

Zoonotic *Escherichia coli* infections: Pathogenic variants, mechanism of disease, transmission routes, and foodborne outbreaks

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Abstract

Escherichia coli is a gram-negative, rod-shaped bacterium with diverse pathogenic strains. Most strains of *E. coli* are motile and capable of causing infections in humans, small and large ruminants, and birds. The zoonotic *E. coli* phenotypes include Shiga toxin-producing *E. coli* (STEC), enteropathogenic *E. coli* (EPEC), enterotoxigenic *E. coli* (ETEC), enterohemorrhagic *E. coli* (EHEC), enteroaggregative *E. coli* (EAEC), and uropathogenic *E. coli* (UPEC), all of which pose a significant threat to public health. These pathogenic variants possess unique virulence factors, including Shiga toxins, adhesins, and enterotoxins, which enable *E. coli* to colonize host cells and disrupt their functions. The transmission of these pathogenic agents often occurs through direct contact with the infected animal, consumption of unhygienic dairy products, and environmental exposure to fecal material. Foodborne outbreaks due to zoonotic *E. coli*, specifically STEC O157:H7, are most often associated with undercooked meat, unpasteurized dairy, and contaminated water. This review highlights the major zoonotic pathotypes of *E. coli*, delineates their disease mechanisms and transmission routes from animals to humans, along with a summary of prominent foodborne outbreaks in humans and animals. It further emphasizes the importance of adopting an integrated One Health approach for the prevention and effective control of zoonotic *E. coli* infections, thereby reducing their incidence and public health burden.

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1. Introduction

Escherichia coli (*E. coli*) is a gram-negative bacterium that colonizes the gastrointestinal tract and other mucosal surfaces of various animals (Puvača and de Llanos Frutos 2021). Most *E. coli* strains cause severe diseases of the gastrointestinal tract, but some also cause extra-intestinal infections (Moxley 2022). The dual nature - a potential deadly disease-causing pathogen and a vital commensal bacterium, makes *E.*

coli an intriguing paradox of the human gut microbiota (Hameed et al. 2024). Commensal *E. coli* plays a vital role in gastrointestinal health and is the most ubiquitous facultative anaerobe of the gut (Zhang et al. 2022). It supports the gut microbial balance by competing with harmful bacteria and binding the mucosal membranes without damaging the normal physiological conditions. However, pathogenic strains disrupt the symbiotic balance and cause diseases (Zaada et al. 2019). Interestingly, while these strains cause acute human illness, they often

thrive as asymptomatic commensals in the primary animal reservoirs such as cattle, sheep, and goats, facilitating widespread environmental and foodborne contamination (Peek et al. 2018). Zoonosis, by definition, includes diseases transmissible from animal species to humans, and *E. coli* is one of the leading causes in this group. Zoonotic *E. coli* infections, especially infections triggered by enterohemorrhagic *E. coli* (EHEC) strains such as O157:H7, pose significant risks of animal-to-human transmission (Liu et al. 2023; Nupur et al. 2023). The EHEC strains are long recognized to have a severe adverse impact on the health of humans and animals, often leading to acute diseases like hemorrhagic colitis, diarrhea, and the life-threatening hemolytic uremic syndrome (HUS) (Hwang et al. 2021). Zoonotic *E. coli* infections, particularly STEC infections, are of considerable public health concern because of their profound impact on food safety, the healthcare system, economies, and culture (Majowicz et al. 2014; Ramos et al. 2020).

Based on its virulence and site of infection, *E. coli* is classified into intestinal pathogenic *E. coli* (IPEC) and extraintestinal pathogenic *E. coli* (ExPEC) (Sora et al. 2021). These pathogenic variants have acquired mobile genetic elements, such as plasmids, transposons, and bacteriophages, which encode a wide variety of virulence factors such as toxins and adhesins. These virulence factors enable *E. coli* to cause immune evasion, tissue colonization, and host cell death (Al-Mustapha et al. 2023). IPEC pathotypes, such as enterotoxigenic *E. coli* (ETEC), Shiga toxin-producing *E. coli* (STEC), and enteropathogenic *E. coli* (EPEC), cause various diarrheal diseases, whereas EPEC strains are mainly associated with sepsis, neonatal meningitis, and urinary tract infections (Manges et al. 2019; Cabrera-Sosa and Ochoa 2020; Meena et al. 2021). With nearly 75% of emerging infectious diseases originating from animals, the global incidence of *E. coli* is rising. This emphasizes the urgent need for comprehensive and integrated control to counteract such inter-species pathogen transmission (Chala and Hamde 2021; Rodriguez 2024). Severe loss of economic resources and disease occurrence can be initiated by outbreaks frequently associated with food contamination and result in life-threatening conditions like HUS and hemorrhagic colitis. The acute clinical manifestations of zoonosis, like hemorrhagic colitis and HUS, make them particularly insidious with cattle as most important reservoir and is usually contracted through contaminated food and water (Ramos et al. 2020). The risk of HUS and other long-term complications is disproportionately high in very young, the elderly, and immunocompromised individuals (Christensen et al. 2021).

A very complicated public health problem is the transmission of *E. coli* from the environment to animals and humans. Contaminated water used for drinking and exposure to the environment can transmit *E. coli*, which resides in stool, water, and soil (Laird et al. 2021). Direct contact between people and animals is also vital, particularly in farms. The use of veterinary services and animal manure as compost in horticulture is another condition that could result in transmission (Govers et al. 2024). These dependencies emphasize the need to have a One Health strategy that brings together environmental, animal, and human health programs. Although these modes are extremely important, transmission from human to human poses a very big challenge, especially in dirty, poor environments (Mwafy et al. 2023; Nadi et al. 2024; Denpetkul et al. 2025). Shiga toxin-producing *E. coli* (STEC)-associated foodborne outbreaks across the globe are a public health priority due to the potential to cause severe illness, substantial economic loss, and an erosion of consumer confidence. More than

39,787 cases and 1,343 outbreaks have occurred since 1982 worldwide, including a major outbreak in Germany in 2011 that caused 3,126 illnesses and 46 deaths (Köckerling et al. 2017). Dairy and meat are most often the original sources of contamination and cause 30-31% of European and 35-40% of American infections, respectively. The increased risk was confirmed by studies in which it was identified that 11.1% of animal feed samples were found to be positive for STEC when tested (Venegas-Vargas et al. 2016). Stricter food safety procedures, such as zero-tolerance for STEC in ready-to-eat foods and creating effective interventions such as vaccination in animals, are immensely recommended in a bid to stem this problem. Public health interventions still grapple with the issue of new STEC serotypes arising despite such interventions and heightened public awareness (Mehrdel et al. 2023; Thomas and McAllister 2024).

This review provides a comprehensive and updated overview of zoonotic *E. coli* infections by characterizing major intestinal pathotypes such as EHEC/STEC, EPEC, and ETEC and their clinical outcomes, ranging from simple diarrhea to life-threatening HUS. It will delineate the complex pathways of transmission from animal reservoirs, particularly cattle, through contaminated food, water, and direct contact, while highlighting the emergence of hypervirulent strains and antibiotic resistance. Lastly, the review highlights major foodborne outbreaks to indicate the effect on public health and calls for a "One Health approach" to harmonize the environmental, animal, and human health sciences for an effective surveillance, management, and mitigation of this global threat.

2. Zoonotic *E. coli* pathotypes

As discussed above, *E. coli* constitutes a diverse group of gram-negative bacteria, where some strains are harmless and others cause severe infections affecting humans, animals, and birds (Puvača and de Llanos Frutos 2021; Yu et al. 2021). The disease-causing strains are classified into various pathotypes based on their virulence factors and mechanisms of infection. Some of the important pathotypes of medical and veterinary importance are discussed as follows.

2.1 Enterohemorrhagic *E. coli* (EHEC) or Shiga-toxin producing *E. coli* (STEC)

A highly infectious pathogen whose detrimental effects on the gastrointestinal system are well documented is EHEC, particularly the O157:H7 serotype and non-O157 STEC isolates, such as O26, O111, and O145 (Holzer et al. 2025). Virulence is caused by core virulence determinants such as hemolysin, enteric attachment intimin, and Shiga toxins (stx1, stx2 - of which stx2 is extremely toxigenic) (Pakbin et al. 2021; Ouarroud et al. 2023; Lee et al. 2024). These proteins synergistically cause host cell lysis and enhance disease severity. Clinically, EHEC infection results in profuse diarrhea that can progress to hemorrhagic colitis (Melinte et al. 2024). In vulnerable individuals, it causes HUS, a potentially lethal syndrome characterized by thrombocytopenia, hemolytic anemia, and sudden onset of kidney failure. Though other ruminants such as goats and sheep are also responsible for the perpetuation of EHEC, cattle are the primary asymptomatic reservoir for the disease (Ray and Singh 2022). Effective transmission to humans, primarily through contaminated food or water, is facilitated by the low infectious dose and persistence of the bacteria in the bovine gastrointestinal tract (Edison et al. 2024). Successful control measures call for continued research into the mechanisms of virulence of EHEC and reservoir dynamics, particularly

in the face of the continued emergence of new serotypes with their possible causation of outbreaks (Strzelecki et al. 2025). Cattle, particularly "supershedders" that infect the environment, are the major reservoir of EHEC/STEC in animals. Xenografts, rabbits, and mice are employed by researchers as models of animals to understand infection of these strains. Clinical manifestations of EHEC, e.g., diarrhea and, in others, clinical manifestations which are indistinguishable from human illnesses like hemolytic uremic syndrome (HUS), are studied through these models. Furthermore, Shiga toxins and locus of enterocyte effacement (LEE) enable the bacteria to attach and kill intestinal cells, and these features make the bacteria pathogenic. Though the animal models do not simulate human disease, they do provide information.

2.2 Uropathogenic *E. coli* (UPEC)

It is the most prevalent extraintestinal pathogenic *E. coli* (ExPEC) sub-pathotype, which is primarily implicated in urinary tract infections (Whelan et al. 2023). It causes urinary tract infections in humans, cats, dogs, cattle, and pigs (García et al. 2023; Oh and Park 2025). It possesses certain adhesion genes, such as pyelonephritis-associated pili (PAP) and S fimbria adhesion (SFA), which facilitate robust colonization, toxins such as hemolysin A (hlyA), and efficient iron-acquiring systems such as siderophores (Sroithongkham et al. 2024). They are responsible for various types of diseases, including urethritis, cystitis, pyelonephritis, and urosepsis (Zagaglia et al. 2022). HlyA is a pore-forming exotoxin that induces host cell lysis and iron release, and cytotoxic necrotizing factor (CNF-1) disrupts host cytoskeleton and signaling pathways, which result in inflammation and further tissue damage (Chaoprasid and Dersch 2021; Moxley 2022).

2.3 Avian Pathogenic *E. coli* (APEC)

APEC is a well-recognized cause of colibacillosis in poultry with severe economic implications. This pathotype shares significant virulence factors with UPEC, such as increased serum survival genes (iss), hemolytic fimbriae (hlyF), outer membrane protein (ompA), iron-scavenging siderophores, and temperature-sensitive hemagglutinin (tsh) that facilitate the adhesion, immune evasion, and iron uptake by the pathotype (Kathayat et al. 2021; Hu et al. 2022). APEC is particularly involved in multiple organ damage and causes airsacculitis, pericarditis, perihepatitis, and septicemia, often mediated via virulence and resistance genes carried on colicin V plasmids (Ezzat et al. 2023; Khairullah et al. 2024; Rahayu et al. 2025). These plasmids in APEC not only enhance pathogenicity but also confer multidrug resistance. Its similarity with UPEC and neonatal meningitis *E. coli* (NMEC) raises concern about its zoonotic potential and hence warrants its appropriate diagnosis by bacterial isolation, PCR for virulence genes, and antibiotic sensitivity testing (Nowaczek et al. 2021).

2.4 Other pathotypes of *E. coli*

Septicemia-associated *E. coli* pathotype is strongly linked to potentially fatal septicemia and bacteremia (Al-Quraishi and Jabar 2022). Similarly, NMEC is a significant etiologic agent of neonatal meningitis (Su et al. 2023). Although the virulence determinants responsible for the pathogenicity of NMEC are increasingly well characterized, the intricate patterns of their transmission and the growing risk of antibiotic resistance among zoonotic ExPEC strains continue to pose major challenges to veterinary medicine as well as public health (Mehmood and Ashraf 2023; Edison and Kariyawasam 2025). Compared to Shiga toxin-producing *E. coli* (STEC), the ETEC and EPEC pathotypes are the significant zoonotic variants generally overlooked.

ETEC is an important cause of traveler's diarrhea and childhood diarrhea in endemic areas and is transmitted mainly through contaminated food and water via the oro-fecal route with chicken as a typical vector (Kantele and Lääveri 2022; Thani 2023). EPEC, on the other hand, is classically associated with infantile diarrhea, but atypical EPEC strains have shown broader host distribution, which raises its potential zoonotic concerns (Garcia and Fox 2021; Mare et al. 2021). These strains may contain virulence markers typically associated with other *E. coli* pathotypes, representing an increased likelihood of disease production and interspecies transmission (de Castro et al. 2012).

3. General mechanism of pathogenesis of *E. coli*

Though there is variability in virulence factors among different strains, the general mechanism of pathogenesis of most of the *E. coli* pathotypes consists of several uniform key steps, such as adherence and colonization, immune evasion, toxin production, tissue invasion and damage, nutrient acquisition, and dissemination (da Silva Santos et al. 2015; Baby et al. 2016; Martin et al. 2017; Holmes et al. 2021; Saman et al. 2025). Initially, bacteria bind to the host cells through different adhesions, such as fimbriae or pili, Intimin, and afimbrial adhesins (Kaper and Nataro 2004; Eto et al. 2007; Cirl et al. 2008; Baby et al. 2016). Secondly, bacteria use certain mechanisms to avoid detection by the host immune system, such as capsule, LPS modifications, antigenic variation, and secretion systems (Govindarajan et al. 2020; Gao et al. 2024; Halabitska et al. 2024; Saman et al. 2025). Thirdly, *E. coli* strains produce toxins that are of various kinds depending on the strain's type. The release of exotoxins (stx, LT/ST, HlyA, CNF-1) or effector proteins causes host cell dysfunction, apoptosis, and inflammation. Fourthly, the invasion and tissue damage are mediated directly by some strains, such as EIEC and UPEC, which replicate intracellularly and cause host cell lysis or indirectly by certain strains, such as STEC, which release toxins extracellularly (da Silva Santos et al. 2015). The next step is nutrient acquisition, in which siderophores (enterobactin, aerobactin, and yersiniabactin) extract tightly bound iron for host proteins to facilitate bacterial persistence (Khasheii et al. 2016; Martin et al. 2017). Lastly, bacteria disseminate systemically or locally, causing bacteremia and septic shock due to endotoxin release in some cases (Barber et al. 2016; Ma et al. 2017; Holmes et al. 2021). Some invasive and enterotoxigenic strains have plasmid-determined features that enhance their infectiveness in domestic animals. There are evidences which indicate that certain calves might serve as super-shedders, where they disproportionately infect the environment and spread STEC strains in a disproportionate manner. Also, wild animal studies such as those conducted on mountain brushtail possums indicate that physical and social contact, not proximity, are of greater significance in transmission. The general mechanisms of pathogenesis of *E. coli* is given in Fig. 1. Some of the important pathotypes of *E. coli*, along with their virulence factors and mode of pathogenesis, are given in Table 1.

4. Transmission pathways of zoonotic *E. coli*

Direct animal contact, typical of farm work and environments such as farms and petting zoos, significantly enhances the risk of zoonotic transmission of *E. coli*, especially of STEC (Isler et al. 2021). These environments facilitate human-animal proximity and enhance the possibility of disease transmission through contact or contamination of surfaces. For example, research has confirmed significant frequencies of positive fecal samples for STEC in petting zoo-housed animals, such as sheep, goats, cattle, and camels (Nada et al. 2023). Outbreaks of *E. coli*

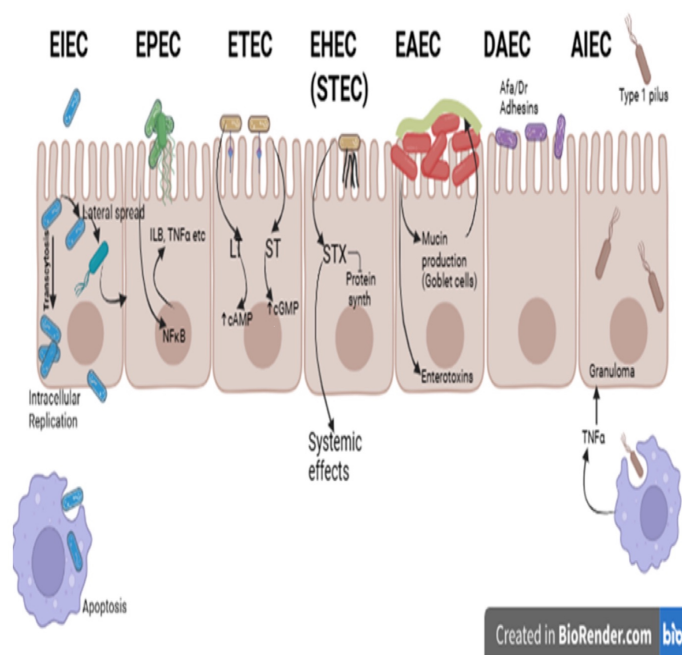


Fig. 1. General mechanism of pathogenesis of *Escherichia coli* pathotypes

EIEC: Enteroinvasive *E. coli*, EPEC: Enteropathogenic *E. coli*, ETEC: Enterotoxigenic *E. coli*, EHEC: Enterohemorrhagic *E. coli*, STEC: Shiga toxin-producing *E. coli*, EAEC: Enteroaggregative *E. coli*, DAEC: Diffusely adherent *E. coli*, AIEC: Adherent invasive *E. coli*

O157 have been documented in petting zoo environments, where genetically identical human and animal strains have demonstrated this direct transmission capacity (Bentancor et al. 2023). The transmission risk is exacerbated by poor hygiene practices, such as inadequate handwashing facilities, a lack of readily available hygiene information, and poor facility layouts (Esposito et al. 2023). Agricultural workers are especially vulnerable due to daily exposure to livestock, contaminated environments, insufficient personal protective equipment, and reduced compliance with biosecurity measures (Gambushe et al. 2022). To minimize contamination and promote better hygiene, redesign of high-contact facilities is required while balancing the educational and recreational benefits of such human-animal interactions (Liguori et al. 2023). A significant and longstanding public health issue is the transmission of several *E. coli* strains, most notably O157:H7, through contaminated meat products (Salman and Steneroden 2023). Though chicken, lamb, and pork are potential threats, undercooked ground beef is the most common carrier because cattle are the primary reservoirs (Willis et al. 2023). During slaughter, intestinal contents may contaminate carcasses, which initiates the infection risk. There have been reports of pig derived products associated with *E. coli* O157:H7 outbreaks, warranting the cautious handling of all types of meat. In addition to the primary contamination, cross-contamination of kitchens worsens the issue of disease outbreaks (da Silva et al. 2024). The pathogens may transfer from raw meat juices to other foods or food preparation surfaces if proper hygiene procedures are not adhered to rigorously (Willis et al. 2023). Therefore, there should be adherence to appropriate food safety practices like proper cooking and careful segregation of raw and cooked foods (Kinanti et al. 2024).

Although meat products are a primary concern, *E. coli* can also be transmitted through other sources like contaminated vegetables and unpasteurized milk or dairy products (Hassani et al. 2023; Sarba et al.

2023). A major public health issue is the foodborne transmission of various strains of *E. coli*, particularly via contaminated products and unpasteurized milk (Wada et al. 2025). Leafy greens, sprouts, and fruits can become contaminated with *E. coli* through contaminated soil, irrigation water, and animal feces (Osafo et al. 2022). The threat is worsened by poor sanitation and the use of contaminated water in agricultural operations (Davidova-Gerzova et al. 2023). Raw milk and farm cheeses can also become contaminated with pathogenic types of *E. coli*, such as STEC, which are generally spread by cow feces (Wada et al. 2025). Zoonotic transmission of *E. coli* and other transmission sources are shown in Fig. 2. Both recreational water sources as well as contaminated drinking water constitute an actual threat to public health via waterborne transmission of *E. coli*, particularly virulent strains such as O157:H7 (Osafo et al. 2022; Lenchenko et al. 2024). Although *E. coli* is sensitive to chlorination, it is still challenging to detect low concentrations of the bacterium in water; hence, vigilant and continuous monitoring needs to be practiced. Epidemiological reports have associated recreational water bodies, such as swimming pools and lakes, with intermittent illness, and these environments are also routinely implicated in outbreaks of *E. coli*, owing to fecal contamination from agricultural runoff, livestock presence, or insufficient wastewater treatment (Osafo et al. 2022). For instance, direct and significant correlation between the presences of *E. coli* within well water and the proximity of the septic tanks have been reported, emphasizing the need for improved sanitation and infrastructure (Paruch and Paruch 2022).

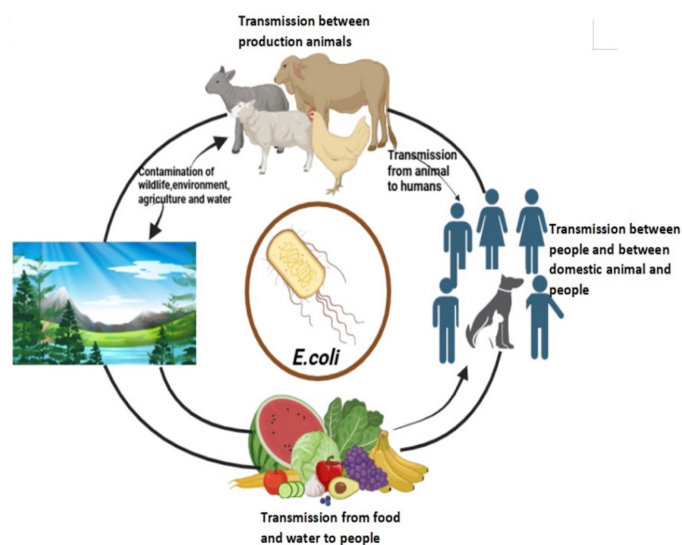


Fig. 2. Zoonotic transmission of *Escherichia coli* and its other potential routes of spread to humans

5. Major foodborne outbreaks in humans

Shiga toxin-producing *Escherichia coli* (STEC) strains (especially O157:H7 and non-O157) cause foodborne outbreaks in humans (Tack et al. 2021). Because of its severity and fatal complications, this strain is the most dangerous. These strains enter humans through contaminated food and water, colonize the intestinal tract, and produce toxins, causing nausea, abdominal cramping, vomiting, and watery to bloody diarrhea (Gambushe et al. 2022; Mansour et al. 2023). Vulnerable groups such as children, the elderly, and immunocompromised individuals are at particular risk of developing HUS (Zhang et al. 2022). In the United

Table 1. Pathotypes of *Escherichia coli* along with their virulence factors and mode of pathogenesis

Pathotype	Virulence factors	Target site	Mode of pathogenesis	Associated diseases	Risk groups	References
Enteropathogenic <i>E. coli</i> (EPEC)	Bundle-forming pilus, intimin	Small intestine	Attack and affect lesions	Infantile diarrhea	Infants, lambs, cats, and dogs	(Lee et al. 2022; Ryandini et al. 2024)
Enterotoxigenic <i>E. coli</i> (ETEC)	Heat-labile / stable toxins	Small intestine	Toxin-mediated fluid secretions	Traveler's diarrhea and watery diarrhea	Traveler's children, calves, and piglets	(Zhang et al. 2022; Hassan et al. 2023)
Enterohemorrhagic <i>E. coli</i> (EHEC)	Shiga toxins (stx1, stx2), intimin	Colon	Cytotoxicity and inflammation	Hemorrhagic colitis and hemolytic uremic syndrome	All ages, especially children, cattle and small ruminants	(Sun et al. 2022)
Enteroinvasive <i>E. coli</i> (EIEC)	Invasion plasmid antigens proteins	Colon	Invasion of epithelial cells	Dysentery-like illness	Humans and all age groups	(Ghosh et al. 2025)
Enteraggregative <i>E. coli</i> (EAEC)	Aggrevate adherence fimbria (AAF), enteroaggregative heat-stable enterotoxin (EHSE)	Small intestine	Biofilm formation and persistent colonization	Persistent diarrhea	Children and immunocompromised patients, dogs, and poultry	(Izquierdo-Vega et al. 2023)
Diffusely adherent <i>E. coli</i> (DAEC)	Afimbrial and Dr family adhesins	Small intestine	Diffuse adherence	Watery diarrhea	Fewer immune patients	(Hosseinasab et al. 2024)
Uropathogenic <i>E. coli</i> (UPEC)	P fimbriae, hemolysin, siderophores	Urinary tract	Ascending infection and inflammation	Urinary tract infections (UTIs)	Female catheterized patients, humans, cats, dogs, cattle	(Whelan et al. 2023)
Neonatal meningitic <i>E. coli</i> (NMEC)	K1 polysaccharide capsule, invasion of brain epithelium proteins (ibe)	Meninges	Blood brain barrier invasion	Neonatal meningitis	Neonates, calves, lambs, piglets	(Liu et al. 2021)
Adherent invasive <i>E. coli</i> (AIEC)	Adherence and invasion factors	Ileum and colon	Intracellular replication	Crohn's disease	Children with IBD	(Mansour et al. 2023)
Shiga toxin-producing <i>E. coli</i> (STEC)	Shiga toxins (stx1 and stx2)	Colon	Toxin-mediated endothelial damage	Hemorrhagic colitis and hemolytic uremic syndrome	All ages groups	(Freedman et al. 2023)
Hybrid strain (e.g., EAHEC)	Shiga toxins, Aggrevate adherence fimbria (AAF), intimin	Colon	EHEC and EAEC mechanisms	hemolytic uremic syndrome (HUS) outbreaks	All ages	(Gomes et al. 2023)
Avian pathogenic <i>E. coli</i> (APEC)	Increased serum survival (iss), colicin V, and iron uptake system	Respiratory and systemic	Septicemia and respiratory infections	Colibacillosis in poultry	Poultry, zoonotic potential	(Joseph et al. 2023)
Animal enterotoxigenic <i>E. coli</i> (aETEC)	F4, F5, F6 fimbriae, heat-labile and heat-stable toxins	Small intestine	Toxin-mediated fluid loss	Neonatal diarrhea in piglets and calves	Livestock neonates	(Von Mentzer and Svennerholm 2024)
Mastitis-associated <i>E. coli</i> (MAEC)	Variable adhesins, iron uptake systems	Mammary gland	Inflammation and colonization	Mastitis in dairy cattle	Dairy cattle	(Meçaj et al. 2023; Ahmed and Nawaz, 2024; Germon et al. 2025)
Septicemic <i>E. coli</i> (SEPEC)	Cytotoxins and serum resistance genes	Blood stream	Bacteremia and septicemia	Sepsis in animals and humans	Immunocompromised individuals	(Karbalaei et al. 2025)
Multidrug resistant <i>E. coli</i> (MDREC)	Extended spectrum beta lactamases (ESBL), carbapenemases,	Various organs	Resistance to antibiotics	Nosocomial infections and resistant urinary tract infections	Hospitalized and immunocompromised individuals	(Rozwadowski and Gawel 2022)
Zoonotic <i>E. coli</i> O157:H7 (ZEC)	Shiga toxins, intimin	Colon	Epithelial destruction and Shiga toxicity	Foodborne outbreaks and hemolytic uremic syndrome	The general population is exposed through contaminated sources	(Frag et al. 2023; Riaz et al. 2023)

States from 2010 to 2017, the health department recorded 466 STEC outbreaks, of which 44% of O157 and 41% of non-O157 outbreaks were observed (Tack et al. 2021). In Germany in 2011, an outbreak of O104:H4 caused 50 deaths, and almost 3,000 cases were seen (Bloch et al. 2012). Another outbreak in the USA affecting 1727 individuals with 144 hospitalizations was linked to non-O157 STEC, including serotypes STEC O111 and O11 of STEC O26, which are mostly associated with dairy products (Luna-Gierke et al. 2014). According to a study in Alberta (Canada) from 2018-2021, 729 overall isolates of non-O157

STEC were identified clinically, from which 42.4% cases were of young people (18 years or less) and 31.1% cases of children (0 to 9 years) (Glassman et al. 2022).

6. Major feedborne outbreaks in animals

E. coli is the main cause of systemic and enteric illness in immunocompromised or young animals, and the most commonly prevalent pathotype is enterotoxigenic *E. coli* (ETEC), a major cause of neonatal diarrhea in piglets and calves (Islam et al. 2023). It results in

dehydration, excessive fluid loss, and death if left untreated. While in poultry, the most prevalent strain is avian pathogenic *E. coli* (APEC), which mostly disturbs internal organs and the respiratory system with predominant signs of perihepatitis, airsacculitis, septicemia, and pericarditis in affected birds (Kromann et al. 2021). STEC has also been identified in animal-origin foods, with prevalence rates in swine ranging from 4.4–68.3% (live swine), 22–86.3% (in slaughtered pigs), and 0.10–80% (retail pig meat) (Haque et al. 2022). Moreover, in retail pig meat, O157:H7 serotype with different virulence gene combinations have been reported from Africa (O157 + stx2 + eae + ehx), the United States (O157 + Shiga toxin gene/stx), and Asia (O157 + Shiga toxin 1 gene/stx1 + Shiga toxin 2 gene/stx2 + enterohemolysin A gene/ehx). While non-O157 serotypes in the U.S. (non-O157 + stx + intimin gene/eae) and unknown serogroups + (stx + eae, stx2 + eae, or stx1 + stx2 + eae) were identified in England (Haque et al. 2022). In Iran from 2008–2016, 75 strains of non-O157 STEC serotypes were identified in humans and animals. O113 serotype was found in cattle (55.5%), goat (22.2%), and red deer (22.2%), whereas, O26, O111, O5, O63, O75, and O128 were isolated from cattle (100%), sheep (100%), pigeon (100%), and from goats (66.6%) and pigeon (33.3%), respectively (Badouei et al. 2023). The prevalence of the top five STEC serotypes in slaughter cattle ranged from 1.0% to 4.5% depending on breed and production system (Badouei et al. 2023). These findings underline the dual relevance of *E. coli* outbreaks for both animal production economics and zoonotic transmission potential.

One health approach is essential to control zoonotic *E. coli* because the bacteria circulate between humans, animals, birds, food, and the environment (Shaheen 2022). In humans, *E. coli* can be controlled by proper hygiene, chlorination of wastewater, avoiding undercooked dairy products, and unpasteurized milk (Slattery and Garvey 2025). In animals, better farm management practices decrease *E. coli* shedding in feces (Kannan et al. 2021). Other than this, vaccination and probiotics can also limit their gut colonization, particularly in poultry birds (Watts and Wigley 2024). In the food chain, better slaughterhouse hygiene, proper pasteurization, and washing of vegetables with clean water are very critical. These approaches can minimize the intensity of *E. coli* infections, but complete removal is quite difficult due to their widespread occurrence as both commensal and pathogenic bacteria.

7. Conclusion

As a one welfare problem of concern, because of its far-reaching implications in food safety, animal welfare, and public health, zoonotic *E. coli* is of serious concern. Acute gastrointestinal and extraintestinal diseases are caused by pathogenic species such as EHEC, EPEC, ETEC, and EAEC. As such, pathogens possess animal reservoirs like cattle and poultry, and are contagious from the reservoirs to human beings or through contaminated food and water, making them the most risky. O157:H7 and other foodborne Shiga toxin-producing *E. coli* (STEC) emphasize the need for additional food safety practices and sanitizing procedures. Treatment is further worsened by increasing antimicrobial resistance of the strains, which points to the significance of using antibiotics responsibly in animal as well as human healthcare. Measures in the future must focus on coming up with new vaccinations for cattle in a bid to stem the spread of the infection at the source and on embarking on improved genomic surveillance in keeping track of emerging strains of *E. coli* and its pattern of resistance. Predictive models based on Artificial Intelligence can also be used to forecast outbreaks, allowing for more proactive and focused responses.

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