A Review on *Escherichia coli* O157:H7-The Super Pathogen

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ABSTRACT: Shiga- toxin producing *Escherichia coli* (STEC) are among most important cause of food diseases. More than 70 different serotypes of Shiga -toxin producing *E. coli* (STEC) that cause disease in humans worldwide have been described. Illnesses range from mild diarrhea to bloody diarrhea to hemorrhagic colitis (HC) and life- threatening Hemolytic Uremic Syndrome (HUS). *E. coli* O157:H7 is STEC strain most often associated with the most severe form of disease. Infections have been associated with bovine food products, direct animal contact, and bovine manure contamination of vegetables; fruits and drinking water have also been implicated. Epidemiological investigations have implicated food and water as most common vehicle for infections cause by *E. coli* O157:H7. *E. coli* O157:H7 has been isolated from surface water and can survive for many weeks in these kinds of environments. It was demonstrated that *E. coli* O157:H7 can enter the lettuce plant through the root system and migrate throughout the edible portion of the plant. There is an obvious risk of E.coli O157:H7 infection arising from contamination of fruit and vegetable crops grown in soil to which abattoir waste especially where the food products (e.g. salad vegetables) are consumed with minimal processing. *E. coli* 0157:H7 serotype is considered as being the most significant and has been associated with large food-borne outbreaks in North America, Europe, and Japan. The Centre for Disease Control estimate that *E. coli* 0157:H7 causes approximately 73,000 illnesses and 61 deaths each year in the USA. After *E. coli* was recognized as a cause of hemorrhagic colitis, the centers for disease control and prevention (CDC) reviewed over 3,000 *E.coli* strains serotyped between 1973 and 1983 and found only one O157:H7 isolate. The largest out break to date occurred in Japan in 1996, affecting over 9000 people with contaminated radish sprout as the possible source of infection. Its resistance

to commonly used antimicrobials generates a public health dilemma. The mini review aims to highlight epidemiology of *E. coli* O157:H7 and the resultant Public and environmental implications of its continuous existence in our ecosystem.

Keywords: *Escherichia coli O157: H*, Hemolytic Uremic Syndrome, food borne disease

Introduction

The genus *Escherichia coli* comprised Gram-negative, facultative anaerobic bacilli, common inhabitant of the gastrointestinal tract of mammals, and belong to the Enterobacteriaceae family. They are bile-tolerant, non fastidious organisms that are easily cultured on routine laboratory media. They ferment lactose and grow best under mesophilic temperatures with an optimum at 37^0C. Most *E. coli* have the b-glucoronidase enzyme that breaks down complex carbohydrates. This enzyme is used in a fluorogenic assay that takes advantage of the breakdown of 4-methyl umbeliferone glucoronide (MUG) byb-glucoronidase yielding a fluorescent compound. However, *E. coli* O157:H7 does not have b-glucoronidase. Further, *E. coli* O157:H7 cannot ferment sorbitol within 24 h, while 90% of *E.coli* can.

Enterohemorrhagic *Escherichia coli* (EHEC) are the cause of serious illness and mortality in outbreaks of food borne illness linked variety of foods. There are a number of different enteropathogenic groups of *E.coli* that have been shown to cause various types of gastrointestinal infections. Six main pathotypes of *E.coli* can be distinguished: enteropathogenic *E.coli* (EPEC), enterotoxigenic *E.coli* (ETEC), enteroinvasive *E.coli* (EIEC), diffusely adhering *E.coli* (DAEC), enteroaggregative *E.coli* (EAEC), and EHEC. All these pathotypes of *E.coli* use multistep systems of pathogenesis, comprised in general of colonization of the mucosal site, evasion of the host defenses, and multiplication and host damage (Kaper, 2005). After the first outbreak in 1982, *E.coli* O157:H7 has become the most widely known EHEC strain. Reports have shown that an *E. coli* O157:H7 strain that was involved in an outbreak of Hemorrhagic Colitis in the United States produced Shiga toxins. Shiga Toxin *E.coli* (STEC) was epidemiologically associated with Hemolytic Uremic Syndrome (Bell, 2002). Generally, *E.coli* can be a member of the normal microflora in animals including humans. However, virulence genes acquired through various ways in the ecosystem enabled *E. coli* to acquired different forms genes that lead to increased

pathogenicity. In general, many pathogenic strains behave biochemically and ecologically like any other nonpathogenic *E.coli*, making their detection among commensal *E.coli* an important problem, especially among EHEC (Bettelheim, 2007). Serotype O157 has been found to be incapable to ferment the carbohydrate Sorbitol (Riley et al., 1983). Compared to other pathogenic *E.coli*, this serotype would cause hemorrhagic colitis (HC) and other severe symptoms. Other serotypes, such as O26, O111, and sorbitol-fermenting O157:NM, have also been related with HC and subsequently classified as EHEC (Armstrong *et al*., 1996). The ability to produce Shiga toxins is the common characteristics of all EHEC that are often referred to as Shiga toxin-producing *E.coli* (STEC).

Pathogenesis and Virulence factors

The production of Shiga toxin is fundermental to the pathogenesis of bloody diarrhoea and haemolytic uraemic syndrome. *E coli* O157 strains have the locus of enterocyte effacement genes but other serogroup strains without these genes have also caused haemolytic uraemic syndrome). The pathogenicity of STEC is determined by several virulence factors that are encoded by chromosomal pathogenicity islands, phage chromosomes integrated in the bacterial genome as well as plasmids. Shiga toxins are members of a toxin family that share many common features. The Shiga toxins identified in EHEC are classified in two distinct subgroups: Stx1 and Stx2. The toxins are produced by the pathogen in the colon and cause local damage. The ability to pass through the bloodstream to the kidney plays a role in causing HC and HUS. *E coli* O157 can produce two different Shiga toxins encoded by bacteriophage. Stx1 is very similar to the type 1 toxin of *Shigella dysenteriae*; Stx2 is genetically and immunologically distinct with 55–60% similarity in genetic and aminoacid sequences. The possession and expression of the Stx2 gene and the variant Stx2c (which often occurs with Stx2) correlate strongly with the causation of bloody diarrhoea and haemolytic uraemic syndrome (Persson *et al*, 2007,Newton *et al*, 2009).

Shiga toxins bind to glycosphingolipid globotriaosylceramide (Gb3), a cell surface receptor. They are then internalised by clathrin-dependent endocytosis, and go on to specifically depurinate 28S eukaryotic rRNA, inhibiting protein synthesis. This step induces a ribotoxic stress response that can lead to cytokine release and apoptotic cell death. In the human kidney, Gb3 is present on glomerular endothelial cells, podocytes, and various tubular epithelial cell

types. Shiga toxin binds to these cells in renal sections from patients with haemolytic uraemic syndrome, and damage markers from these cells can be detected in their urine; biopsy samples from these patients show apoptosis of glomerular and tubular cell types and fibrin-rich glomerular microangiopathy (Tarr *et al.,* 2005). Blood from patients with haemolytic uraemic syndrome showed an increase in microparticles with surface-bound tissue factor and in functional tissue factor. Tissue factor can contribute to a pro thrombotic state (Stahl *et al*, 2009).

Other structures that help EHEC in adhering to host cells are fimbriae and fimbrial adhesins, thread-like structures that extend out from the bacterial surface. Type 1 fimbriae are the first adhesins described in *E.coli* and are the most common adhesins produced. These adhesins mediate the adherence of the pathogen to mannose-containing glycoproteins found on the surfaces of eukaryotic cells. EHEC clinical isolates from HUS patients have been found to have a distinct virulence profile. Strains capable of producing both Shiga toxins have been found to be highly associated with bloody diarrhea or HUS, while strains with only Stx1 are rarely found in HUS patients. In addition, clinical strains associated with HUS have also been found to be more enterohemolytic and are more likely to possess intimin (Law, 2000). The high virulence of STEC strains like *E.coli* O157:H7 is not only dependent on virulence factors but partially also on the ability to survive environmental unfavourable conditions, such as resistance to low pH levels found in the gastrointestinal tract which contributes to very low infectious dose of 50–100 cells or lower (Armstrong *et al*., 1996).

Prevalence and Epidemiology

STEC and specifically *E. coli* O157:H7 are considered as emerging food borne pathogens that occur globally. In the United States, E. coli O157: H7 is estimated to cause 73,480 illnesses annually, with approximately 2168 hospitalizations and 61 deaths (Mead et al., 1999). In Europe, 14,000 cases in over 24countries have occurred from 2000 to 2005, of which 62% belong to the O157 serogroup (Fisher and Meakins, 2006). A review of 90 outbreaks in Britain, Ireland, Scandinavia, Canada, USA and Japan indicated that about 20% of outbreak cases resulted from secondary spread (Snedeker *et al*., 2009). However, the duration of outbreaks shows that continued transmission thereafter in the affected communities is very rare (Nataro and Kaper, 1998).

The primary habitat of *E.coli* is the intestinal tract of warm-blooded animals as well as humans. *E.coli* infections in humans are transmitted directly from animals mainly through contaminated foods. Enteric pathogens are distributed from livestock to food crops and can occur in various ways such as application of manures, irrigation with contaminated water, dispersal by air, and dispersal via biological vectors, such as wildlife and insects (Janisiewicz *et al*., 1999). Many studies have measured the prevalence of *E coli* O157 in cattle. Comparisons of reported data have shown big differences between studies. For dairy cattle, the prevalence estimated by testing faeces ranged from 0.2% to 48.8%. In the USA (prevalence in calves 0.4–40%) and Canada, Italy, Japan, and the UK (prevalence in calves 1.7–48.8%).The highest figure was for carriage by calves with a functioning rumen rather than cows or heifers. Prevalence was higher in warmer months than in cooler months. (Hussain and Bolinger 2005; Bell, 2002).

E. coli O157 can also be present in sheep and pigs; in a study conducted in Great Britain in 2003, intestinal contents of 4.7% of cattle, 0.7% of sheep, and 0.3% of pigs tested positive for *E coli* O157 at slaughter as reported by Milnes, 2008. The type of cattle (female breeding cattle) and cattle stress (movement and weaning) were identified as risk factors. Results from a study of 474 Scottish cattle farms have identified a robust pattern in which about 80% of transmission arises from the 20% of animals that are most infectious. Bovine super shedding is associated with the colonization of a lymphoid follicle-dense mucosal region at a short distance proximal to the recto-anal junction. Cattle colonized at this site shed higher numbers of organisms for a longer period than those colonized at other sites. The presence of these animals on a farm is associated with a high prevalence of low-level shedders, and they are likely to infect another animal in the same pen. Risk factors for the presence of super shedders on farms have been studied in Scotland (Chase-Tapping *et al.*, 2007).

Ground beef is still the most frequently implicated source of E. coli O157:H7 outbreaks, accounting for 75% of *E. coli* O157:H7 outbreaks (Vugia *et al*., 2006). Dairy products and undercooked minced beef can be directly contaminated by cattle feces during either milking or slaughtering processes. Results from a study of 90 outbreaks confirmed microbiologically in the UK, Ireland, Denmark, Norway, Finland, USA, Canada, and Japan, occurring between 1982 and 2006, showed that the source of transmission was food in 42.2% of the outbreaks, dairy products in 12.2%,animal contact in 7.8%, water in 6.7%, environmental in2.2%, and

unknown in 28.9% (Snedeker *et al*, 2009). Many foods and dairy products have acted as vectors—ground beef hamburgers, ready-to-eat cold meats including poultry, pork and beef products, cheese, milk; butter; yoghurt; ice cream, apple juice, coleslaw; lettuce, spinach, sprouts, and melons (Rangel *et al*, 2005). The list continues to expand—eg, consumption of prepackaged raw cookie dough was strongly associated with a multistate outbreak in the USA in 2009, with 72 cases of *E coli* O157 infection, ten with haemolytic uraemic syndrome (CDC, 2009). Waterborne outbreaks have been associated with recreational waters (lakes, ponds, and paddling and swimming pools), drinking water (municipal and local, from springs and wells), and ice. Outbreaks attributable to direct and indirect contact with ruminant animals have occurred on farms, agricultural shows (UK), county fairs (USA), open farms and camps.

The dominance of ground beef as a vector in the USA has been striking; it was the transmission route in 41% of food borne outbreaks between 1982 and 2002. Such outbreaks are rare in the UK, where butcher-associated outbreaks have occurred much more often than in any other country; 30 outbreaks were recorded between 1995 and 2004 (FSA, 2007). In the USA, 24 multistate outbreaks were recorded between1992 and 2002, with at least one occurring per annum. In 2006, the incidence of infection per 100 000 in European countries was 2.1 in England and Wales, 2.87 in Ireland, 4.7 in Scotland (from 1998 to 2007 the mean yearly rate was 4.28), 0.43 in Germany, and 0.08 in France in the same year, the incidence was about 1.3 and in Canada. (EFSA, 2010; CDC, 2009). The incidence of all Shiga-toxin-producing organisms (including *E coli* O157) in Japan from April, 1999, to October, 2004, was 2.74 (Sakuma *et al*, 2006).

Major national differences exist also in the proportion of isolates of verocytotoxin-producing organisms accounted for by *E coli* O157, ranging from 99.6% in the UK, to 93.7% in Canada (2004), 74.27% in the USA (2005), to 30.5% in Germany, where serogroups O103, O26, O91,O145, and a sorbitol-fermenting strain of serogroup O157are common(EFSA, 2010). Such strains of *E coli* O157 have also been found in the Czech Republic, Austria, Finland, Scotland, and Australia. The serogroup O111 has had an important public health effect in Australia, whereas O157 has predominated in New Zealand (Leotta *et al*, 2008). Substantial regional variations exist within countries. Studies in Sweden and Canada using geographical information systems have shown that the incidence of human disease is greater in rural areas, which have high densities of cattle and sheep, than in urban areas (Kistemann *et al.*, 2004).

The review of secondary spread in the 90 outbreaks that arose between 1982 and 2006 (Snedeker *et al.,* 2009) ranked the route of secondary transmission as person to person in the home (45.6%); person to person in nurseries(11.1%); recreational water (ie, swimming and paddling pools, 10%); person to person in institutions (4.5%); and others and unknown (5.5%). The highest mean proportion of secondary cases was recorded in outbreaks in which patients had a median age of less than 6 years. The lowest was in outbreaks in which median age of patients was 17–59 years.

A recent study on the persistence of *E. coli* O157 in irrigation waters that could potentially be transmitted to fresh produce was conducted in Kubanni River in Nigeria (Chigor *et al*., 2010). The prevalence of the pathogen in the river was studied over a 10-month period. The detection rate for *E. coli* O157 was 2.1% and fecal coliform counts exceeded acceptable limits. The researchers concluded that the Kubanni River represented a public health risk and unfit for fresh produce irrigation. The factors responsible for the emergence of the problem are:

1. Changes in the produce industry
    - Intensification and centralization of production
    - Wider distribution of produce over longer distances
    - Introduction of minimally processed produce
    - Increased importation of fresh produce
2. Changes in consumer habits
    - Increased consumption of meals outside the home
    - Increased popularity of salad bars
    - Increased consumption of fresh fruits and vegetables, and fresh fruit juices
    - Increased size of at-risk population
3. Enhanced epidemiological surveillance
4. Improved methods to identify and track pathogens
5. Emerging pathogens with low infectious dose. (Brandl, 2006)

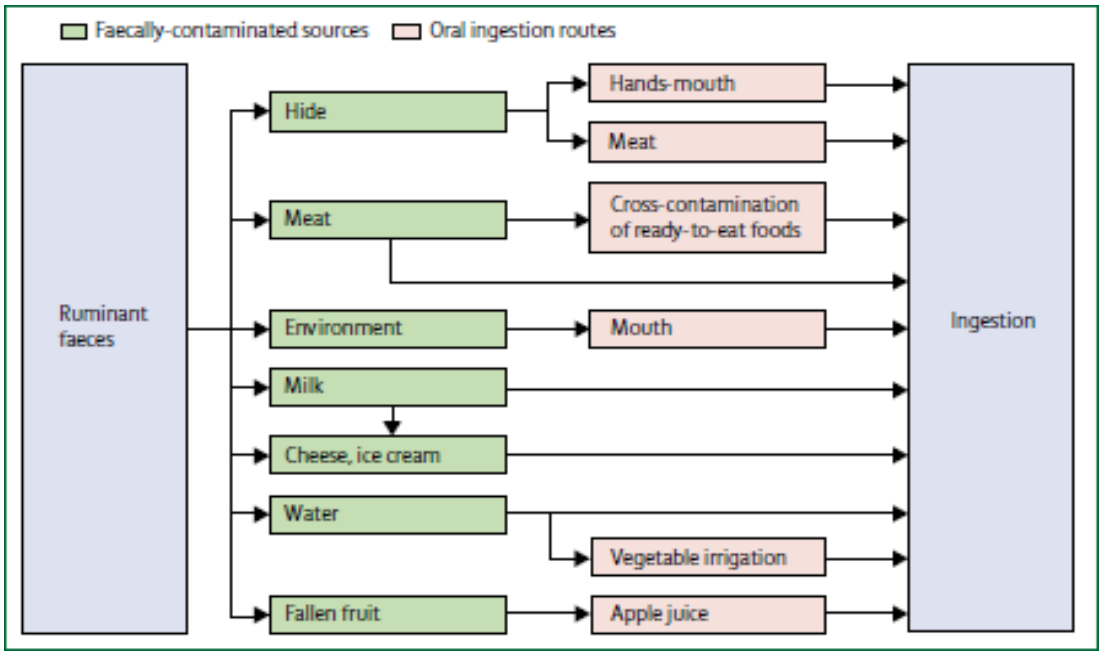

Figure 1: Routes of Transmission

Disease Caused by *E coli* O157

One of the main characteristics of EHEC that are required to cause disease in humans is their ability to attach to intestinal cells and to colonize the human gut .The infectious dose of EHEC is very low(1-100 CFU) which is a very lower compared to that of other pathogens (Paton and Paton, 1998). Since the 1980s, EHEC strains have been established as food borne pathogens associated with an array of human infections including Hemolytic Colitis, milder forms of diarrheal illness, and as the major etiologic agent responsible for the fatal infection, HUS. In general, infection with EHEC O157:H7 is self-limiting, which depends on the virulence of the infecting strain. Hemolytic Colitis is the principal disease associated with EHEC and is characterized by severe abdominal cramping and bloody diarrhea. HUS may eventually develop as a sequel to EHEC infection and HC. Approximately 8% of those infected with EHEC O157:H7 will develop HUS (Tarr, 2009). The endothelial cell damage leads to swollen detached endothelial cells, which in turn exposes the basement membrane. Hemolytic anemia is an abnormal breakdown of erythrocytes. This results from clots and possible side effects from leukocytes on the erythrocyte cell membranes.

Outbreaks vary in the severity of illness and the frequency of the most serious complication, the haemolytic uraemic syndrome; because of differences in the virulence of the causative *E coli* O157 strains (Manning *et al*, 2008). The effects of an *E.coli* O157 infection range from asymptomatic to lethal. Children less than 5 years of age have a higher incidence of HUS. They express higher levels of the Gb3 receptor present on the renal endothelial cells and form an attachment to Shiga toxin that may be circulating due to infection. Renal injury occurs from leukocyte infiltrates and clots that may lead to acute renal failure and azotemia. Azotemia is characterized by the increase of nitrogenous compounds due to poor filtering by the kidneys (Tarr *et al*., 2005).

Isolation and Diagnosis of *E coli* O157

According to the USDA's Food Safety and Inspection Service (FSIS), ground beef is considered adulterated if as little as 1 CFU of EHEC O157:H7 is detected in 25 g of ground beef. Rapid diagnosis is essential. Early separation of infected individuals from their siblings will substantially reduce secondary transmission, and the development of oligoanuric renal failure is associated with delays in the start of intravenous volume expansion (Pallock *et al*, 2009). The earlier epidemiological investigations of outbreaks start, the sooner control measures can be implemented.

Enteric bacteria have similar physiological characteristics and therefore enrichment may cause the outgrowth of competitive microflora as well). *E coli* O157 is identified by culture on selective indicator media (Sorbitol MacConkey or the same agar containing cefi xime and tellurite) (HPA, 2010; CDC, 2009). To improve selective growth of *E. coli* O157:H7, selective media have been developed through the addition of antibiotics. There are three types of enrichment media that are often used when recovering *E. coli* O157:H7: buffered peptone water supplemented with 8 mg/L Vancomycin, 10 mg/L cefsulodin, and 0.05 mg/L cefixime, modified EC broth (mEC with novobiocin), or mTSB with 20 mg/L novobiocin or 10 mg/L acriflavin (OIE, 2006). Strains of *E. coli* O157 are relatively easy to isolate because of their unique biochemical characteristics. STECO157 is unable to ferment the carbohydrate Sorbitol, which led to the development of the SMAC agar used for its isolation. More specific media have also been developed, such as Rainbow Agar, CHROM agar, and O157:H7 ID agar, that are able to recover STECO157 along with sorbitol-fermentingO157 and non-O157 strains

(Bettelheim, 2007). SMAC containing Cefixime and Tellurite (CT-SMAC) provides highly selective recovery of E.coli O157:H7 from other E. coli and enteric bacteria. Currently, CT-SMAC is widely used to isolate E. coli O157:H7 followed byPCRor latex agglutination confirmation. However, the use of CT-SMAC is not recommended for detection of non-O157 EHEC because most non-O157 EHECs that produce Shiga toxins behave physiologically the same as other commensal *E. coli* strains (Arthur *et al*., 2002). Overnight colonies are colorless and have a diameter of 2–3 mm. Their identity is confirmed by agglutination with specific antiserum.

Immunomagnetic beads have been designed for the capture of the O antigen of O157 and some have been developed for the most commonly reported non-O157 strains such as O111 andO26 (Oxoid, Inc.). Enrichment broth culture and immunomagnetic separation with antibody-coated beads are used to increase the sensitivity of culture methods in outbreak investigations and food testing. Retrospective diagnoses are sometimes made by measurement of antibodies to lipopolysaccharide (Chart and Cheasty, 2008). Non-O157 serotypes can be differentiated from commensal *E. coli* by using specialized molecular techniques such as multiplex PCR. In the UK, strain differentiation by phage typing and pulsed-field gel electrophoresis is done at reference laboratories in London and Edinburgh. A few phage types dominate; pulsed-field gel electrophoresis is much more discriminatory than is phage typing and has been widely used in outbreak investigations (Pennington, 2001).

Antimicrobial Resistance

Multidrug resistant strains of *E. coli* from food, animal and humans are increasingly being encountered. The most frequently reported resistance phenotype of *E.coli* 0157:H7 and non-0157 isolates are found to resist streptomycin, sulfixoxazole and tetracycline. Increasing resistance of Fosfomycin the drug of choice for paediatric gastrointestinal infections due to shiga toxin *E. coli* infection in Japan has been documented (White *et al*., 2002). Resistance level in *E. coli* have been reported to be high for broad spectrum penicillins and trimethoprim, and low for third generation cephalosporins and nitrofurantoin (Van Baum and Marre, 2005).It is hypothesized that adaptation to antimicrobials by bacteria is an essential survival strategy particularly for microbes having their environment within the host (Mayhofer *et al*., 2004). An aspect of gene transfer that is particularly worrisome is that genes resistance to a

number of antimicrobials can move en mass from one microbe to another, thereby enabling a single horizontal transfer to confer multi drug resistance as reported.

Control

i) Temperature

Thermal processing is one of the most common interventions applied to foods to inactivate EHEC. The heat sensitivity of the pathogen has been extensively studied and reviewed. In recent years, mild heat treatments and high temperature short time treatments have been evaluated to inactivate STEC on raw produce and meat. However, a new
study raised an important question. Pasteurization temperatures have been validated for STEC, but not for free Shiga toxin (Rasooly and Do, 2010).

ii) High pressure

The application of high pressure processing (HHP) to enhance the safety of seeds or sprouts has been studied in the past (Penaset *al.*, 2008) and various degrees of efficacy have been shown.

iii) Ionizing irradiation

Food irradiation uses high-energy gamma rays, electron beams, or X-rays; all are penetrating processes and are used commercially to eliminate pathogens from meat products. Irradiation may be better than most technologies in penetrating fresh produce and it could be a powerful tool if used correctly in different produce items and among different varieties. Irradiation is able to effectively eliminate E. coli O157: H7 from lettuce (Niemira *et al*., 2002).

iv) Ozone

Ozone destroys microorganisms through progressive oxidation of critical cellular components, with the cell surface suggested as the primary target of the process. Chlorine, one of the most commonly used disinfecting agents, destroys certain intracellular enzyme systems, while ozone causes widespread oxidation of internal cellular proteins ultimately leading to rapid cell death (Komanapalli and Lau, 1996).

v) Cinnamaldehyde

There has been an increased interest in the development and application of new effective and nontoxic antimicrobial compounds. Plant essential oils (EOs) have been found to have antimicrobial activity against a multitude of pathogens and show promise as an alternative to the currently used sanitizers. Plant-derived EOs can be used as flavoring agents in foods and beverages and have potential as natural agents for food preservation due to their content of antimicrobial compounds. Cinnamon oil is commonly used in the food industry because of its special aroma. Amalaradjou *et al*. (2010) treated polystyrene plates and urinary catheters inoculated with uropathogenic *E.coli* (5–6.0 log CFU) with difference concentrations of trans-cinnamaldehyde at 37^0C. They found that all concentrations of the antimicrobial resulted in effectively preventing the pathogen from forming a biofilm on plates and catheters, while producing no cytotoxic effects on human bladder epithelial cells.

vi) Electrochemically activated water

Electrochemically activated water (EAW) has been reported to have strong bactericidal effects on most pathogenic bacteria that are important to food safety (Huang et al., 2008). EAW is produced by passing a diluted salt solution through an electrolytic cell that contains an anode and cathode separated by a membrane. By subjecting the electrodes to direct current voltages, negatively charged ions such as chloride and hydroxide in the diluted salt solution move to the anode and become oxygen gas, chlorine gas, hypochlorite ion, hypochlorous acid, and hydrochloric acid, while positively charged ions move to the cathode to take up electrons becoming hydrogen gas and sodium hydroxide The main advantage of EAW is its safety.

Vaccination

Vaccination is used to prevent pathogen colonization and fecal excretion in ruminants, and it is based on inducing the animal's immune system to protect itself from antigens expressed by E. *coli* O157:H7. In a recent clinical vaccine trial, commercially fed cattle were used to test the effect of a two-dose regimen of a vaccine against type III secreted proteins of *E. coli* O157:H7 (Smith *et al*., 2008). The study found that pens of vaccinated cattle were less likely to test positive for E.coli O157:H7.

Feed management

Feed management has been suggested as a viable method to affect conditions within ruminant gastrointestinal tracts and ultimately modify the survival of *E.coli* O157:H7. Corn silage, barley and beet pulp have been found to increase the prevalence of O157 in cattle (Berg et al., 2004). A Lactobacillus acidophilus culture has demonstrated effectiveness at reducing *E.coli* O157:H7 in feedlot cattle by up to 50%. This particular product is currently available commercially in the United States and is being used in many large U.S. feedlots.

Conclusion

Pathogenic property of *E. coli* O157:H7 coupled with its ability to survive environmental stress make it a powerful threat to public health efforts. Commonly used methods for controlling bacteria such as boiling can not eliminate the effect of the Shiga toxin produce by the *E.coli*. Preventive measures applied to arrest the spread or transmission of the pathogen remained the best alternative. There is a need for food handlers to be educated on good hygienic practices and all necessary measures to be taken to control waste disposal especially agricultural waste to reduce the health burden that can arise from food and water sources.

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