The epidemiology, pathogenicity and microbiology of foodborne *Escherichia coli* O157:H7

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Escherichia coli are generally considered to be members of the common, indigenous microbiota of the human and other animal intestinal tracts. In fact, they are generally beneficial in several ways. Several virotypes (particular virulent strains) within the species that have the capacity for causing gastrointestinal disease in humans were discovered within the last 50 years. The virotypes have been studied particularly for their epidemiology and the mechanisms by which they are able to cause disease. The virotypes of *E. coli* described in this review are enterotoxigenic, enteropathogenic, enteroaggregative, enteroinvasive, diffusely-adherent, and enterohemorrhagic (EHEC). Many of the virotypes are responsible for causing widespread outbreaks of diarrheal disease, most of which are self-limiting. The exceptions are the EHEC that are featured in this review. EHEC produce shiga-like toxins and are capable of causing hemorrhagic enterocolitis and hemolytic uremic syndrome, a potentially fatal disease.

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Keywords: Escherichia coli, virotypes, EHEC, O157:H7, foodborne, gastroenteritis.

INTRODUCTION

*Escherichia coli* presents a large array of serotypes including some that are responsible for causing enteritis in humans. These serotypes are now placed into categories based upon their virulence profiles, termed virotypes. The virulence features enable the strains within these virotypes to compete better with the intestinal microbiota, cause disease in the host and eventually to be transmitted to a secondary host. *Escherichia coli* enteric diseases can sometimes be so characteristic that specific syndromes can be further described. In this regard, it is important to understand that this is a unique situation where disease is not necessarily related to a particular genus and species, but to specific subpopulations within a normally innocuous bacterium, which happen to contain the genetic elements responsible for virulence in the host.

VIROTYPES OF *E. COLI*

Enterotoxigenic *E. coli* (ETEC)

ETEC is one of the most common agents of diarrhea in developing countries [1,2]. ETEC infection results in a non-inflammatory diarrhea...
caused by a heat labile exotoxin that closely resembles the cholera toxin of *Vibrio cholerae*. The toxin is absorbed into the intestinal cells, thus resulting in the elaboration of fluids into the intestinal lumen. ETEC attachment to the intestinal tissue is mediated by proteinaceous surface appendages (colonization factors) and is an important first step for eventual secretion of labile exotoxin.

**Enteropathogenic *E. coli* (EPEC)**

EPEC have a peculiar capability of causing disease after attaching intimately to intestinal cells. The mechanism produces distinct cup-like pedestals around each bacterium resulting in the destruction of local microvilli, and is referred to as an attaching and effacing (A/E) phenomenon. This pathology presumably results in the debilitating diarrhea attributed to EPEC serotypes.

Intestinal colonization by EPEC is apparently dependent upon intimin-mediated intimate contact, which involves a type III secretion system that first translocates an intimin receptor into the host cell [4]. This concept is in contrast to an earlier three-stage model, which proposed the bundle-forming pilus as the important component for the initial attachment of EPEC to the host cell surface.

**Enteroaggregative *E. coli* (EAEC)**

EAEC bind to Hep-2 cells but in a pattern unlike the localized adherence pattern of EPEC. The binding of EAEC was initially described as diffuse adherence, but was then further divided into two subgroups: aggregative adherence and diffuse adherence [1]. The EAEC do not secrete toxins but cause a chronic or persistent form of diarrhea [5]. They possess two types of fimbriae: aggregative adherence fimbriae I (AAF/I) and II (AAF/II).

**Enteroinvasive *E. coli* (EIEC)**

EIEC have the ability to invade colonic cells and multiply while spreading laterally to adjacent cells [6]. The disease syndrome produced by the EIEC infection is similar to shigellosis produced by *Shigella flexneri*, *S. boydii* and *S. sonnei*. EIEC, however, do not produce shiga toxin. Otherwise, EIEC and *Shigella* spp. exhibit similar genetic and pathogenic features.

**Diffusely adherent *E. coli* (DAEC)**

The DAEC appear to be diarrheagenic in children older than infants. However, an increased risk of diarrhea was associated with DAEC in children between the ages of 1 through 5 years in Santiago, Chile [7]. Conversely, other studies indicate that DAEC has no association with diarrhea [1]. Potential virulence features remain to be completely understood.

**ENTEROHEMORRHAGIC *E. coli* (EHEC)**

Discovery

EHEC O157:H7 was first isolated in 1975 from a woman with bloody diarrhea, but it was not considered a significant human pathogen until 1982. At that time it was associated with two foodborne outbreaks in the USA traced to the ingestion of undercooked, contaminated hamburger meat [8,9]. Considered a rare isolate, the O157:H7 serotype of *E. coli* was implicated as the agent of disease. Hemolytic uremic syndrome (HUS), a disease syndrome characterized by thrombocytopenia, microangiopathic hemolytic anemia and acute renal failure, was also associated with several of the victims in the 1982 outbreaks.

The discovery of cytotoxin-producing *E. coli* in stools from outbreak victims led Karmali et al. [10] to speculate that there was a connection between the toxin in the stools and the onset of HUS. Three independent researchers reported the production of a cytotoxin by certain strains of *E. coli* isolated from feces [11–13]. Konowalchuk et al. were the first group to discover the toxin's cytotoxic properties against Vero cells (tissue-cultured kidney cells from the African green monkey) [11]. The term VTEC (Vero-toxigenic *E. coli*) was therefore coined to describe *E. coli* cells that produced this cytotoxin. O'Brien et al. [12,14] later determined that the in vitro cytotoxicity effects of *E. coli* extracts could be neutralized by the addition of antiseraum specific for *Shigella dysenteriae* 1 toxin (shiga toxin), and thus called the *E. coli* toxins 'shiga-like toxins'. *Escherichia coli* strains that produced shiga toxin (Stx) were named either VTEC (for the cytotoxic effects on vero cells) or STEC (for the toxin homology to shiga toxin). VTEC and STEC referred to the same types of bacteria: i.e., *E. coli* that produced one or more Stx.
With the advent of rapid methods to identify shiga toxin in foods and stools, an alarming number of E. coli serotypes have been identified that produce the toxin. Most of these serotypes, however, have not yet been associated with human disease [15]. Thus the term EHEC was coined to further categorize STEC by describing all strains of E. coli that expressed Stx, caused hemorrhagic colitis (HC) and/or HUS, produced A/E (attaching and effacing) lesions, and that possessed a 60-MDa plasmid [2]. Since the original classification of E. coli O157:H7 as a foodborne pathogen in 1982, the O157:H7 serotype has been implicated in an increasing number of outbreaks each year. Indeed, E. coli O157:H7 was the first documented EHEC and is the most common diarrheal isolate implicated in hemolytic uremic syndrome in the USA. For these reasons, E. coli O157:H7 is considered the prototypical serotype of enterohemorrhagic E. coli.

It should be noted that the incidence of EHEC outbreaks in the USA and other developed nations is not nearly as high as the incidence of outbreaks caused by some other virotypes of E. coli. The worldwide attention given to O157:H7 is due mainly to the severity of the associated disease syndrome. While the other virotypes of E. coli cause self-limiting diarrheal disease, EHEC has been involved in many devastating disease syndromes including explosive bloody diarrhea, kidney failure, neurological damage and death [16]. For this reason, the EHEC virotype is featured in this review.

Evolution of E. coli O157:H7

The introduction of E. coli O157:H7 into the world’s food supply within the past two decades is an example of the consequences when a deadly emerging pathogen enters the food chain. With the exception of 1996 and 1997, the number of reported outbreaks in the USA (Table 1) has increased every year since the first two reported outbreaks in 1982; however, these data have been attributed in part to increased surveillance and improved detection technology [17]. Retrospective data suggest that this pathogen did cause infections prior to 1982, but at a very low incidence rate. The questions that today’s epidemiologists and systematists ask relate to how and why E. coli O157:H7 gained a worldwide niche as a formidable foodborne pathogen. Current epidemiological evidence suggests that E. coli O157:H7 had evolved from an EPEC ancestor, as revealed using techniques such as pulsed-field gel electrophoresis and restriction fragment length polymorphism techniques. Feng et al. [18] provided a logical sequence of events by which E. coli O157:H7 might have evolved from an ancestral EPEC strain. Using multilocus enzyme electrophoresis to study changes in the uidA gene (encodes the enzyme glucuronidase, GUR), as well as correlating phenotypic changes in GUR production, sorbitol fermentation ability and toxin production (Stx), Feng provided genetic evidence that E. coli O157:H7 evolved sequentially from an O55:H7 ancestor.

VIRULENCE FACTORS

Shiga toxins

Shiga toxin, perhaps more than any other virulence factor, is responsible for the devastating disease syndrome caused by EHEC infections. E. coli O157:H7 can express one or both of the antigenically distinct (not cross-reactive) toxins: Stx1 only, Stx2 only, or both toxins. Stx1 structure is highly conserved and almost identical in structure and function to shiga toxin produced by S. dysenteriae type 1 [19]; Stx2 has several variants, each of which differs from shiga toxin. The ability of E. coli strains to transfer the capacity to produce Stx from one strain to another was described first by Smith and Linggood in 1971, but they were unable to determine the mechanism of transference until 1983, when the link between Stx production and

Table 1. Outbreaks in the USA involving E. coli O157:H7 from 1982 to 1998.

<table>
<thead>
<tr>
<th>Year</th>
<th>Outbreaks</th>
<th>Total cases</th>
<th>Median cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1982-1992</td>
<td>23</td>
<td>823</td>
<td>26</td>
</tr>
<tr>
<td>1993</td>
<td>17</td>
<td>1000</td>
<td>11</td>
</tr>
<tr>
<td>1994</td>
<td>32</td>
<td>543</td>
<td>12</td>
</tr>
<tr>
<td>1995</td>
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<td>298</td>
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</tr>
<tr>
<td>1998</td>
<td>42</td>
<td>777</td>
<td>7</td>
</tr>
<tr>
<td>Total</td>
<td>197</td>
<td>4384</td>
<td></td>
</tr>
</tbody>
</table>

*Adapted from information bulletins provided by the Food Safety Initiative Activity, National Center for Infectious Diseases, Centers for Disease Control and Prevention (courtesy of P. H. Sparling).
the lysogenic cycle of a bacteriophage was confirmed [20].

The structure of the Stx toxin produced by EHEC is consistent with the traditional A–B subunit family of toxins. The structure is that of a 32-kDa protein ‘A’ subunit that is non-covalently bound to five ‘B’ subunits of 7-kDa each. The ‘B’, or ‘binding’, subunits are responsible for attaching to the eukaryotic cell surface receptor glycolipid Gb3 (globotriosyl ceramide). Basically, any cell that contains the Gb3 receptor is in danger of being destroyed by this toxin. Karmali et al. [21] hypothesized that the shiga toxin of E. coli O157:H7 may enter the bloodstream and cause damage to the vascular endothelial cells of the kidney, which have high concentrations of the Gb3 receptor.

**Enterohemolysin**

Escherichia coli O157:H7 has been shown to produce several hemolysins. The enterohemolysin (ehx) is encoded on a 60-MDa plasmid (called PO157) that is found in most E. coli O157:H7 and other Stx producing serotypes of E. coli [22]. The ehx belongs to the RTX family of hemolysins and has a 60% DNA homology with the hemolysins seen in uropathogenic E. coli (hlyA) [22,23], which lysed eukaryotic cells by forming pores in the cellular membranes. While the association of ehx with EHEC strains is significant enough to provide a molecular diagnostic tool for EHEC strains [24], the role of ehx in pathogenesis is not clear.

**Intimin**

The ability of EHEC to attach to (and subsequently efface) intestinal epithelial cells is largely the result of a 94–97-kDa outer membrane protein called ‘intimin’. This protein, encoded by the eae gene, is located on the LEE (locus of enterocyte effacement) pathogenicity island of the chromosome. It has been determined to play an essential role in ‘intimate’ binding not unlike that of enteropathogenic E. coli (EPEC). EHEC and EPEC intimin share about 86% nucleotide homology [25], and about 50% homology at the C-terminal end, which is the receptor-binding region. This may be one reason why EHEC infections localize in the large intestine, while EPEC infections are located in the small intestine.

**Translocated intimin receptor (Tir)**

Tir, a 90-kDa protein, is mapped within the LEE pathogenicity island and is translocated into the host via a type III secretion system. Tir consists of at least three domains, one of which functions as a receptor for intimin, and another involved in actin alignment and eventual pedestal formation.

**Extracellular serine protease (EspP)**

The extracellular serine protease EspP is encoded on the large plasmid of enterohemorrhagic E. coli (EHEC) O157:H7. EspP is widespread among EHEC of the serogroup O157 and also exists in serogroup O26. Patients suffering from EHEC infections have demonstrable antibodies against this protein. EspP appears to be a protease capable of cleaving pepsin A and human coagulation factor V, which might in turn lead to the bloody diarrhea observed in hemorrhagic colitis [26].

**DISEASE CHARACTERISTICS**

**Hemorrhagic colitis (HC)**

Hemorrhagic colitis due to EHEC infections was first characterized during the two early outbreaks of E. coli O157:H7 [9]. Bloody diarrhea has since become the hallmark symptom for E. coli O157:H7 infections around the world. However, E. coli O157 has also been determined to cause mild, non-bloody diarrhea symptoms among young to middle-aged adults and may often be undetected [27].

A prospective study in 1987 by Ostroff et al. [28], which involved 93 patients throughout the state of Washington in the USA with E. coli O157:H7 infection, reported that the earliest symptoms were both non-bloody diarrhea and abdominal cramps. Bloody diarrhea did not begin until the second or third day of illness (but occurred in 95% of the cases). The incubation period for HC was 3–4 days with an average duration of 7 days [28]. In most cases, HC is a self-limiting disease; however hospitalization is recommended due to the possibility of human-to-human transmission. Six to twelve percent of the cases may progress to more severe forms of disease such as hemolytic-uremic syndrome (vide infra).
The estimated infectious dose of E. coli O157:H7 has been somewhat equivocal. Because of extensive critical disease syndromes caused by this organism, human volunteer studies have not been performed. Extrapolation of quantitative data from foods implicated in outbreaks has placed the dose between 10 and 100 colony-forming units per gram [29]. This number is similar to Shigella spp., which has been shown to cause dysentery at a very low infectious dose. This would explain outbreaks associated with human-to-human and waterborne transmission, including an outbreak associated with swimming in a freshwater lake in Oregon [30].

Hemolytic uremic syndrome (HUS)

HUS is the leading cause of kidney failure among children in the USA [31]. HUS can manifest as hemolytic anemia, thrombocytopenia and possibly renal failure. The association between HUS and EHEC was not established until 1985 [32], although epidemiological data supports the probability that cases of HUS may have been caused by EHEC as early as 1970 [33].

Escherichia coli O157:H7 adherence factors are critical for development of the initial diarrheal symptoms, and Stx development is critical for progression of the more severe complication of HUS. HUS develops in about 6% of patients with E. coli O157:H7 infections, most often occurring in young children or the elderly [34]. The age pattern of HUS development suggests that immunity may play a role in protection against disease progression. Interestingly, recurrent cases of HUS (about 2.6% of all reported cases up to 1993) [35] showed no symptoms of diarrhea prior to the second onset of HUS. This suggests a protective immunity against bacterial factors responsible for diarrhea, but no protection against the infection and subsequent toxin release.

EPIDEMIOLOGY

Reservoir

Beef cattle have been recognized as carriers of E. coli O157:H7 since the first outbreaks were traced to hamburgers. However, E. coli O157:H7 has also been isolated from several non-ruminant animals, including sheep [36], goats [37], pigs [38], horses [39], dogs [39], birds [39], as well as flies [39]. Cattle are the most important source of this pathogen in human disease, as most of the outbreaks of EHEC O157:H7 are traced to the consumption of undercooked ground beef. The prevalence of E. coli O157:H7 carriage in cattle has been reported in several studies to be 1.1–6.1% in midwestern herds of the USA [39], 0.8–22.4% on Dutch dairy farms [40], 0.62% in Japanese cattle [41], and 1% in Norwegian cattle herds [42]. Studies involving carriage in ruminants showed a higher prevalence among calves and a higher shedding rate in the summer months.

The low prevalence of E. coli O157 among cattle is interesting as it is the most clinically significant EHEC in the USA. This fact suggests that significant contamination problems exist during the processing of cattle carcasses. The potential for transmission from farm animals to humans was confirmed by serological surveys of Canadian dairy farmers. Sera collected from dairy farmers had a threefold higher antibody level to O157 lipopolysaccharide (LPS) than those collected from urban families [43].

Transmission

Escherichia coli O157:H7 is classified as an ‘adulterant’ in raw meat products by the Food Safety Inspection Service (FSIS), a branch of the United States Department of Agriculture (USDA) responsible for the microbiological testing and regulation of the meat and poultry supply in the USA. This is the first microbiological pathogen in history that the FSIS has labeled as an adulterant, and appropriately so because of the low infectious dose that can cause such a potentially devastating disease syndrome. Classification as an ‘adulterant’ means that the presence of a single cell of E. coli O157:H7 in any raw meat or poultry product will require condemnation of the product. This classification has had a significant impact on the meat industry in the USA, resulting in the recall of contaminated meats. The largest recall to date is the 25 million pounds of hamburger meat by Hudson Foods, Inc. in the USA in 1997 [44].

Most of the outbreaks involving E. coli O157:H7 are traced to a single food source, suggesting ingestion as the major route of entry for the organism. Ground beef has been implicated in most outbreaks of E. coli O157:H7 in the USA,
the largest being traced to undercooked hamburgers served from a fast food chain in December 1992 through January 1993. During this occurrence, known as the 'Jack-in-the-Box' outbreak, 732 individuals were affected in Washington, California, Idaho and Nevada [29]. The production of ground beef has become a centralized operation in which thousands of cattle carcasses are mixed together in a single batch process. Despite the low prevalence of E. coli O157:H7 in beef cattle, a single carcass carrying the organism is able to contaminate the entire batch of ground beef.

Different types of foods have been implicated in outbreaks with E. coli O157:H7, including many of which were originally thought to be safe because of intrinsic acidic or low moisture conditions. Some foods from which EHEC have been isolated include roast beef [27], venison jerky [45], salami [46], raw milk [47], yogurt [48], unpasteurized apple cider [50], or juice [51], cantaloupe [52,53], potatoes [54], radish sprouts [55], and alfalfa sprouts [55]. Outbreaks associated with high intrinsic acidity (e.g., apple cider) and low water activity (e.g., deer jerky, salami), and fresh produce, suggests that new guidelines are necessary for the production, sale, and consumption of ready-to-eat foods that were once thought to be safe.

Waterborne outbreaks associated with swimming water [30] and drinking water [56] have been documented, and secondary cases of infection due to inter-personal transmission have also been documented [57]. These EHEC O157 infections support the hypothesis that very low doses of E. coli O157:H7 can cause disease.

Therapy

The recommended therapy for EHEC infections in the USA is currently limited to supportive care. Retrospective studies of the 1996 Japan outbreak by Shiomi et al. [58] determined that administration of oral fluoroquinolone therapy within 3 days of illness is effective in decreasing the risk of developing HUS. Antibiotic therapy, however, has associated risks. Antibiotic therapy with drugs that inhibit cell wall synthesis has been shown to induce an increase in production and release of Stx from E. coli O157:H7 [59], which may elicit a worsening of symptoms. Prospective studies are required for further analysis to ascertain if antibiotic therapy is effective for E. coli O157:H7 infections.

Prevention

Hazard Analysis and Critical Control Points (HACCP) criteria are a step in the right direction to curb the prevalence of E. coli O157:H7. Microbial analyses will determine where contamination occurs, prompting immediate action such as plant remediation, product recalls and consumer bulletins. Current manufacturing processes allow a single contaminated carcass to contaminate huge batches of ground beef that can now be distributed worldwide. Current farming legislation does not effectively control the distribution of contaminated manure onto crops and produce.

While it is acid resistant, E. coli O157:H7 is not considered heat resistant, and, in fact, is more heat sensitive than Salmonella [60]. In this regard, consumer education may be the most effective defense against this pathogen by informing consumers that thorough cooking of all raw meat products should be conscientiously observed. The USDA recommends using a meat thermometer in hamburgers to ensure that an internal temperature of 155°F (68.5°C) is maintained for 16s. Farms and packing plants that handle vegetables and fruits may also require HACCP protocols to help prevent the spread of this pathogen.

Recent research has discovered that the carriage and shedding of EHEC by cattle is dependent upon the type of food and duration of feeding prior to slaughter. Hovde et al. [61] discovered that cattle fed a hay-based diet shed E. coli O157:H7 longer than cattle fed a grain-based diet. The implication was that cattle should be switched to a grain-based diet prior to slaughter. Slaughterhouses have experimented with the application of a variety of organic and inorganic acid and hot water sprays to decontaminate the surfaces of carcasses containing E. coli O157:H7. Several studies have shown that acetic acid and lactic acid, trisodium phosphate and hot water or steam are good methods of reducing, but not eliminating E. coli O157:H7 from the surfaces of contaminated carcasses [62]. Currently, the only effective means of ensuring the elimination of this pathogen from every type of food product is irradiation [63]. The general consumer has concerns with using
gamma irradiation to treat food products, most of which are not substantiated (such as the fear of eating radioactive food).

Detection

Escherichia coli O157:H7 has two major characteristics that allow this serotype to be distinguished from other E. coli using traditional culture methods. First, approximately 85% of non-O157:H7 E. coli ferment sorbitol, while O157:H7 isolates will either ferment sorbitol slowly or not at all [64]. Secondly, unlike the O157:H7 serotype, approximately 92% of E. coli produces the enzyme glucuronidase (GUR). To make use of this feature, 5-bromo-4-chloro-3-indoxyl-β-D-glucuronide is added to solid media that produces a visible, pigmented compound when GUR is present [65]. These two characteristics are currently used to differentiate this pathogen from the other ‘background’ E. coli microbiota.

The first plating medium used for the isolation of E. coli O157:H7, called SMAC, is a MacConkey’s-based medium that substitutes sorbitol for lactose, allowing the differentiation between sorbitol and non-sorbitol fermenters [64]. Another diagnostic medium for the isolation of EHEC in general, and E. coli O157:H7 in particular from food and fecal material is Rainbow O157 Agar (Biolog, Inc., Hayward, California, USA). This medium is more selective and specific than SMAC for detecting and isolating E. coli O157:H7, as well as for differentiating many other EHEC serotypes from non-pathogenic E. coli [66].

Selective enrichment broths have been designed to propagate E. coli O157:H7 prior to direct plating. Enrichment is commonly necessary to enhance the sensitivity of direct plating due to the low infectious dose and low prevalence in food and environmental samples. Many of the newer ‘rapid assays’ listed below are used in conjunction with an enrichment step necessary for increased sensitivity. Common enrichment broths include a modified E. coli broth supplemented with novobiocin (mEC+n, 20 mg/l) and modified trypticase soy broth supplemented with cefixime (50 μg/l) and vancomycin (40 mg/l).

Regardless of the enrichment and plating medium used for the isolation of E. coli O157:H7, presumptive isolates must be confirmed biochemically as E. coli (Table 2) and serologically as O157. Several Escherichia hermanii strains have been known to serologically cross-react with the O157 serotype; therefore, cellobiose should be added to the list of biochemicals (E. hermanii ferments cellobiose and E. coli does not). Diagnostic kits combining antiserum to the O157 LPS antigen with latex beads are commercially available (LMD Laboratories, Inc., Carlsbad, California, USA.; Oxoid Limited, Basingstoke, Hampshire, UK; Remel Microbiology Products, Lenexa, Kansas, USA; Meridian Diagnostics, Cincinnati, Ohio, USA) which are much easier to perform than the standard tube agglutination method. Escherichia coli O157:H7 may require several passes through motility medium before it is able to agglutinate with the H7 antiserum. The Centers for Disease Control and Prevention (Atlanta, Georgia, USA) recommends the reporting of all sorbitol-negative E. coli isolates that agglutinate with the O157 antiserum as presumptive E. coli O157:H7.

A new filter monitor-based system, termed E. coli SELeCT (Salmonella, E. coli, Listeria and Campylobacter Test), incorporating a colony-lift immunoassay has been developed to detect and quantify E. coli O157:H7 from beef and poultry in 24 h [67]. It is recommended as a replacement for the time-consuming and arduous most probable number method.

Rapid immunological methods have been developed to detect E. coli O157:H7 in foods and stools that offer simplicity and facilitate the processing of large numbers of samples. En-

Table 2. Typical biochemical signature of Escherichia coli.a

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
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<tbody>
<tr>
<td>Indole</td>
<td>Positive</td>
</tr>
<tr>
<td>Methyl red</td>
<td>Positive</td>
</tr>
<tr>
<td>Voges-proskauer</td>
<td>Negative</td>
</tr>
<tr>
<td>Simmon’s citrate</td>
<td>Negative</td>
</tr>
<tr>
<td>Triple sugar iron</td>
<td>Acid/acid, gas, no H₂S</td>
</tr>
<tr>
<td>Lysine decarboxylase</td>
<td>Positive</td>
</tr>
<tr>
<td>Ornithine decarboxylase</td>
<td>Variable</td>
</tr>
<tr>
<td>Lactose</td>
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<tr>
<td>Sucrose</td>
<td>Variable</td>
</tr>
<tr>
<td>D-Mannitol</td>
<td>Variable</td>
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<tr>
<td>D-Sorbitol</td>
<td>Variable</td>
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<tr>
<td>L-Rhamnose</td>
<td>Variable</td>
</tr>
<tr>
<td>Motility</td>
<td>Variable</td>
</tr>
</tbody>
</table>

a Adapted from The Bergey’s Manual of Determinative Bacteriology, Ninth Edition, Table 5.2, p. 209 [68].

b Test results based on cultures of E. coli grown at 36 ± 1°C for 24 h. Variable = 10–90% Positive results obtained at 36 ± 1°C in 48 h.
zyme-linked immunosorbent assay (ELISA) methods utilize the specificity of antibody–antigen interactions, which lowers the rate of false-negative reactions. ELISA strategies have used both polyclonal and monoclonal antibodies specific for the O157 LPS antigens, shiga toxins (Stx1 and Stx2), and products of the pO157 plasmid, that are conjugated to fluorescein, peroxidase or phosphatase enzymes.

Immunomagnetic separation (Dynal Inc., Lake Success, New York, USA) has become a useful and innovative method for concentrating E. coli O157:H7 from a variety of samples and has been used to enhance the sensitivity of direct plating, DNA-based methods, and some immunological methods. The polymerase chain reaction (PCR) has been useful for the detection of E. coli O157:H7, but is more commonly used to detect STEC and EHEC in general. Primers for the detection of E. coli O157:H7 have been developed to isolate genes encoding the O157 LPS antigen, Stx1 and Stx2, intimin (eae), GUR (uidA) and enterohemolysin. Polymerase chain reaction (PCR) is considered specific for E. coli O157:H7 only when the above targets are used together in a ‘multiplex’ PCR format.

CONCLUSIONS

Escherichia coli, a relatively benign enteric organism, has been found to contain virotypes that are capable of causing intestinal disease in humans. One of the virotypes, EHEC, is capable of causing severe hemorrhagic colitis and possibly HUS, especially in young children, primarily through the production of Stx. The main reservoir is cattle, but EHEC has also been found in various other animals, vegetables and fruit. The organism is easily grown in the laboratory and a large number of identification methodologies are available. In the clinical setting, it is recommended that all bloody stools (especially those from young children) should be cultured for E. coli O157:H7. The USDA and Food and Drugs Administration have implemented intervention procedures and recommendations in an effort to protect the public from exposure to EHEC.

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REVIEWS IN MEDICAL MICROBIOLOGY (2002) 13(2)

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Recommended additional reading


