetles [27], collected on farms with animals shedding *E. coli* O157:H7, contain detectable levels of these bacteria. These insects frequent fecal deposits and may be able to transfer these bacteria to foods, feed and water. In experiments with houseflies, *E. coli* O157:H7 survived and replicated in the mouthparts and crop of the flies for up to 4 days [28].

Fruit flies collected from a compost pile of decaying apples and peaches contaminated with *E. coli* contained these bacteria both internally and externally and were able to transfer them to wounds in uncontaminated apples. *E. coli* O157:H7 can grow rapidly in apple wounds. Fruit flies could contribute to widespread contamination of wounded apples that may be processed into cider [29].

Slugs are known vegetarian pests that frequently traverse leafy vegetables and may be present on these foods when harvested. Slugs ingest bacteria from the environment and also accumulate bacteria in the mucus surrounding their bodies. Some common gray field slugs collected on a farm in Scotland were found to carry the same pathogenic strain of *E. coli* as detected in feces from sheep grazing there. Slugs may travel 12m or more per night so there is a potential for slugs to carry *E. coli* O157:H7 from manure to vegetables [30].

Routes of Human Infection

Various routes for human infection with *E. coli* O157:H7 were reviewed in a recent article on the epidemiology of outbreaks of this bacterium in the U.S. (1982 - 2002) [31]. Studies in Canada and France demonstrated that the incidence of Hemolytic uremic syndrome (HUS) and Verotoxigenic or verotoxin-producing *E. coli* (VTEC) infection in humans is correlated with indicators of cattle density [32,33]. *E. coli* O157:H7 in ruminant feces may be directly ingested by persons interacting or working with animals. Fecal material may contaminate meat during slaughter, may enter lakes or drinking water sources by action of rain or wind, and may be deposited on fruits and vegetables inadvertently or by use of manure for fertilization. In addition, some animals may transport these bacteria from a fecal source to drinking water or foods [34].

Direct contact: *Escherichia coli* O157:H7 shed by infected animals may be spread to many surfaces in enclosures where ruminants are kept including the hides of other animals [35]. Depending on moisture and humidity, these bacteria may persist on gates, stiles and other farm surfaces for more than four weeks [36]. *E. coli* O157:H7 survives in cattle feces for up to 18 weeks at 15°C. This helps explain why a substantial number of people residing on dairy farms have evidence of current (stool cultures) or past (serologic status) infection with

Person-to-person spread of *E. coli* O157:H7 has been the primary mode of infection in many outbreaks in day cares, schools and hospitals, particularly where there have been lapses in hygiene [34]. In many other outbreaks, some of the cases who consumed contaminated food or water passed the infection directly to others. Although a majority of children infected with *E. coli* O157:H7 shed these bacteria in their feces for only a few days, in more seriously ill children, cells of *E. coli* O157:H7 may be shed for 20 - 30 days or longer. VTEC bacteria may be present in stool samples even after children become asymptomatic [38].

Contaminated food: Beef, lamb, and mutton can be contaminated during slaughter and processing by exposure to feces or hides containing *E. coli* O157:H7. In a recent study in the Midwest, more than 45% of over 330 carcasses tested during July–August contained detectable levels of *E. coli* O157:H7 [39]. Milk from dairy cows, sheep, and goats may be contaminated with *E. coli* and other bacteria from the environment. Proper pasteurization will kill these bacteria. Outbreaks of *E. coli* O157:H7 due to contaminated dairy products are usually as-

sociated with unpasteurized milk but there have been some cases of post-pasteurization contamination. Foods can also be contaminated with *E. coli* O157:H7 by cross-contamination during food preparation and by infected workers who do not practice good hygiene. Several restaurant outbreaks in Oregon and Washington in 1993 were associated with a variety of items from the salad bar but not with steak. All the restaurants obtained their beef from the same source, and it was the practice to trim, macerate, and marinate the beef in the same kitchens used for preparation of fruits and vegetables for the salad bar. It appeared that the beef itself was cooked well enough to destroy *E. coli* O157:H7 but that some raw beef was the source of contamination [40].

Contaminated water: Water used for drinking or recreation has been reported as the vehicle of infection for 49 outbreaks: 6 outbreaks associated with water parks and pools, 18 with lakes, springs, canals, and streams, 10 with well water, 12 with “drinking water,” and 3 with tap water. Fecal material from ruminant animals, domestic and/or wild, is the probable source of *E. coli* O157:H7 in lakes, streams, and wells and for some “drinking water” outbreaks. Drinking water from unchlorinated source was implicated in a large outbreak [41].

Conclusions

Escherichia coli O157: H7, is a rod shaped, motile, non-spore forming, Gram negative bacterium, which has emerged as an important zoonotic pathogen of global importance. The organism is common commensal inhabitant of the gastrointestinal tract of mammals and birds. It is an adaptable organism that can persist for extended periods outside of a host in water, soil or on plants. Fecal materials may contaminate meat during slaughter, may enter lakes or drinking water sources by action of rain or wind, and may be deposited on fruits and vegetables inadvertently or by use of manure for fertilization. In addition, some animals may transport these bacteria from a fecal source to drinking water or foods. It is emphasized that all food processing enterprises should adopt good hygienic practice and hazard analysis and critical control in order to provide safe food to the consumers. This will certainly mitigate the food poisoning caused by various pathogens, including *E. coli* O157:H7.

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Bibliography

1. Pal M and Mahendra R. “*Escherichia coli* O157: H7: An emerging bacterial zoonotic foodborne pathogen of global significance”. *International Journal of Interdisciplinary and Multidisciplinary Studies* 4.1 (2016): 1-4.
2. Kiranmayi B and Krishnaiah N. “Detection of *Escherichia coli* O157: H7 prevalence in foods of animal origin by cultural methods and PCR technique”. *Veterinary World* 3.1 (2010): 13-16.
3. Kaper JB, *et al.* “Pathogenic *Escherichia coli*”. *Nature Reviews Microbiology* 2.2 (2004): 123-140.
4. Beutin L and Martin A. “Outbreak of Shiga toxin-producing *Escherichia coli* (STEC) O104: H4 infection in Germany causes a paradigm shift with regard to human pathogenicity of STEC strains”. *Journal of Food Protection* 75.2 (2012): 408-418.
5. Erdogan H, *et al.* “Enterohemorrhagic *Escherichia coli* O157: H7: case report”. *Turkish Journal of Pediatrics* 50.5 (2008): 488-491.
6. Garvey P and McKeown P. “Epidemiology of verotoxigenic *E. coli* O157 in Ireland”. National Disease Surveillance Centre (2003): 25-27.
7. Belanger L, *et al.* “*Escherichia coli* from animal reservoirs as potential source of human extraintestinal pathogenic *E. coli*”. *FEMS Immunology and Medical Microbiology* 62.1 (2011): 1-10.
8. Centers for Disease Control. Food Net Surveillance Report for 2004 (Final Report) (2006): 203.

9. Kassenborg HD, *et al.* "Farm visits and undercooked hamburgers as major risk factors for sporadic *Escherichia coli* O157: H7 infection: Data from a case-control study in 5 FoodNet sites". *Clinical Infectious Diseases* 38.3 (2004): 271-278.
10. Caprioli A, *et al.* "Enterohaemorrhagic *Escherichia coli*: emerging issues on virulence and modes of transmission". *Veterinary Research* 36.3 (2005): 289-311.
11. Hussein HS and Bollinger LM. "Prevalence of Shiga toxin-producing *Escherichia coli* in beef". *Meat Science* 71.4 (2005): 676- 689.
12. Honish L, *et al.* "An outbreak of *E. coli* O157: H7 hemorrhagic colitis associated with unpasteurized gouda cheese". *Canadian Journal of Public Health* 96.3 (2005): 182-184.
13. Craun GF, *et al.* "Outbreaks associated with recreational water in the United States". *International Journal of Environmental Health Research* 15.4 (2005): 243-262.
14. Michino H, *et al.* "Massive outbreak of *Escherichia coli* O157: H7 infection in schoolchildren in Sakai City, Japan, associated with consumption of white radish sprouts". *American Journal of Epidemiology* 150.8 (1999): 787-796.
15. Reiss G, *et al.* "*Escherichia coli* O157: H7 infection in nursing homes: Review of literature and report of recent outbreak". *Journal of the American Geriatrics Society* 54.4 (2006): 680-684.
16. Grif K, *et al.* "Importance of environmental transmission in cases of EHEC O157 causing hemolytic uremic syndrome". *European Journal of Clinical Microbiology and Infectious Diseases* 24.4 (2005): 268-271.
17. Bender JB. "Compendium of measures to prevent disease associated with animals in public settings". *Morbidity and Mortality Weekly Report* 54 (2005): 1-13.
18. Varma JK, *et al.* "An outbreak of *Escherichia coli* O157 infection following exposure to a contaminated building". *Journal of the American Medical Association* 290.20 (2003): 2709-2712.
19. Williams LD, *et al.* "An outbreak of *Escherichia coli* O157: H7 involving long term shedding and person-to-person transmission in a child care center". *Journal of Environmental Health* 59 (1997): 9-14.
20. Aksoy A, *et al.* "Verotoxin Production in Strains of *Escherichia coli* Isolated from Cattle and Sheep, and Their Resistance to Antibiotics". *Journal of Veterinary and Animal Science* 31.4 (2007): 225-231.
21. Kang SJ, *et al.* "Occurrence and characteristics of enterohemorrhagic *Escherichia coli* O157 in calves associated with diarrhoea". *Veterinary Microbiology* 98.3-4 (2004): 323-328.
22. Wallace JS, *et al.* "Isolation of verocytotoxin-producing *Escherichia coli* O157 from wild birds". *Journal of Applied Microbiology* 82 (1997): 399-404.
23. Ejidokun OO, *et al.* "Human Vero cytotoxigenic *Escherichia coli* (VTEC)O157 infection linked to birds". *Epidemiology of Infection* 134.2 (2006): 421-423.
24. Grossmann K, *et al.* "Racing, ornamental and city pigeons carry Shiga toxin producing *Escherichia coli* (STEC) with different Shiga toxin subtypes, urging further analysis of their epidemiological role in the spread of STEC". *Berliner Und Munchener Tierarztliche Wochenschrift* 118.11-12 (2005): 456-463.

25. Alam MJ and Zurek L. "Association of *Escherichia coli* O157: H7 with houseflies on a cattle farm". *Applied Environmental Microbiology* 70.12 (2004): 7578-7580.
26. Keen JE., *et al.* "Shiga-toxigenic *Escherichia coli* O157 in agricultural fair livestock, United States". *Emerging Infectious Diseases* 12.5 (2006): 780-786.
27. Xu JG., *et al.* "Isolation of *Escherichia coli* O157: H7 from dung beetles *Catharsius molossus*". *Microbial Immunology* 47.1 (2003): 45-49.
28. Sasaki T., *et al.* "Epidemiological potential of excretion and regurgitation by *Musca domestica* (Diptera: Muscidae) in the dissemination of *Escherichia coli* O157: H7 to food". *Journal of Medical Entomology* 37.6 (2000): 945-949.
29. Janisiewicz WJ., *et al.* "Fate of *Escherichia coli* O157: H7 on fresh-cut apple tissue and its potential for transmission by fruit flies". *Applied and Environmental Microbiology* 65.1 (1999): 1-5.
30. Sproston EL., *et al.* "Slugs: Potential novel vectors of *Escherichia coli* O157". *Applied and Environmental Microbiology* 72.1 (2006): 144-149.
31. Rangel JM., *et al.* "Epidemiology of *Escherichia coli* O157: H7 outbreaks, United States, 1982-2002". *Emerging Infectious Disease* 11.4 (2005): 603-609.
32. Haus-Cheymol R., *et al.* "Association between indicators of cattle density and incidence of paediatric haemolytic uraemic syndrome (HUS) in children under 15 years of age in France between 1996 and 2001: an ecological study". *Epidemiology and Infection* 134.4 (2006): 712-718.
33. Valcour JE., *et al.* "Associations between indicators of livestock farming intensity and incidence of human shiga toxin-producing *Escherichia coli* infection". *Emerging Infectious Disease* 8.3 (2002): 252-257.
34. Pennington TH. "VTEC: lessons learned from British outbreaks". *Journal of Applied Microbiology* 88.1 (2000): 90S-98S.
35. Childs KD., *et al.* "Molecular characterization of *Escherichia coli* O157: H7 hide contamination routes: Feedlot to harvest". *Journal of Food Protection* 69.6 (2006): 1240-1247.
36. Williams AP., *et al.* "Persistence of *Escherichia coli* O157 on farm surfaces under different environmental conditions". *Journal of Applied Microbiology* 98.5 (2005): 1075-1083.
37. Wilson J., *et al.* "Verocytotoxigenic *Escherichia coli* infection in dairy farm families". *Canada Communicable Disease Report* 24.3 (1998): 17-20.
38. Karch H., *et al.* "Long-term shedding and clonal turnover of enterohemorrhagic *Escherichia coli* O157 in diarrheal diseases". *Journal of Clinical Microbiology* 33.6 (1995): 1602-1605.
39. Elder RO., *et al.* "Correlation of enterohemorrhagic *Escherichia coli* O157 prevalence in feces, hides, and carcasses of beef cattle during processing". *Proceedings of the National Academy of Sciences of the United States of America* 97.7 (2000): 2999-3003.
40. Jackson SG., *et al.* "*Escherichia coli* O157: H7 diarrhoea associated with well water and infected cattle on an Ontario farm". *Epidemiology and Infection* 120.1 (1998): 17-20.

41. Swerdlow DL, *et al.* "A waterborne outbreak in Missouri of *Escherichia coli* O157: H7 associated with bloody diarrhea and death". *Annals of Internal Medicine* 117.10 (1992): 812-819.

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