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# Clinical Significance of Escherichia coli O157:H7 and Its Plasmid O157 – A review

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**ABSTRACT**
Enterohemorrhagic Escherichia coli O157:H7 is a common foodborne pathogen that causes serious illness in humans across the world. Bovine food items and fresh produce contaminated with bovine faeces are the most prevalent origins of disease outbreaks in the United States, and healthy cattle are a reservoir for E. coli O157:H7. E. coli O157:H7 is also a good survivor in the environment. E. coli O157:H7 must be able to adapt to a wide range of environments in order to cause human illness, colonise the bovine gastrointestinal system, and live in the environment. Shiga toxins, products of the pathogenicity island dubbed the locus of enterocyte effacement, and products of the F-like plasmid pO157 have all been identified as important virulence factors of E. coli O157:H7. pO157 has an important role among these virulence variables. The role of pO157 is the least known of these virulence factors. This article gives a general overview of E. coli O157:H7, with a focus on pO157.

**Keywords**
E. coli O157:H7, pO157

## I. ESCHERICHIA COLI O157:H7

Escherichia coli (E. coli) is a Gram-negative, rod-shaped, facultative anaerobic bacteria. Theodor Escherich was the first to characterise this bacterium in 1885. The majority of E. coli strains inhabit people and animals' gastrointestinal tracts as natural flora. However, certain strains of E. coli have developed into pathogenic E. coli as a result of virulence factors acquired via plasmids, transposons, bacteriophages, and/or pathogenicity islands. Serogroups, pathogenicity mechanisms, clinical symptoms, and virulence factors can all be used to classify pathogenic E. coli [33, 47]. Enterohemorrhagic E. coli (EHEC) is a kind of pathogenic E. coli that produces Shiga toxins (Stxs) that causes hemorrhagic colitis (HC) and the potentially fatal sequelae hemolytic uremic syndrome (HUS) in humans. Several EHEC serotypes, such as O26:H11, O91:H21, O111:H8, O157:NM, and O157:H7, have been linked to human illnesses [44, 51]. This study focuses on E. coli O157:H7, the most often isolated serotype of EHEC from unwell people in the United States, Japan, and the United Kingdom.

### History

In 1982, EHEC serotype O157:H7 was identified as a human pathogen connected to epidemics of bloody diarrhoea in Oregon and Michigan, United States of America [57, 71], as well as rare instances of HUS in 1983 [34]. Since then, several EHEC outbreaks have been documented in the United States, and E. coli O157:H7 has emerged as one of the most dangerous foodborne infections.

### Prevalence and Economic Cost

E. coli O157:H7 infections are projected to cause 73,000 illnesses, 2,200 hospitalizations, and 60 fatalities in the United States each year by the Centers for Disease Control and Prevention (CDC) [43]. According to CDC outbreak monitoring statistics, E. coli O157:H7 infections are declining following a high in 1999. Large outbreaks and isolated instances, however, continue to occur. E. coli O157:H7 infections cost $405 million per year in lost productivity, medical treatment, and untimely deaths [21]. Because of the high expense of sickness, more effort is needed to combat this infection.

### Isolation and Identification

Somatic (O) antigen 157 and flagella (H) antigen 7 are both expressed by E. coli O157:H7. E. coli O157:H7 is distinguished by its inability to produce -glucuronidase, which can hydrolyze the synthetic compound 4-methyl-umbelliferyl-D-

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glucuronide (MUG) [68] and its delayed D-sorbitol fermentation (>24 h). For the detection of E. coli O157:H7, Sorbitol MacConkey (SMAC) agar spiked with MUG was utilised. Cefixime, potassium tellurite, and vancomycin have been added to SMAC agar plates to boost the selectivity for E. coli O157:H7 and to suppress other Gram-negative flora. A commercially available latex agglutination assay can be used to confirm serotypes O157 and H7.

**Genomic Organization**

E. coli O157:H7 has a chromosomal size of 5.5 Mb. A 4. 1 Mb backbone sequence found in all E. coli strains is included in this genome. The rest are only found in E. coli O157:H7 [53]. Furthermore, a genome comparison of E. coli O157:H7 and nonpathogenic E. coli K12 reveals that E. coli O157:H7 lacks 0.53 Mb of DNA, suggesting that genomic reduction had a role in E. coli O157:H7 evolution [17, 53]. Horizontally transmitted foreign DNAs such as prophage and prophage-like elements make up the bulk of E. coli O157:H7-specific DNA sequences (1.4 Mb). There are 463 phage-associated genes in E. coli O157:H7 compared to only 29 in E. coli K-12 [72]. Putonti et al. [55] calculated that at least 53 distinct species contributed to these unique sequences in E. coli O157:H7, and a change in G+C contents is one of the indicators that a genomic area has been acquired through horizontal transfer. Two sequenced E. coli O157:H7 strains had almost similar virulence-associated genes (99 percent ). Both the acquisition and loss of DNA have clearly played a role in the development of E. coli O157:H7 pathogenesis.

**Evolution**

E. coli O157:H7 may have derived from the non-toxigenic and less virulent strain E. coli O55:H7, according to several comparative and epidemiological research [72]. Acquisition of a stx2-containing bacteriophage, (ii) acquisition of pO157 and the rfb region, (iii) acquisition of the stx1-containing bacteriophage, and (iv) loss of the ability to ferment D-sorbitol and loss of beta-glucuronidase (GUD) activity were the four sequential events that led to the emergence of E. coli O15:H7.

**Animal Reservoir**

Cattle are the most common reservoir for E. coli O157:H7, and this reservoir host is usually asymptomatic when infected. This serotype has been linked to a few occurrences of diarrheal illness in young calves. At any given period, the proportion of cattle shedding fluctuates. E. coli O157:H7 has also been detected in the faeces of sheep, goats, pigs, and turkeys.

**Molecular Subtyping**

To better understand the epidemiology of E. coli O157:H7 outbreaks, a range of molecular subtyping approaches have been developed. PFGE, restriction fragment length polymorphisms (RFLP), amplified fragment-length polymorphisms (AFLP), and phage typing are examples of these approaches [65, 73]. CDC standardised the PFGE technique, which has been used successfully to distinguish outbreak-associated, sporadic, or unrelated illnesses since 1993 [3].

**Infection**

In North America, Europe, and other parts of the world, E. coli O157:H7 infection is a serious public health risk. Despite the fact that the overall number of E. coli O157:H7 infections is fewer than other enteric pathogens like Salmonella or Campylobacter spp., the illnesses caused by E. coli O157:H7 have substantially higher hospitalisation and death rates [43]. Human infection induced by E. coli O157:H7 can have a wide range of symptoms, from asymptomatic to fatal. The majority of cases begin with non-bloody diarrhoea and resolve on their own without further complications. In 1–3 days, however, some individuals have bloody diarrhoea or HC. The condition can proceed to life-threatening complications such as HUS or thrombocytopenic purpura (TTP) in 5–10% of HC patients [1]. In the United States, E. coli O157:H7 is the most prevalent cause of HUS. Severe clinical signs such as HUS are more common in children and the elderly.

Several treatment options have been investigated, including antibiotics and immunisation. Antibiotics may be contraindicated in the case of E. coli O157:H7 infection since there is no particular therapy. As a result, therapy is mostly supportive in nature, with the goal of limiting the duration of symptoms and preventing systemic consequences. Given this situation, extremely effective E. coli O157:H7 infection prevention and control strategies are critical.

**Transmission**

Consumption of contaminated food and water is the most common mode of transmission

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for E. coli O157:H7 infections in the United States [56]. It can also be passed directly from person to person and from animal to human, especially in kid day-care centres. People who visited petting zoos, dairy farms, or camp areas where cattle had previously grazed have been infected [28, 31]. A polluted facility with an animal show has recently been identified as having the potential for airborne transmission [70]. Foodborne (52 percent), unknown (21 percent), person-to-person (14 percent), waterborne (9 percent), and animal contact (3 percent) were the established transmission pathways in the 350 outbreaks reported to the CDC between 1982 and 2002 [56]. Figure 1 depicts the E. coli O157:H7 transmission model, which has been revised from Gansheroff and O'Brien's model [23]. The relatively low infectious dosage (50 CFU) of E. coli O157:H7 explains the multiple transmission pathways. E. coli is found in cattle naturally. E. coli O157:H7 is a strain of E. coli that is resistant to antibiotic E. coli is carried by and shed by between 1% and 50% of healthy cattle. At any one moment, they may have E. coli O157:H7 in their faeces [13, 18, 27]. E. coli is most commonly spread through contaminated ground beef. Outbreaks of E. coli O157:H7 During slaughter, germs can enter beef products, and the grinding process can transmit infections from the meat's surface to its inside. As a result, germs can persist if ground beef is not thoroughly cooked. In addition to ground beef, a number of other contaminated food vehicles have been connected to E. coli O157:H7 outbreaks, including unpasteurized milk, drinking water, salami, beef jerky, and fresh vegetables like lettuce, radish sprouts, fresh spinach, and apple cider. The greatest incidence was linked to radish sprout contamination in Osaka, Japan, in 1996, when 7,966 people were diagnosed with illnesses [45]. These food products appear to have been contaminated with bovine faeces, according to epidemiological investigations. As a result, one of the most significant management techniques for E. coli O157:H7 in cattle is to avoid it.

**Acid Resistance of E. coli O157:H7**

The capacity of bacteria to protect themselves against extremely low pH (pH 3.0) is known as acid resistance (AR). One of the initial host defences against foodborne enteric infections is a low pH in the stomach (pH 1.5 to 3.0) [54]. Bacteria that can live in the acidic environment of the stomach have a greater chance of colonising the intestines and causing illness. Acid resistance has been linked to a reduction in enteric pathogen infectious doses [60]. One of the most well-known properties of E. coli O157:H7 is its low infectious dosage, which makes it extremely contagious. The AR of E. coli O157:H7 strains has been documented in several investigations [5, 12]. Three effective AR systems were discovered as a result of these researches. RpoS, an alternate sigma factor, and glucose repression are required for the first AR system. In experimentally infected mice and calves, the rpoS mutant of E. coli O157:H7 shed in reduced quantities. The addition of arginine to the second AR system is required after acidic exposure. This second AR system revealed the arginine decarboxylase (adiA) and the adiA regulator (cysB). For protection in low pH conditions, the third AR system requires glutamate. Two glutamate decarboxylase isozymes (gadA and gadB), as well as a suspected glutamate, -amino butyric acid antiporter, are essential components of this AR system (gadC). At pH 2.5, just one of the two glutamate decarboxylase isozymes is required for protection, but at pH 2.0, both are required. Previous research has shown that glutamate-dependent AR provides the most efficient protection in complex media at pH 2.0. E. coli O157:H7 has three AR systems that overlap, but each AR system's control and needs for AR activity are different.

Several proteins involved in E. coli O157:H7 AR have been found in addition to these three AR systems. Chaperone HdeA, RNA polymerase-associated protein SspA, and DNA-binding protein Dps are among these proteins. Furthermore, it was discovered that changes in the cell wall membrane or colonic acid production are linked to AR success.

**Colonization of Cattle**

E. coli O157:H7 naturally colonises cattle's gastrointestinal tracts, and the rectoanal junction (RAJ) mucosa, a lymphoid follicle-dense mucosa near the distal rectum, is recognised to be a primary site of colonisation in cattle [39, 48].

There have previously been three unique patterns of E. coli O157:H7 carriage in cattle [14, 39, 58]. For starters, animals can remain culture positive for a few days and are termed passive shedders, meaning they are unlikely to colonise the RAJ mucosa. Second, cattle can be colonised and shed germs for an average of one month, but seldom more than two months.

Finally, a few uncommon animals get colonised for an extended period of time and shed

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germs for 3 to 12 months or longer. This unusual condition, in which a few animals acquire long-term E. coli O157:H7 colonisation (>2 months), is most likely owing to bacterial association at the RAJ mucosa; however, it might also be due to the bacteria colonising a site(s) other than the RAJ mucosa.

Individual cattle's age, food, and immunity may all have an impact on bacterial colonisation. Calves shed E. coli O157:H7 for longer than adult cattle when given the same quantity of E. coli O157:H7 inoculums, according to Cray and Moon [14]. Reducing E. coli O157:H7 carriage in cattle, which is a significant source of E. coli O157:H7 infection, would help to reduce the risk of human infection. Understanding E. coli O157:H7 colonisation factors will be crucial for developing successful methods for minimising or preventing E. coli O157:H7 carriage in cattle.

**Environmental Survival**

E. coli O157:H7 may survive and persist in a variety of settings, including soil, water, food, and animal reservoirs (Fig. 2). E. coli O157:H7 has been shown to persist for a year in manure-treated soil and for 21 months in raw, uncomposted manure [30]. If the temperature is kept above 50°C for 6 days, composting manure is efficient in killing E. coli O157:H7. E. coli O157:H7 may persist in water for a long period, particularly at low temperatures. Water trough sediments polluted with bovine excrement can serve as a long-term (>8 months) reservoir of E. coli O157:H7, and the bacteria that survive in contaminated troughs can cause illness [38]. E. coli O157:H7 lives and replicates in Acanthamoeba polyphaga, according to Barker et al. [2]. A. polyphaga is a widespread environmental protozoan that may be found in soil, water, and faeces. As a result, it might be an effective E. coli O157:H7 transmission vehicle in these conditions. E. coli O157:H7 requires the capacity to adapt to fluctuations or dramatic changes in temperature, pH, and osmolarity that occur in nature in order to thrive in a variety of situations. For example, the generation of exopolysaccharide (EPS) by E. coli O157:H7 is linked to heat and acid tolerance, and heat stress causes changes in lipid content in membranes [77].

These environmental adaptations of E. coli O157:H7 are critical for the microorganism's survival and spread on farms, as well as the increased transmission from cattle to cattle. Furthermore, the pathogen's capacity to live outside of the host reservoir raises the possibility of contamination of crops and products through bovine dung pollution, irrigation with contaminated water, or direct contact with sick animals [42].

**Major Virulence Factors**

Numerous research have focused on defining the virulence factors and processes of E. coli O157:H7 pathogenesis (Fig. 3). Although the generation of Stxs is thought to be important, it is not completely responsible for illness. Furthermore, E. coli O157:H7, which has been linked to severe human disease, must colonise the intestinal mucosa, and the presence of pO157 is linked to the capacity to induce disease. Each of these features is discussed in detail below.

**Shiga Toxins (Stxs)**

Stx is a bacteriophage-encoded cytotoxin with a high potency. Stx is a single transcriptional unit that has been extended to induce harm to a range of cell types [29]. Stxs are separated into two groups: Stx1 and Stx2, although they do not produce cross-reactive antibodies since their amino acid sequences are 56 percent identical. The only difference between Stx1 and Stx from Shigella dysenteriae I is a single amino acid. Stx1 exclusively, Stx2 solely, or both toxins can be expressed by virulent E. coli O157:H7 strains. Stx2 strains are known to be more hazardous than Stx1 strains and are more frequently linked with HC or HUS in human infections [6, 50].

One enzymatically active A subunit (A1) and five identical receptor-binding B subunits make up Stx's structure (B5). The B5 subunit interacts to the globotriaosylceramide (Gb3) or globotetraosylceramide (Gb4) host receptors [47]. The A subunit is internalised to the cytoplasm after Stx (A1B5) binds to the host cell. Al suppresses protein synthesis by removing a single adenine residue from the 60S ribosomal subunit's 28S rRNA [59]. The precise processes of Stx translocation to different tissues are unknown.

**The Locus of Enterocyte Effacement**

Attaching and effacing (A/E) lesions are a kind of histopathological lesion caused by E. coli O157:H7 colonisation of the intestinal mucosa. Microvilli effacement and bacterial adhesion to the epithelial cell membrane are two characteristics of the A/E lesion. Attached bacteria increase the amount of actin polymerization in the host cell, resulting in a higher attachment pedestal [11]. The genes that cause A/E lesions are found in a 13-region called the locus of enterocyte effacement,

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according to genetic research (LEE). The LEE of E. coli O157:H7 is also preserved in EPEC, and the presence of the LEE is well recognised to be related with illness [24]. When compared to EPEC strains, the LEE of E. coli O157:H7 is 43 kb in size and contains an extra 7.5 kb prophage sequence. The function of this extra sequence is unclear. The LEE is made up of at least 41 genes organised into three major regions: (i) a type III secretion system (TTSS) that exports effector molecules; (ii) an adhesion called intimin and its translocated receptor, Tir, which is translocated into the host cell membrane by the TTSS; and (iii) several secreted proteins (Esp) as a part of the TTSS, which are important in the modification of host cell signal transduction during the formation of A/E lesions [15, 52]. Non-LEE encoded effectors have recently been discovered, and elucidating their functions will help us better comprehend the pathogenic events in E. coli O157:H7 infections [16].

**Plasmid O157 (pO157)**

A plasmid is a piece of extrachromosomal DNA that can replicate without the help of chromosomal DNA. Plasmids are mobile components that provide host advantages such as antibiotic and heavy metal resistance, the generation of poisons and other virulence factors, hydrocarbon biotransformations, and symbiotic nitrogen fixation [22]. Many enteropathogenic bacteria, such as Shigella, Yersinia, Salmonella, and E. coli, require plasmid-encoded genes for complete pathogenesis.

**pO157**

pO157 is a highly conserved plasmid found in E. coli O157:H7. The plasmid pO157 is a nonconjugative F-like plasmid with a range of 92 to 104 kb in size. The full sequencing of pO157 has been reported in two separate epidemic isolates [10, 41]. The plasmid pO157 has a dynamic structure and contains a variety of mobile genetic components such as transposons, prophages, insertion sequences (IS), and fragments from other plasmids. The co-responses to functional zones of pO157 can be delimited by the heterogeneous composition of pO157. IS or IS remnants are typically coupled with virulence-related regions, which are comparable to the components of Shigella sppbig .'s virulence plasmid [10, 41]. These findings suggest that the genuine pO157 is generated by the integration of fragments from evolutionarily distinct species origins into an F-like plasmid, and hence the virulence factors or potential virulence factors on the various segments of pO157 may come from various origins. The whole pO157 sequence has 100 open reading frames (ORFs) [10]. 43 ORFs had enough resemblance to known proteins to indicate functions, whereas 22 ORFs had no credible match to any known proteins. Thirty-five proteins are thought to be involved in the pathogenesis of E. coli O157:H7 infections, but only 19 genes have been identified, including a hemolysin (ehxA) [63], a catalase-peroxidase (katP) [9], a type II secretion system apparatus (etp) [62], a serine protease (espP) [8, a putative adhesin (to The biological importance of pO157 in pathogenesis, however, remains unknown.

**Hemolysin (ehx)**

Hemolysin was the first pO157 virulence component to be discovered [4, 61]. Because it possesses a different G+C percent and codon use than the surrounding genetic components, the hemolysin operon (ehxCABD) might be of foreign origin. The genes essential for hemolysin production and transport are encoded by a 3.4-kb fragment, which has been utilised as a diagnostic probe for E. coli O157:H7 and EHEC isolates. Several investigations have found that hemolysin is largely conserved among EHEC serotypes such as O157:H7, O111:H8, and O8:H19, however it is unknown if they have the same biological functions [7].

**Catalase-Peroxidase (katP)**

From pO157, a gene for catalase-peroxidase activity (katP) was discovered [9]. The bacterial bifunctional catalase-peroxidase gene is extremely comparable to this gene, which is 2.2 kb in size. KatP enzyme activity was detected in both the cytoplasm and periplasm fractions of E. coli O157:H7. This enzyme is thought to be transported across the cytoplasmic membrane, based on the N-terminal signal sequence. All E. coli O157:H7 strains have the katP gene, however EPEC, ETEC, EIEC, and EAggEC strains do not. This enzyme may aid E. coli O157:H7 colonisation of human intestines by lowering oxidative stress and using the by-product oxygen in low or no oxygen environments.

**Type II Secretion System (T2SS) (etp)**

The ORFs etpC to etpO encoded by pO157 are very similar to Gram-negative bacteria's T2SS [62]. These genes are near the hemolysin

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gene. The etp and ehx genes were discovered to have an IS911-like insertion element positioned far away. etp genes were detected in all E. coli O157:H7 strains, some non-O157 EHEC strains, but not in EPEC, ETEC, EIEC, or EAggEC strains, similar to the katP gene. Although this T2SS is comparable to Klebsiella oxytoca's pullulanase secretion route (pulO), its function is unknown.

**Serine Protease (espP)**

EspP is a type V secreted serine protease encoded by pO157 that has been shown to cleave pepsin A and human coagulation factor V [8]. PssA in EHEC O26:H-, EspC in EPEC, and IgA1 protease in Neisseria species are examples of secreted or surface-bound proteins that are comparable to this extracellular enzyme [69]. EspP impacts calves' intestinal colonisation and adhesion to bovine primary intestinal epithelial cells, according to Dziva et al. [19]. Furthermore, EspP-mediated degradation of human coagulation factor V might play a role in the mucosal bleeding seen in HC patients.

**Metalloprotease (stcE)**

StcE, a metalloprotease encoded on pO157, cleaves the C1 esterase inhibitor particularly [37]. Multiple proteolytic cascades associated to inflammatory pathways, including as classical complement, intrinsic coagulation, and contact activation, are regulated by the C1 esterase inhibitor. T2SS encoded on pO157 secretes StcE, which is controlled by the LEE-encoded regulator (ler) [20, 37]. StcE can contribute to E. coli O157:H7 adhesion to Hep2 cells in vitro, according to Grys et al. [25]. The stcE gene was detected in all E. coli O157:H7 strains, some EPEC serotype O55:H7 strains, and none of the other diarrheagenic E. coli strains.

**Putative Adhesion (toxB)**

The toxB gene is encoded by a 9.5-kilobyte sequence, and its projected product shares 20% of its amino acid sequence with Clostridium difficile toxin B [41]. ToxB promotes E. coli O157:H7 adhesion to Caco-2 cells by increasing the production of TTSS, according to recent research [67]. Furthermore, a sequence analysis indicated that ToxB has 28% amino acid identity and 47% similarity to the anticipated product of efa-1/lifA, a virulence gene commonly located on the chromosome of EPEC and non-O157 EHEC isolates [46]. ToxB might be implicated in suppressing host lymphocytes because the efa-1/lifA gene has been shown to prevent the activation of human and murine gastrointestinal lymphocytes [36]. In calves and lambs, however, a mutation in the toxB and efa-1 genes had no effect on intestinal colonisation [66].

**Eae Gene-Positive Conserved Fragments (ecf)**

We recently discovered that pO157 encodes the ecf operon (ecf1–4), which is temperature controlled by inherently curved DNA [76]. Both ecf1 and ecf2 are unique to pO157 and encode a putative polysaccharide deacetylase and an LPS-1, 7-N-acetylglucosamine transferase, respectively [32]. ecf3 resembles the putative outermembrane protein in E. coli K1, which has been linked to bacterial invasion [49]. The second copy of a lipid A myristoyl transferase is encoded by ecf4, also known as msbB2 [35, 76]. The lipid A structure and membrane fatty acid composition of the double mutant bearing deletions in the ecf4 and its chromosomal copy lpxM of E. coli O157:H7 were changed, and it demonstrated lower persistence in bovine gastrointestinal tracts [76]. However, when compared to wild-type E. coli O157:H7, a single ecf4 mutation showed no significant change.

**Pathogenesis of pO157**

Following the first revelation that pO157 was essential for the production of fimbriae and epithelial cell adhesion, other research [74] reported inconsistent results on the function of pO157 in epithelial cell adhesion. Animal models such as the mouse, rabbit, and gnobiotic piglet were used in in vivo investigations of pO157, with mixed results. In vivo investigations, on the other hand, have limitations due to the lack of an appropriate animal model that can replicate all elements of the illness. As a result, the exact involvement of pO157 in the pathogenesis of E. coli O157:H7 remains unknown. We recently discovered that the pO157 gene influences the effectiveness of E. coli O157:H7 colonisation and survival in acidic circumstances in healthy cattle [40, 64]. In comparison to wild type, an isogenic pO157 E. coli O157:H7 mutant is more resistant to acidic synthetic bovine gastric fluid and bile [40]. Higher glutamate decarboxylase (GAD) expression is responsible for the pO157 mutant's increased acid resistance. The method by which pO157 regulates gad is unknown, however it is most likely owing to pO157 control of chromosomal genes. The pO157 mutant survives transit through the gastrointestinal tracts of bovines better than wild

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type, but it does not colonise the bovine RAJ mucosa as well as wild type [40, 64].

**pO157-Like Plasmids in EHEC**

Most non-O157 EHEC isolates, but not all human isolates, have large plasmids mimicking pO157, with sizes ranging from 70 to 200 kb [26]. The hemolysin operon (ehx) is generally carried by these plasmids, although the etpC-O, katP, and espP genes are detected in less than half of the isolates [11]. Some of these EHEC–hemolysin plasmids have been linked to adhesion, whereas others have not. The presence of this EHEC–hemolysin plasmid is linked to the development of HUS rather than diarrhoea, according to epidemiological research. A variety of additional plasmids ranging in size from 2 to 87 kb have been found in E. coli O157:H7 isolates in addition to pO157 or EHEC–hemolysin plasmids. However, there has been no link discovered between the presence of any of these plasmids and clinical illness.

**In vitro**

| Year | Target | Pathogenesis | Effect |
|---|---|---|---|
| 1987 | Whole plasmid | Expression of fimbriae<br>Adherence to epithelial cells | Yes |
| 1990 | Whole plasmid | Adherence to epithelial cells | Yes |
| 1993 | Whole plasmid | Production of pilli<br>Adherence to epithelial cells | No |
| 2001 | toxB gene on pO157 | Adherence to epithelial cells | Yes |
| 2005 | stcE gene on pO157 | Adherence to epithelial cells | Yes |
| 2007 | espP gene on pO157 | Adherence to bovine primary intestinal epithelial cells | Yes |

**In vivo**

| Year | Target | Pathogenesis | Effect |
|---|---|---|---|
| 1987 | Whole plasmid | Attaching and effacing lesion in gnotobiotic piglets | No |
| 1990 | Whole plasmid | Colonization of mouse | No |
| 1993 | Whole plasmid | Clinical symptoms in rabbit | No |
| 2006 | Whole plasmid | Colonization of cattle | Yes |
| 2007 | Whole plasmid | Colonization of cattle | Yes |
| 2007 | espP gene on pO157 | Colonization of calves | Yes |

## II. CONCLUSION

This article focuses on the E. coli O157:H7 serotype and its 92-kb plasmid. E. coli O157:H7 is a bacteria that causes serious human illness all over the world. Shiga toxins, products of the pathogenicity island known as the locus of enterocyte effacement, and products of the F-like plasmid pO157 are three key virulence factors. From its quiet reservoir in healthy cattle to the agricultural environment, this virus thrives in a variety of settings. Bacterial adhesion to eukaryotic cells, colonisation of cattle, and acid resistance are all influenced by genes expressed on the pO157. Further research on the aetiology and persistence of E. coli O157:H7 in the environment will lead to more effective treatments to avoid human illness.

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