Saudi Journal of Medicine and Public Health

https://saudijmph.com/index.php/pub

https://doi.org/10.64483/202522505

Epidemiology and Public Health of *Escherichia coli* Infections-An Updated Review

Yasser Yahya Abduh Wasili [(1)] , Abdulrahman Ali Yahya Bakri [(2)] , Abdullah Soidei Ali Alkyadi [(2)] , Ahmed Abduh Ahmed Hamzi [(3)] , Jabril Yousef Ahmed Abdali [(3)] , Abdullah Saleh Essa Aldurayhim [(4)] , Abdulrhman Mohammed Abdulrhman Bin talah [(5)] , Mohammed Omar Hakami [(6)] , Hafiz Mohammed Ali Saghayir [(7)] , Faruq Omar Hakami [(7)] , Abdulaziz Abdulmajeed Almalki [(8)]

(1) Ministry of Health - Al-Hasa health Cluster, Omran General Hospital, Saudi Arabia,
(2) Heraa General Hospital, Ministry of Health, Saudi Arabia,
(3) KSA, Ministry Of Health Alaradh Primary Health Care Center, Saudi Arabia,
(4) public health department/Riyadh First health Cluster, Saudi Arabia,
(5) Public Health Department at the First Health Cluster in Riyadh, Saudi Arabia,
(6) Erada Hospital for Mental Health in Jazan, Ministry of Health, Saudi Arabia,
(7) King Abdullah Airport health monitoring Center, Ministry of Health, Saudi Arabia,
(8) Al Iman general hospital, Ministry of Health, Saudi Arabia

Abstract

Background: *Escherichia coli* is a gram-negative bacterium that constitutes part of the normal human intestinal microbiota but is also one of the most important causes of intestinal and extraintestinal infections worldwide. Pathogenic strains are responsible for a wide clinical spectrum ranging from self-limiting diarrhea to life-threatening conditions such as sepsis and hemolytic uremic syndrome, representing a significant public health concern.

Aim: This review aimed to provide an updated overview of the epidemiology, pathogenic mechanisms, clinical features, diagnostic approaches, and management strategies related to *E. coli* infections, with emphasis on implications for public health and healthcare systems.

Methods: A narrative review approach was applied, integrating current evidence on *E. coli* pathotypes, modes of transmission, disease burden, diagnostic modalities, treatment options, and prevention strategies, including infection control and antimicrobial stewardship.

Results: Intestinal *E. coli* infections are categorized into major pathotypes—ETEC, EPEC, EAEC, EHEC/STEC, and EIEC—each characterized by distinct virulence factors and epidemiological patterns. Extraintestinal infections, particularly urinary tract infections and bacteremia, account for a substantial burden in healthcare settings. Advances in molecular diagnostics have improved pathogen identification, while rising antimicrobial resistance, including ESBL- and carbapenemase-producing strains, has complicated treatment decisions.

Conclusion: *E. coli* infections remain a major cause of morbidity worldwide. Early recognition, accurate diagnosis, rational antimicrobial use, and strong preventive strategies are essential to reduce disease burden and limit the spread of resistant strains.

Key words: *Escherichia coli*; Epidemiology; Pathogenesis; Antimicrobial resistance; Public health..

Introduction

Escherichia coli, a gram-negative, facultative anaerobic bacillus, forms a natural component of the human intestinal microbiota and is also ubiquitously present in environmental reservoirs, including water, soil, and food sources [1]. While most strains exist as commensals, several pathogenic variants possess virulence factors that enable colonization, toxin production, and evasion of host immune responses, resulting in a wide spectrum of clinical manifestations. These infections range from self-limiting gastrointestinal disturbances to severe systemic complications, including hemolytic uremic syndrome, septic shock, and multi-organ failure, highlighting the pathogen's clinical significance. The growing prevalence of antimicrobial resistance among E coli strains further exacerbates the challenge of effective treatment, positioning E coli as a major contributor to both community-acquired and healthcare-associated infections. Clinically, E coli infections are categorized into intestinal and extraintestinal forms. Intestinal pathogenic E coli strains are classified based on distinct pathogenic mechanisms, including enterotoxigenic E coli (ETEC), responsible for traveler's diarrhea; enterohemorrhagic or Shiga toxin-producing E coli (EHEC/STEC), associated with hemorrhagic colitis and renal injury; enteropathogenic E coli (EPEC),

*Corresponding author e-mail: yywasili@moh.gov.sa (Yasser Yahya Abduh Wasili).
Receive Date: 04 December 2025, Revise Date: 27 December 2025, Accept Date: 31 December 2025

linked to infantile diarrhea; enteroinvasive E coli (EIEC), which mimics Shigella infections; and enteroaggregative E coli (EAEC), causing persistent diarrhea, particularly in children [2]. Extraintestinal infections primarily involve the urinary tract, bloodstream, lungs, and peritoneal cavity, producing urinary tract infections, bacteremia, pneumonia, and peritonitis, often in patients with underlying comorbidities. Distinguishing these subtypes and understanding their pathophysiology is critical for timely diagnosis, the selection of appropriate therapeutic strategies, and the implementation of preventive measures aimed at mitigating morbidity and mortality associated with E coli infections [1][2].

Etiology:

Escherichia coli exists as a normal component of the human intestinal microbiota, where it functions as a commensal organism without causing disease [3][4]. It is the most prevalent gram-negative bacterium in the gastrointestinal tract, contributing to nutrient metabolism and maintaining microbial balance. Outside the intestinal environment, however, E coli can act as an opportunistic pathogen, causing a variety of infections. Its pathogenic potential is evident in urinary tract infections (UTIs), bacteremia, pneumonia, and peritonitis, particularly in immunocompromised patients or those with indwelling medical devices [3][5]. E coli is also a leading cause of healthcare-associated infections. Catheter-associated UTIs and ventilator-associated pneumonia represent major nosocomial complications linked to this bacterium, highlighting its clinical importance in hospital and long-term care settings [6]. Environmental reservoirs further facilitate its transmission. The bacterium is present in soil, contaminated water, raw or undercooked meat, and fresh produce, making ingestion a common route for intestinal pathogenic strains. Specific pathogenic variants, including enterotoxigenic, enterohemorrhagic, enteropathogenic, enteroinvasive, and enteroaggregative E coli, acquire virulence factors that enable colonization, toxin production, and mucosal invasion, resulting in gastrointestinal disease. Understanding the dual role of E coli as a commensal and pathogen is essential for implementing preventive measures and guiding effective clinical management.

Epidemiology

Escherichia coli is a versatile pathogen capable of causing disease both within and outside the intestinal tract. Its diverse pathogenic potential is categorized based on serotypic differences, primarily defined by the O and H antigens. The O antigen corresponds to the repeating polysaccharide units of the bacterium's lipopolysaccharide outer membrane, whereas the H antigen is determined by the structure of its flagellum [2]. These antigenic variations are critical for distinguishing the multiple pathotypes of E coli and understanding their epidemiological patterns. Intestinal infections caused by E coli are classified into five principal pathotypes, each exhibiting distinct mechanisms of pathogenicity, reservoirs, and transmission dynamics. Enterotoxigenic E coli (ETEC) is a leading cause of watery diarrhea in regions with limited sanitation. Transmission occurs primarily through contaminated food and water, and a relatively high infectious dose—approximately 100 million organisms—is required to produce disease in healthy individuals. ETEC is the predominant cause of traveler's diarrhea worldwide and contributes significantly to dehydrating diarrheal illness in infants and children in resource-constrained settings [7]. Enteropathogenic E coli (EPEC) was the first pathotype identified in association with infantile watery diarrhea, particularly in low-resource regions. It is implicated in both sporadic cases and epidemic outbreaks. Transmission primarily occurs via ingestion of contaminated materials, although direct person-to-person spread has also been documented [8][9]. Enteroaggregative E coli (EAEC) has been increasingly recognized as a cause of both acute and chronic diarrhea across diverse socioeconomic contexts, including high-income countries, and is also implicated in travel-associated diarrheal illness [10][11].

Enterohemorrhagic or Shiga toxin-producing E coli (EHEC/STEC) represents a highly virulent pathotype capable of producing Shiga toxins, with serotype O157:H7 being among the most well-characterized. EHEC/STEC infections frequently result from the consumption of contaminated produce, undercooked beef, and raw dairy products. The infectious dose for EHEC/STEC is relatively low, facilitating rapid environmental and interhuman transmission [12][13][14][15][16]. While EHEC/STEC infections can affect all age groups, children under five years of age are most susceptible to the development of hemolytic uremic syndrome (HUS) [17]. Enteroinvasive E coli (EIEC), closely related to Shigella species, is less commonly encountered in clinical practice, partly due to the high infectious dose required. It is primarily transmitted via undercooked meats and contaminated vegetables, though recent data suggest that EIEC infections may be underdiagnosed [18]. Extraintestinal E coli infections arise when the bacterium translocates from its intestinal reservoir or is introduced into sterile environments, particularly within healthcare settings. E coli is the leading cause of extraintestinal gram-negative infections, including urinary tract infections, abdominal and pelvic infections, pneumonia, bacteremia, and neonatal meningitis. Surveillance data from the United States between 2009 and 2016 identified 71,909 extraintestinal E coli infections in hospitalized patients, with urinary tract infections representing 66% of these cases. Approximately half of all cases of E coli bacteremia originate from genitourinary sources [19][20]. These data highlight

the dual burden of E coli as both an intestinal and extraintestinal pathogen and underscore its significant impact on public health, particularly in healthcare-associated environments where transmission and infection control remain critical concerns. Understanding the epidemiology of E coli, including its pathotype-specific transmission patterns and clinical implications, is essential for effective surveillance, prevention strategies, and the management of both community-acquired and nosocomial infections.

Pathophysiology

The pathophysiology of Escherichia coli infections is multifaceted, reflecting the organism's ability to adapt to diverse host environments and overcome host defenses. E coli is a gram-negative bacillus with a characteristic cell envelope composed of an inner cytoplasmic membrane, a rigid peptidoglycan cell wall, and an outer membrane that contains lipopolysaccharide (LPS) and associated proteins. The LPS component is highly immunogenic and can trigger systemic inflammatory responses if released during bacterial lysis. Pathogenic E coli strains possess a range of virulence factors encoded on mobile genetic elements, including plasmids, transposons, and bacteriophages, which facilitate colonization, immune evasion, and toxin production [21]. The pathophysiology differs substantially between intestinal and extraintestinal infections, largely depending on the bacterial pathotype involved and the site of infection. In intestinal infections, enterotoxigenic E coli (ETEC) mediates disease primarily through secretory toxins. Colonizing fimbriae allow the bacteria to adhere to the small intestinal mucosa, where they secrete either a heat-labile (LT) or heat-stable (ST) toxin. LT activates adenylate cyclase, elevating intracellular cyclic adenosine monophosphate (cAMP) in enterocytes, which stimulates chloride secretion and inhibits sodium chloride absorption. ST, by contrast, activates guanylate cyclase, increasing cyclic guanosine monophosphate (cGMP), leading to similar electrolyte imbalances. The net effect of both toxins is the movement of water into the intestinal lumen, producing the characteristic watery diarrhea associated with ETEC infection [22][23].

Enteropathogenic E coli (EPEC) employs a distinct mechanism involving the localized adherence to enterocytes mediated by a bundle-forming pilus encoded on the pEAF plasmid. The outer membrane protein intimin, encoded on the eae gene within the locus of enterocyte effacement (LEE) chromosomal island, strengthens adherence. The LEE encodes approximately 20 effector proteins secreted via a type III secretion system, which alter cytoskeletal architecture, disrupt tight junctions, and efface microvilli, resulting in impaired absorption and secretory diarrhea. Additional effectors, including EspF, EspG, and EspG2, compromise intestinal barrier integrity by disrupting endocytic pathways and chloride absorption, further contributing to diarrheal pathophysiology [24][25][26][27]. Enteroaggregative E coli (EAEC) is distinguished by its "stacked-brick" pattern of adherence to intestinal epithelium. The transcriptional activator AggR, encoded on a virulence plasmid, orchestrates the expression of aggregative adherence fimbriae, dispersin, and multiple enterotoxins including Pet, EAST-1, ShET1, and ShET2. These factors facilitate persistent colonization, epithelial damage, and both acute and chronic watery diarrhea [28][29][30]. Enterohemorrhagic E coli or Shiga toxin-producing E coli (EHEC/STEC) causes disease through Shiga toxins Stx1 and Stx2, which inhibit protein synthesis in enterocytes, triggering cell death, mucosal inflammation, and hemorrhagic colitis. Low infectious doses allow efficient transmission through contaminated food or water. EHEC/STEC infections are associated with hemolytic uremic syndrome (HUS), characterized by microangiopathic hemolytic anemia, thrombocytopenia, and acute renal failure, particularly in children under five years [31][32][33][34][35].

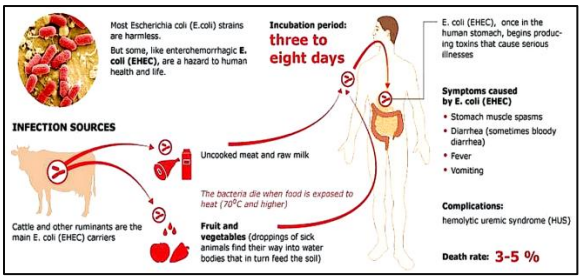

Fig. 1: E. Coli Infection.

Enteroinvasive E coli (EIEC) produces inflammatory diarrhea through mechanisms similar to Shigella, involving colonic invasion, intracellular replication, and cell-to-cell spread. The resultant mucosal destruction produces inflammatory colitis, sometimes with bloody diarrhea [18][28]. Extraintestinal infections result from the translocation of commensal or environmental E coli to normally sterile sites. The urinary tract is the most frequent site, with ascending infection leading to cystitis and pyelonephritis, particularly in women due to urethral anatomy. E coli is also implicated in ventilator-associated pneumonia, often resulting from aspiration, and less commonly causes severe community-acquired pneumonia. Bacteremia typically arises secondary to a primary infection such as a urinary tract infection or intra-abdominal focus [36][37][38][39][40][41][42]. The diverse virulence strategies of E coli, including adherence mechanisms, toxin production, and immune modulation, underlie its capacity to cause a spectrum of intestinal and extraintestinal diseases, making it a significant pathogen in both community and healthcare settings.

History and Physical

A comprehensive clinical history is central to establishing a diagnosis of Escherichia coli infection. Detailed inquiry into symptom onset, duration, severity, and progression provides critical information for distinguishing E coli from other enteric pathogens. Clinicians should assess for aggravating or alleviating factors, including any prior use of over-the-counter medications, which may influence symptom presentation. A key diagnostic consideration is differentiating between watery and bloody diarrhea, as this can narrow the differential toward specific E coli pathotypes. Travel history, recent dietary exposures, and ingestion of potentially contaminated food or water are particularly relevant, as enterotoxigenic E coli (ETEC) is a leading cause of traveler's diarrhea in resource-rich regions and requires a high index of clinical suspicion. Onset of symptoms in E coli infections typically occurs more than sixteen hours after exposure, which may help differentiate it from other bacterial or viral causes of diarrheal illness. For extraintestinal manifestations, clinicians must evaluate prior infections and assess for risk factors associated with antimicrobial resistance. In cases of urinary tract involvement, detailed questioning should address the presence of indwelling devices, such as Foley catheters or ureteral stents, which increase the risk of complicated infection. Additionally, underlying comorbidities, immunosuppressive therapy, and recent hospitalizations are relevant in predicting severity and guiding empirical antimicrobial therapy [40][41][42].

Physical examination complements the history by assessing the severity and systemic impact of infection. Vital signs should be carefully recorded, with particular attention to indicators of systemic illness, such as fever, tachycardia, hypotension, and altered mental status, which may necessitate hospital-based management. Clinicians should evaluate hydration status by examining skin turgor, mucous membranes, and capillary refill. Cardiorespiratory assessment, including auscultation of heart and lungs, is essential, particularly in patients with sepsis or bacteremia. Focused examination should correlate with the patient’s reported symptoms. Abdominal assessment, including inspection, palpation, percussion, and auscultation, is critical for patients with gastrointestinal manifestations. Genitourinary examination should be performed when urinary symptoms are present, assessing suprapubic tenderness or costovertebral angle tenderness indicative of pyelonephritis. In patients presenting with systemic signs, a complete head-to-toe evaluation is warranted to identify potential sources of sepsis and complications of E coli bacteremia. Overall, an integrated approach combining meticulous history-taking with thorough physical examination enables accurate diagnosis, risk stratification, and tailored management for patients affected by E coli infection [40][41][42].

Evaluation

The evaluation of patients with suspected Escherichia coli infection relies on a combination of clinical assessment, laboratory testing, and microbiologic analysis, tailored to the severity of illness and risk of systemic complications. In patients presenting with mild, self-limited diarrheal illness who are otherwise well-appearing, routine laboratory studies are generally not required, as the disease often resolves spontaneously. However, laboratory evaluation becomes essential in patients demonstrating systemic symptoms, prolonged diarrhea, dysentery, or other concerning features suggestive of invasive disease, particularly in the context of EHEC/STEC infection, which carries a risk of hemolytic uremic syndrome and other severe complications. In these scenarios, initial evaluation should include a complete blood count and a basic metabolic panel to assess electrolyte imbalances, hemoconcentration, and early indicators of renal compromise. Stool cultures should be obtained to identify pathogenic E coli subtypes in patients with persistent or severe gastrointestinal symptoms. Given the inability to distinguish E coli pathotypes based solely on appearance, biochemical or molecular testing is required for definitive identification [44]. Traditionally, E coli identification relied on selective culture techniques. These organisms are non-spore-forming, facultatively anaerobic, flagellated gram-negative bacilli that ferment lactose and produce indole. MacConkey agar has been the mainstay medium, facilitating differentiation based on lactose fermentation. Indole testing further supports E coli identification. EHEC/STEC strains, particularly O157:H7, can be distinguished using sorbitol-MacConkey agar, as this serotype typically does not ferment sorbitol, though non-O157:H7 strains with variable sorbitol fermentation have been increasingly recognized. Molecular assays, including polymerase chain reaction (PCR) and nucleic acid amplification tests (NAAT), have enhanced detection accuracy, allowing differentiation of pathotypes through virulence gene identification. For instance, ETEC is detected via heat-labile or heat-stable toxin genes, EPEC through the pEAF plasmid encoding bundle-forming pili, EAEC via the AggR regulon, EHEC/STEC through Stx1 and Stx2 genes, and EIEC by the lacY gene [46].

Extraintestinal infections require targeted culture of blood, urine, or respiratory specimens, depending on the clinical presentation. Identification of E coli in these specimens informs appropriate antimicrobial therapy and may necessitate susceptibility testing due to the increasing prevalence of antibiotic-resistant strains. Extended-spectrum beta-lactamase (ESBL) producing E coli are resistant to many beta-lactam antibiotics, including cephalosporins and monobactams, whereas carbapenemase-producing strains confer resistance to carbapenems such as imipenem, ertapenem, and

meropenem. Additionally, E coli may harbor resistance determinants against fluoroquinolones, aminoglycosides, and other commonly used antimicrobial classes [50]. Accurate identification and susceptibility profiling are therefore crucial to guide evidence-based management, particularly in healthcare-associated infections and high-risk patient populations. In conclusion, evaluation of E coli infection integrates clinical assessment with targeted laboratory and microbiologic investigations, with molecular diagnostics increasingly used for pathotyping differentiation. Extraintestinal infections necessitate culture and susceptibility testing to guide antimicrobial therapy in the context of emerging resistance. Comprehensive evaluation ensures appropriate treatment, mitigates complications, and informs public health surveillance strategies.

Treatment / Management

Management of Escherichia coli infections depends on the strain involved and the clinical presentation, with approaches tailored to intestinal versus extraintestinal disease. For patients with intestinal infections, particularly those with mild diarrheal illness, the cornerstone of therapy is supportive care, focusing on hydration and symptom control [44][51]. Oral rehydration remains first-line therapy, providing adequate fluid and electrolyte replacement in most patients. Intravenous fluids are indicated in cases where oral intake is insufficient or impractical, such as in severe dehydration or persistent vomiting. Symptom management may include antimotility agents such as loperamide or bismuth subsalicylate to alleviate distressing diarrhea, particularly in adults, although these agents are contraindicated in children with infectious diarrhea or in patients with suspected EHEC/STEC infection due to the increased risk of hemolytic uremic syndrome (HUS). Antibiotic therapy is generally reserved for patients with severe disease, characterized by high stool output, prolonged diarrhea, fever, dehydration requiring hospitalization, or dysentery. Recommended agents include fluoroquinolones, azithromycin, and rifaximin, with dosing regimens adjusted according to age and infection severity. Antibiotics are not recommended for EHEC/STEC infections, especially in children and older adults, because antimicrobial use may precipitate HUS. In all patients, supportive care and hydration remain the principal interventions, and antimotility agents are avoided in those with inflammatory or bloody diarrhea [44][51].

Hospitalization is indicated for patients with EHEC/STEC infection, particularly children under 12 years, to allow for close monitoring and to mitigate community spread [52]. Intravenous isotonic fluids such as 0.9% saline or Lactated Ringer's solution are recommended. Careful avoidance of nephrotoxic medications, including NSAIDs, is essential to prevent worsening renal function. In patients who develop HUS, hemolytic anemia and thrombocytopenia may require transfusions, though early red cell transfusions are reserved for hemodynamically unstable patients, and platelet transfusions are limited to severe thrombocytopenia or active bleeding to reduce thrombotic risk [53][54]. Extraintestinal E coli infections, such as urinary tract infections, bacteremia, or pneumonia, necessitate pathogen-directed antimicrobial therapy guided by local susceptibility patterns and antibiograms. Commonly used antibiotics include beta-lactams (cephalosporins, monobactams, carbapenems), fluoroquinolones, nitrofurantoin, trimethoprim-sulfamethoxazole, and fosfomycin. For multidrug-resistant strains, such as ESBL-producing E coli, therapy may require fourth-generation cephalosporins or carbapenems. Infections caused by carbapenemase-producing strains may necessitate combination therapy with agents such as ceftazidime-avibactam, colistin, or polymyxin B [55][56]. Selection of oral versus intravenous therapy is guided by the severity of illness, site of infection, and patient comorbidities, ensuring both adequate drug exposure and optimization of clinical outcomes. Overall, the management of E coli infections requires a nuanced, pathogen-specific approach, balancing supportive care, judicious antibiotic use, and close monitoring for complications, particularly in high-risk populations. Adherence to local resistance patterns, avoidance of nephrotoxic agents, and appropriate hospitalization are critical components of effective care.

Differential Diagnosis

The clinical presentation of intestinal illness is nonspecific, necessitating a comprehensive differential diagnosis to identify the causative pathogen accurately. Watery diarrheal illness is most frequently attributed to viral agents, notably norovirus and rotavirus, which predominate in both sporadic cases and outbreaks, particularly among children and immunocompromised populations [57]. However, bacterial pathogens can also produce a similar clinical picture. Staphylococcus aureus, often associated with foodborne intoxication, Bacillus cereus, and Vibrio cholerae must be considered in patients presenting with profuse, watery diarrhea, especially when epidemiologic clues such as recent food exposure or travel history are present. In cases where inflammatory or bloody diarrhea is observed, the differential broadens to include bacterial enteropathogens such as Shigella species, Salmonella species, Campylobacter jejuni, and Yersinia enterocolitica. Each of these pathogens has unique epidemiological and clinical features that can aid in distinguishing them from Escherichia coli infections [57]. Extraintestinal infections require additional consideration, as the pathogenic spectrum varies widely depending on the affected organ system. For urinary tract infections, differential diagnosis should

include Klebsiella species, Proteus species, Enterococcus species, and Pseudomonas aeruginosa. In cases of bacteremia, sepsis, or pneumonia, Staphylococcus aureus, Streptococcus pneumoniae, and other Gram-negative bacilli must be considered. Recognition of risk factors, such as indwelling catheters, recent hospitalization, or immunocompromised states, is critical for narrowing the differential and guiding empiric therapy. Accurate pathogen identification often requires a combination of stool culture, molecular assays, and clinical judgment, as overlapping clinical manifestations can obscure the etiology. In addition, patient history including dietary intake, travel, comorbidities, and prior antibiotic exposure provides essential context to differentiate E coli from other viral, bacterial, or mixed infections [57].

Prognosis

The overall prognosis for E coli infections is generally favorable, particularly for cases presenting watery diarrhea, which are typically self-limiting. In these instances, supportive care, including rehydration and symptomatic management, is sufficient, and patients usually recover fully without long-term sequelae [57]. The use of antibiotics is reserved for severe cases or specific subtypes, such as certain enterotoxigenic or enteropathogenic strains, and treatment is generally effective when appropriately administered. The prognosis becomes more guarded in children who develop hemolytic uremic syndrome (HUS) secondary to EHEC/STEC infections. In this population, approximately 4% of affected children may experience mortality, and an additional 5% are at risk for long-term complications such as end-stage renal disease, stroke, or persistent renal impairment [58]. Another 20% to 30% may develop various sequelae, whereas the majority of children recover within two weeks without enduring health consequences [59][60][61]. For patients with extraintestinal E coli infections, prognosis is largely influenced by baseline comorbid conditions and the severity of the primary illness rather than the pathogen itself. While uncomplicated cystitis has an excellent prognosis, infections such as E coli-induced bacteremia, spontaneous bacterial peritonitis, or ventilator-associated pneumonia are associated with higher morbidity and mortality due to underlying illness and physiological compromise. For instance, spontaneous bacterial peritonitis in patients with ascites carries a mortality risk of up to 4%, even with prompt and appropriate antimicrobial therapy [62]. Consequently, clinical outcomes in extraintestinal E coli infections depend on both timely recognition and treatment of the infection, as well as the patient's underlying health status and capacity to tolerate systemic illness.

Complications

Patients with Escherichia coli-induced diarrheal illness are at risk of both acute and long-term complications, depending on the strain involved, host factors, and disease severity. Acute complications most commonly arise from fluid and electrolyte imbalances. Profuse watery diarrhea can rapidly deplete intravascular volume, leading to dehydration, hypotension, and, in severe cases, acute kidney injury. Prompt recognition and early intervention through oral or intravenous rehydration significantly reduce the risk of these outcomes, and patients who receive timely supportive care generally recover without sequelae [57][58]. In addition to dehydration, patients may experience electrolyte disturbances, including hyponatremia, hypokalemia, and metabolic acidosis, which can exacerbate cardiovascular or neurologic complications in susceptible populations such as the elderly or individuals with chronic comorbidities. Long-term gastrointestinal complications are less frequent but may include chronic diarrhea, post-infectious irritable bowel syndrome, or persistent dysmotility. These conditions occur in a minority of patients but can result in prolonged morbidity and diminished quality of life. Specific E coli subtypes carry additional risk profiles. Hemolytic uremic syndrome (HUS), a potentially life-threatening complication, is associated predominantly with Shiga toxin-producing EHEC/STEC strains. The risk of HUS is highest in children under five years of age and older adults over sixty. Expression of Stx2 significantly increases HUS incidence, with rates reported up to 24% among infected individuals [57][59]. HUS is characterized by microangiopathic hemolytic anemia, thrombocytopenia, and acute kidney injury, which may necessitate renal replacement therapy in severe cases. Among pediatric patients who survive EHEC/STEC-induced HUS, approximately 5% develop end-stage renal disease or cerebrovascular events, while an additional 20% to 30% experience long-term sequelae such as hypertension, persistent proteinuria, and subclinical declines in glomerular filtration rate [60][61]. Complications associated with extraintestinal E coli infections are heterogeneous and largely determined by the affected organ system, host comorbidities, and the presence of antimicrobial resistance, and they are not the primary focus of this discussion.

Consultations

Interdisciplinary consultations are critical for the management of complex or severe E coli infections, as timely specialist involvement improves diagnostic accuracy, guides targeted therapy, and mitigates the risk of complications. Infectious disease specialists play a central role in interpreting laboratory and microbiological data, particularly when drug-resistant strains, including extended-spectrum beta-lactamase (ESBL) or carbapenemase-producing E coli, are identified. Their input is essential for selecting appropriate antimicrobial therapy, adjusting dosages, and preventing further resistance development. Additionally, infectious disease consultation assists in the evaluation of

atypical or recurrent infections and contributes to infection control strategies within healthcare settings [57][62]. Nephrology consultation is indicated for patients with EHEC/STEC-induced HUS or those exhibiting evidence of renal compromise. Nephrologists provide close monitoring of renal function, manage fluid and electrolyte imbalances, and determine the need for renal replacement therapy. Pediatric and adult nephrology teams are particularly valuable in preventing irreversible kidney damage and managing long-term sequelae such as chronic kidney disease or hypertension [59][60].

Critical care specialists are essential for patients with systemic involvement, including sepsis, hemodynamic instability, or multi-organ dysfunction. Their expertise ensures comprehensive monitoring, prompt fluid resuscitation, hemodynamic support, and management of complications related to organ failure. Similarly, gastroenterology input may be warranted in cases of severe, persistent, or unexplained diarrheal illness. Endoscopic evaluation or advanced diagnostic testing guided by gastroenterologists can help identify underlying mucosal pathology, guide targeted interventions, and exclude alternative diagnoses. Overall, coordinated consultation across infectious disease, nephrology, critical care, and gastroenterology ensures optimal patient outcomes by facilitating early recognition of complications, guiding individualized therapy, and promoting comprehensive management of both acute and long-term consequences of E coli infections [57][62].

Patient Education

Effective prevention of Escherichia coli infections relies on both personal hygiene and food safety measures. Hand hygiene remains the cornerstone of disease prevention, particularly after using the restroom, handling raw foods, or contact with contaminated surfaces. Proper washing of fruits and vegetables, thorough cooking of meats, and avoiding consumption of raw or undercooked animal products significantly reduce the risk of acquiring pathogenic E coli strains. Travelers to regions with suboptimal sanitation are at increased risk for diarrheal illness; therefore, consuming purified or appropriately treated water, thoroughly cooking food, and washing produce with purified water are essential preventive measures [51]. For individuals at elevated risk of complications from E coli infection, such as immunosuppressed patients or those with underlying comorbidities, prophylactic antibiotics may be considered under professional guidance. The International Society of Travel Medicine (ISTM) recommends chemoprophylaxis using rifaximin or bismuth-subsalicylate for travelers at risk of developing traveler's diarrhea. Rifaximin is prescribed at 200 mg one to three times daily for the duration of travel, not exceeding two weeks, while bismuth-subsalicylate can be administered at 524 mg every 30 to 60 minutes as needed, up to eight doses within 24 hours [51]. Prevention of extraintestinal infections is largely context-specific and relies on interventions that mitigate environmental or iatrogenic risk factors. Reducing the use of indwelling medical devices decreases the incidence of catheter-associated urinary tract infections, while protocols in critical care settings, such as elevating the head of the bed to 30 degrees, minimizing aspiration and lowering the rates of ventilator-associated pneumonia [63]. For high-risk populations, chemoprophylaxis can also reduce the incidence of spontaneous bacterial peritonitis, particularly in patients with advanced liver disease or ascites [64]. Educating patients and caregivers on these preventive strategies, alongside timely recognition of early symptoms, empowers individuals to actively reduce infection risk and mitigates complications from both intestinal and extraintestinal E coli infections.

Enhancing Healthcare Team Outcomes

The clinical spectrum of Escherichia coli infections ranges from self-limiting diarrhea to life-threatening extraintestinal illnesses, including urinary tract infections, bacteremia, pneumonia, and peritonitis. Pathogenic strains of E coli exploit diverse virulence factors and increasingly exhibit resistance to commonly prescribed antibiotics, heightening the need for accurate, timely diagnosis and evidence-based therapeutic interventions to optimize patient outcomes. The interprofessional healthcare team is central to mitigating morbidity, improving safety, and ensuring effective management [66]. Travel medicine specialists are particularly critical in identifying patients at elevated risk for travel-associated diarrheal illnesses and in providing prophylactic interventions based on updated guidelines. These clinicians, often supported by nursing staff, provide comprehensive counseling on hygiene practices, dietary precautions, and appropriate use of chemoprophylaxis to prevent ETEC and other pathogenic strains during international travel [65]. Managing E coli infections requires coordinated efforts among multiple healthcare professionals. Physicians, general practitioners, and advanced practitioners are responsible for patient assessment, high-risk stratification, and selection of diagnostic and therapeutic strategies. Nurses play a key role in monitoring hydration status, administering therapies, and reinforcing patient education regarding prevention, early symptom recognition, and adherence to prescribed regimens. Pharmacists contribute by reviewing antimicrobial therapy, advising on resistance concerns, ensuring appropriate dosing, and supporting antimicrobial stewardship programs. Interprofessional collaboration promotes timely communication, coordinated interventions, and adherence to best practices, all of which reduce

the risk of complications such as hemolytic uremic syndrome, multidrug-resistant infections, and healthcare-associated E coli infections. This collaborative approach not only improves individual patient outcomes but also strengthens population-level public health initiatives, contributing to the broader containment of E coli transmission and prevention of outbreaks [66].

Conclusion:

Escherichia coli continues to pose a substantial challenge to public health due to its dual role as both a commensal organism and a versatile pathogen. The wide range of intestinal and extraintestinal diseases caused by pathogenic *E. coli* strains reflects the organism's diverse virulence mechanisms and adaptive capacity. Intestinal pathotypes account for a large proportion of diarrheal disease worldwide, particularly in children and travelers, while extraintestinal infections—most notably urinary tract infections and bacteremia—represent a major cause of healthcare-associated morbidity and mortality. The increasing prevalence of antimicrobial-resistant *E. coli*, including ESBL- and carbapenemase-producing strains, significantly complicates clinical management and underscores the necessity for robust antimicrobial stewardship programs. Advances in molecular diagnostics have improved pathotype identification and facilitated early intervention; however, their implementation remains uneven, particularly in resource-limited settings. Effective prevention of *E. coli* infections depends on comprehensive public health strategies, including food safety measures, improved sanitation, infection control in healthcare settings, and patient education. Interprofessional collaboration among clinicians, microbiologists, pharmacists, and public health professionals is essential to optimize patient outcomes and limit transmission. Strengthening surveillance systems and promoting evidence-based practices are critical steps toward reducing the global burden of *E. coli* infections.

References:

1. Foster-Nyarko E, Pallen MJ. The microbial ecology of Escherichia coli in the vertebrate gut. FEMS microbiology reviews. 2022 May 6:46(3):. doi: 10.1093/femsre/fuac008.
2. Nataro JP, Kaper JB. Diarrheagenic Escherichia coli. Clinical microbiology reviews. 1998 Jan:11(1):142-201
3. Mylotte JM, Tayara A, Goodnough S. Epidemiology of bloodstream infection in nursing home residents: evaluation in a large cohort from multiple homes. Clinical infectious diseases : an official publication of the Infectious Diseases Society of America. 2002 Dec 15:35(12):1484-90
4. McCue JD. Gram-negative bacillary bacteremia in the elderly: incidence, ecology, etiology, and mortality. Journal of the American Geriatrics Society. 1987 Mar:35(3):213-8
5. Jain S, Self WH, Wunderink RG, Fakhran S, Balk R, Bramley AM, Reed C, Grijalva CG, Anderson EJ, Courtney DM, Chappell JD, Qi C, Hart EM, Carroll F, Trabue C, Donnelly HK, Williams DJ, Zhu Y, Arnold SR, Ampofo K, Waterer GW, Levine M, Lindstrom S, Winchell JM, Katz JM, Erdman D, Schneider E, Hicks LA, McCullers JA, Pavia AT, Edwards KM, Finelli L, CDC EPIC Study Team. Community-Acquired Pneumonia Requiring Hospitalization among U.S. Adults. The New England journal of medicine. 2015 Jul 30:373(5):415-27. doi: 10.1056/NEJMoa1500245.
6. Sligl W, Taylor G, Brindley PG. Five years of nosocomial Gram-negative bacteremia in a general intensive care unit: epidemiology, antimicrobial susceptibility patterns, and outcomes. International journal of infectious diseases : IJID : official publication of the International Society for Infectious Diseases. 2006 Jul:10(4):320-5
7. Kotloff KL, Nataro JP, Blackwelder WC, Nasrin D, Farag TH, Panchalingam S, Wu Y, Sow SO, Sur D, Breiman RF, Faruque AS, Zaidi AK, Saha D, Alonso PL, Tamboura B, Sanogo D, Onwuchekwa U, Manna B, Ramamurthy T, Kanungo S, Ochieng JB, Omore R, Oundo JO, Hossain A, Das SK, Ahmed S, Qureshi S, Quadri F, Adegbola RA, Antonio M, Hossain MJ, Akinsola A, Mandomando I, Nhampossa T, Acácio S, Biswas K, O'Reilly CE, Mintz ED, Berkeley LY, Muhsen K, Sommerfelt H, Robins-Browne RM, Levine MM. Burden and aetiology of diarrhoeal disease in infants and young children in developing countries (the Global Enteric Multicenter Study, GEMS): a prospective, case-control study. Lancet (London, England). 2013 Jul 20:382(9888):209-22. doi: 10.1016/S0140-6736(13)60844-2.
8. Ochoa TJ, Contreras CA. Enteropathogenic escherichia coli infection in children. Current opinion in infectious diseases. 2011 Oct:24(5):478-83. doi: 10.1097/QCO.0b013e32834a8b8b.
9. Regua AH, Bravo VL, Leal MC, Lobo Leite ME. Epidemiological survey of the enteropathogenic Escherichia coli isolated from children with diarrhoea. Journal of tropical pediatrics. 1990 Aug:36(4):176-9. doi: 10.1093/tropej/36.4.176.
10. Huang DB, Nataro JP, DuPont HL, Kamat PP, Mhatre AD, Okhuysen PC, Chiang T. Enteroaggregative Escherichia coli is a cause of acute diarrheal illness: a meta-

analysis. Clinical infectious diseases : an official publication of the Infectious Diseases Society of America. 2006 Sep 1:43(5):556-63
11. de la Cabada Bauche J, Dupont HL. New Developments in Traveler's Diarrhea. Gastroenterology & hepatology. 2011 Feb:7(2):88-95
12. Karch H, Bielaszewska M. Sorbitol-fermenting Shiga toxin-producing Escherichia coli O157:H(-) strains: epidemiology, phenotypic and molecular characteristics, and microbiological diagnosis. Journal of clinical microbiology. 2001 Jun:39(6):2043-9
13. Tarr PI. Escherichia coli O157:H7: clinical, diagnostic, and epidemiological aspects of human infection. Clinical infectious diseases : an official publication of the Infectious Diseases Society of America. 1995 Jan:20(1):1-8; quiz 9-10
14. Mead PS, Griffin PM. Escherichia coli O157:H7. Lancet (London, England). 1998 Oct 10:352(9135):1207-12
15. Boyce TG, Swerdlow DL, Griffin PM. Escherichia coli O157:H7 and the hemolytic-uremic syndrome. The New England journal of medicine. 1995 Aug 10:333(6):364-8
16. Raffaelli RM, Paladini M, Hanson H, Kornstein L, Agasan A, Slavinski S, Weiss D, Fennelly GJ, Flynn JT. Child care-associated outbreak of Escherichia coli O157:H7 and hemolytic uremic syndrome. The Pediatric infectious disease journal. 2007 Oct:26(10):951-3
17. Tack DM, Ray L, Griffin PM, Cieslak PR, Dunn J, Rissman T, Jervis R, Lathrop S, Muse A, Duwell M, Smith K, Tobin-D'Angelo M, Vugia DJ, Zablotsky Kufel J, Wolpert BJ, Tauxe R, Payne DC. Preliminary Incidence and Trends of Infections with Pathogens Transmitted Commonly Through Food - Foodborne Diseases Active Surveillance Network, 10 U.S. Sites, 2016-2019. MMWR. Morbidity and mortality weekly report. 2020 May 1:69(17):509-514. doi: 10.15585/mmwr.mm6917a1.
18. Kaper JB, Nataro JP, Mobley HL. Pathogenic Escherichia coli. Nature reviews. Microbiology. 2004 Feb:2(2):123-40
19. Bonten M, Johnson JR, van den Biggelaar AHJ, Georgalis L, Geurtsen J, de Palacios PI, Gravenstein S, Verstraeten T, Hermans P, Poolman JT. Epidemiology of Escherichia coli Bacteremia: A Systematic Literature Review. Clinical infectious diseases : an official publication of the Infectious Diseases Society of America. 2021 Apr 8:72(7):1211-1219. doi: 10.1093/cid/ciaa210.
20. Begier E, Rosenthal NA, Gurtman A, Kartashov A, Donald RGK, Lockhart SP. Epidemiology of Invasive Escherichia coli Infection and Antibiotic Resistance Status Among Patients Treated in US Hospitals: 2009-2016. Clinical infectious diseases : an official publication of the Infectious Diseases Society of America. 2021 Aug 16:73(4):565-574. doi: 10.1093/cid/ciab005.
21. Croxen MA, Finlay BB. Molecular mechanisms of Escherichia coli pathogenicity. Nature reviews. Microbiology. 2010 Jan:8(1):26-38. doi: 10.1038/nrmicro2265.
22. Levine MM, Caplan ES, Waterman D, Cash RA, Hornick RB, Snyder MJ. Diarrhea caused by Escherichia coli that produce only heat-stable enterotoxin. Infection and immunity. 1977 Jul:17(1):78-82
23. Zhang Y, Tan P, Zhao Y, Ma X. Enterotoxigenic Escherichia coli: intestinal pathogenesis mechanisms and colonization resistance by gut microbiota. Gut microbes. 2022 Jan-Dec:14(1):2055943. doi: 10.1080/19490976.2022.2055943.
24. Lee W, Sung S, Ha J, Kim E, An ES, Kim SH, Kim SH, Kim HY. Molecular and Genomic Analysis of the Virulence Factors and Potential Transmission of Hybrid Enteropathogenic and Enterotoxigenic Escherichia coli (EPEC/ETEC) Strains Isolated in South Korea. International journal of molecular sciences. 2023 Aug 12:24(16):. doi: 10.3390/ijms241612729.
25. Pinaud L, Sansonetti PJ, Phalipon A. Host Cell Targeting by Enteropathogenic Bacteria T3SS Effectors. Trends in microbiology. 2018 Apr:26(4):266-283. doi: 10.1016/j.tim.2018.01.010.
26. Gaytán MO, Martínez-Santos VI, Soto E, González-Pedrajo B. Type Three Secretion System in Attaching and Effacing Pathogens. Frontiers in cellular and infection microbiology. 2016:6():129
27. Viswanathan VK, Hodges K, Hecht G. Enteric infection meets intestinal function: how bacterial pathogens cause diarrhoea. Nature reviews. Microbiology. 2009 Feb:7(2):110-9. doi: 10.1038/nrmicro2053.
28. Pakbin B, Brück WM, Rossen JWA. Virulence Factors of Enteric Pathogenic Escherichia coli: A Review. International journal of molecular sciences. 2021 Sep 14:22(18):. doi: 10.3390/ijms22189922.

29. Nataro JP. Enteroaggregative Escherichia coli pathogenesis. Current opinion in gastroenterology. 2005 Jan:21(1):4-8
30. Morin N, Santiago AE, Ernst RK, Guillot SJ, Nataro JP. Characterization of the AggR regulon in enteroaggregative Escherichia coli. Infection and immunity. 2013 Jan:81(1):122-32. doi: 10.1128/IAI.00676-12.
31. Melton-Celsa AR. Shiga Toxin (Stx) Classification, Structure, and Function. Microbiology spectrum. 2014 Aug:2(4):EHEC-0024-2013. doi: 10.1128/microbiolspec.EHEC-0024-2013.
32. Perna NT, Plunkett G 3rd, Burland V, Mau B, Glasner JD, Rose DJ, Mayhew GF, Evans PS, Gregor J, Kirkpatrick HA, Pósfai G, Hackett J, Klink S, Boutin A, Shao Y, Miller L, Grotbeck EJ, Davis NW, Lim A, Dimalanta ET, Potamousis KD, Apodaca J, Anantharaman TS, Lin J, Yen G, Schwartz DC, Welch RA, Blattner FR. Genome sequence of enterohaemorrhagic Escherichia coli O157:H7. Nature. 2001 Jan 25:409(6819):529-33
33. Stevens MP, Frankel GM. The Locus of Enterocyte Effacement and Associated Virulence Factors of Enterohemorrhagic Escherichia coli. Microbiology spectrum. 2014 Aug:2(4):EHEC-0007-2013. doi: 10.1128/microbiolspec.EHEC-0007-2013.
34. Bielaszewska M, Aldick T, Bauwens A, Karch H. Hemolysin of enterohemorrhagic Escherichia coli: structure, transport, biological activity and putative role in virulence. International journal of medical microbiology : IJMM. 2014 Jul:304(5-6):521-9. doi: 10.1016/j.ijmm.2014.05.005.
35. Boyer O, Niaudet P. Hemolytic-Uremic Syndrome in Children. Pediatric clinics of North America. 2022 Dec:69(6):1181-1197. doi: 10.1016/j.pcl.2022.07.006.
36. Govindarajan DK, Eskeziyaw BM, Kandaswamy K, Mengistu DY. Diagnosis of extraintestinal pathogenic Escherichia coli pathogenesis in urinary tract infection. Current research in microbial sciences. 2024:7():100296. doi: 10.1016/j.crmicr.2024.100296.
37. Russo TA, Johnson JR. Medical and economic impact of extraintestinal infections due to Escherichia coli: focus on an increasingly important endemic problem. Microbes and infection. 2003 Apr:5(5):449-56
38. Takhar SS, Moran GJ. Diagnosis and management of urinary tract infection in the emergency department and outpatient settings. Infectious disease clinics of North America. 2014 Mar:28(1):33-48. doi: 10.1016/j.idc.2013.10.003.
39. Hsieh VC, Hsieh ML, Chiang JH, Chien A, Hsieh MS. Emergency Department Visits and Disease Burden Attributable to Ambulatory Care Sensitive Conditions in Elderly Adults. Scientific reports. 2019 Mar 7:9(1):3811. doi: 10.1038/s41598-019-40206-4.
40. Phillips-Houlbracq M, Ricard JD, Foucrier A, Yoder-Himes D, Gaudry S, Bex J, Messika J, Margetis D, Chatel J, Dobrindt U, Denamur E, Roux D. Pathophysiology of Escherichia coli pneumonia: Respective contribution of pathogenicity islands to virulence. International journal of medical microbiology : IJMM. 2018 Mar:308(2):290-296. doi: 10.1016/j.ijmm.2018.01.003.
41. Sievert DM, Ricks P, Edwards JR, Schneider A, Patel J, Srinivasan A, Kallen A, Limbago B, Fridkin S, National Healthcare Safety Network (NHSN) Team and Participating NHSN Facilities. Antimicrobial-resistant pathogens associated with healthcare-associated infections: summary of data reported to the National Healthcare Safety Network at the Centers for Disease Control and Prevention, 2009-2010. Infection control and hospital epidemiology. 2013 Jan:34(1):1-14. doi: 10.1086/668770.
42. John TM, Deshpande A, Brizendine K, Yu PC, Rothberg MB. Epidemiology and Outcomes of Community-Acquired Escherichia coli Pneumonia. Open forum infectious diseases. 2022 Jan:9(1):ofab597. doi: 10.1093/ofid/ofab597.
43. Donnenberg MS, Narayanan S. How to diagnose a foodborne illness. Infectious disease clinics of North America. 2013 Sep:27(3):535-54. doi: 10.1016/j.idc.2013.05.001.
44. Shane AL, Mody RK, Crump JA, Tarr PI, Steiner TS, Kotloff K, Langley JM, Wanke C, Warren CA, Cheng AC, Cantey J, Pickering LK. 2017 Infectious Diseases Society of America Clinical Practice Guidelines for the Diagnosis and Management of Infectious Diarrhea. Clinical infectious diseases : an official publication of the Infectious Diseases Society of America. 2017 Nov 29:65(12):e45-e80. doi: 10.1093/cid/cix669.
45. Ojeda A, Prado V, Martinez J, Arellano C, Borczyk A, Johnson W, Lior H, Levine MM. Sorbitol-negative phenotype among enterohemorrhagic Escherichia coli strains of different serotypes and from different sources. Journal of clinical microbiology. 1995 Aug:33(8):2199-201

46. Anderson NW, Tarr PI. Multiplex Nucleic Acid Amplification Testing to Diagnose Gut Infections: Challenges, Opportunities, and Result Interpretation. Gastroenterology clinics of North America. 2018 Dec:47(4):793-812. doi: 10.1016/j.gtc.2018.07.006.
47. Glaser MA, Hughes LM, Jnah A, Newberry D. Neonatal Sepsis: A Review of Pathophysiology and Current Management Strategies. Advances in neonatal care : official journal of the National Association of Neonatal Nurses. 2021 Feb 1:21(1):49-60. doi: 10.1097/ANC.0000000000000769.
48. Ramakrishnan K, Scheid DC. Diagnosis and management of acute pyelonephritis in adults. American family physician. 2005 Mar 1:71(5):933-42
49. Papazian L, Klompas M, Luyt CE. Ventilator-associated pneumonia in adults: a narrative review. Intensive care medicine. 2020 May:46(5):888-906. doi: 10.1007/s00134-020-05980-0.
50. Kerek Á, Román I, Szabó Á, Kovács D, Kardos G, Kovács L, Jerzsele Á. Antibiotic resistance genes in Escherichia coli - literature review. Critical reviews in microbiology. 2025 Apr 18:():1-35. doi: 10.1080/1040841X.2025.2492156.
51. Riddle MS, Connor BA, Beeching NJ, DuPont HL, Hamer DH, Kozarsky P, Libman M, Steffen R, Taylor D, Tribble DR, Vila J, Zanger P, Ericsson CD. Guidelines for the prevention and treatment of travelers' diarrhea: a graded expert panel report. Journal of travel medicine. 2017 Apr 1:24(suppl_1):S57-S74. doi: 10.1093/jtm/tax026.
52. Werber D, Mason BW, Evans MR, Salmon RL. Preventing household transmission of Shiga toxin-producing Escherichia coli O157 infection: promptly separating siblings might be the key. Clinical infectious diseases : an official publication of the Infectious Diseases Society of America. 2008 Apr 15:46(8):1189-96. doi: 10.1086/587670.
53. Grisaru S, Xie J, Samuel S, Hartling L, Tarr PI, Schnadower D, Freedman SB, Alberta Provincial Pediatric Enteric Infection Team. Associations Between Hydration Status, Intravenous Fluid Administration, and Outcomes of Patients Infected With Shiga Toxin-Producing Escherichia coli: A Systematic Review and Meta-analysis. JAMA pediatrics. 2017 Jan 1:171(1):68-76. doi: 10.1001/jamapediatrics.2016.2952.
54. Freedman SB, Xie J, Neufeld MS, Hamilton WL, Hartling L, Tarr PI, Alberta Provincial Pediatric Enteric Infection Team (APPETITE), Nettel-Aguirre A, Chuck A, Lee B, Johnson D, Currie G, Talbot J, Jiang J, Dickinson J, Kellner J, MacDonald J, Svenson L, Chui L, Louie M, Lavoie M, Eltorki M, Vanderkooi O, Tellier R, Ali S, Drews S, Graham T, Pang XL. Shiga Toxin-Producing Escherichia coli Infection, Antibiotics, and Risk of Developing Hemolytic Uremic Syndrome: A Meta-analysis. Clinical infectious diseases : an official publication of the Infectious Diseases Society of America. 2016 May 15:62(10):1251-1258. doi: 10.1093/cid/ciw099.
55. Aliabadi S, Jauneikaite E, Müller-Pebody B, Hope R, Vihta KD, Horner C, Costelloe CE. Exploring temporal trends and risk factors for resistance in Escherichia coli-causing bacteraemia in England between 2013 and 2018: an ecological study. The Journal of antimicrobial chemotherapy. 2022 Feb 23:77(3):782-792. doi: 10.1093/jac/dkab440.
56. Flannery DD, Akinboyo IC, Mukhopadhyay S, Tribble AC, Song L, Chen F, Li Y, Gerber JS, Puopolo KM. Antibiotic Susceptibility of Escherichia coli Among Infants Admitted to Neonatal Intensive Care Units Across the US From 2009 to 2017. JAMA pediatrics. 2021 Feb 1:175(2):168-175. doi: 10.1001/jamapediatrics.2020.4719.
57. Mody RK, Gu W, Griffin PM, Jones TF, Rounds J, Shiferaw B, Tobin-D'Angelo M, Smith G, Spina N, Hurd S, Lathrop S, Palmer A, Boothe E, Luna-Gierke RE, Hoekstra RM. Postdiarrheal hemolytic uremic syndrome in United States children: clinical spectrum and predictors of in-hospital death. The Journal of pediatrics. 2015 Apr:166(4):1022-9. doi: 10.1016/j.jpeds.2014.12.064.
58. Alconcher LF, Coccia PA, Suarez ADC, Monteverde ML, Perez Y Gutiérrez MG, Carlopio PM, Missoni ML, Balestracci A, Principi I, Ramírez FB, Estrella P, Micelli S, Leroy DC, Quijada NE, Seminara C, Giordano MI, Hidalgo Solís SB, Saurit M, Caminitti A, Arias A, Rivas M, Risso P, Liern M. Hyponatremia: a new predictor of mortality in patients with Shiga toxin-producing Escherichia coli hemolytic uremic syndrome. Pediatric nephrology (Berlin, Germany). 2018 Oct:33(10):1791-1798. doi: 10.1007/s00467-018-3991-6.
59. Spinale JM, Ruebner RL, Copelovitch L, Kaplan BS. Long-term outcomes of Shiga toxin hemolytic uremic syndrome. Pediatric nephrology (Berlin, Germany). 2013

Nov:28(11):2097-105. doi: 10.1007/s00467-012-2383-6.

60. Loos S, Aulbert W, Hoppe B, Ahlenstiel-Grunow T, Kranz B, Wahl C, Staude H, Humberg A, Benz K, Krause M, Pohl M, Liebau MC, Schild R, Lemke J, Beringer O, Müller D, Härtel C, Wigger M, Vester U, Konrad M, Haffner D, Pape L, Oh J, Kemper MJ. Intermediate Follow-up of Pediatric Patients With Hemolytic Uremic Syndrome During the 2011 Outbreak Caused by E. coli O104:H4. Clinical infectious diseases : an official publication of the Infectious Diseases Society of America. 2017 Jun 15:64(12):1637-1643. doi: 10.1093/cid/cix218.
61. Garg AX, Suri RS, Barrowman N, Rehman F, Matsell D, Rosas-Arellano MP, Salvadori M, Haynes RB, Clark WF. Long-term renal prognosis of diarrhea-associated hemolytic uremic syndrome: a systematic review, meta-analysis, and meta-regression. JAMA. 2003 Sep 10:290(10):1360-70
62. Rimola A, Salmerón JM, Clemente G, Rodrigo L, Obrador A, Miranda ML, Guarner C, Planas R, Solá R, Vargas V. Two different dosages of cefotaxime in the treatment of spontaneous bacterial peritonitis in cirrhosis: results of a prospective, randomized, multicenter study. Hepatology (Baltimore, Md.). 1995 Mar:21(3):674-9
63. Klompas M, Branson R, Eichenwald EC, Greene LR, Howell MD, Lee G, Magill SS, Maragakis LL, Priebe GP, Speck K, Yokoe DS, Berenholtz SM, Society for Healthcare Epidemiology of America (SHEA). Strategies to prevent ventilator-associated pneumonia in acute care hospitals: 2014 update. Infection control and hospital epidemiology. 2014 Aug:35(8):915-36. doi: 10.1086/677144.
64. Cohen MJ, Sahar T, Benenson S, Elinav E, Brezis M, Soares-Weiser K. Antibiotic prophylaxis for spontaneous bacterial peritonitis in cirrhotic patients with ascites, without gastro-intestinal bleeding. The Cochrane database of systematic reviews. 2009 Apr 15:2009(2):CD004791. doi: 10.1002/14651858.CD004791.
65. Hill DR, Ericsson CD, Pearson RD, Keystone JS, Freedman DO, Kozarsky PE, DuPont HL, Bia FJ, Fischer PR, Ryan ET, Infectious Diseases Society of America. The practice of travel medicine: guidelines by the Infectious Diseases Society of America. Clinical infectious diseases : an official publication of the Infectious Diseases Society of America. 2006 Dec 15:43(12):1499-539
66. Vermeir P, Vandijck D, Degroote S, Peleman R, Verhaeghe R, Mortier E, Hallaert G, Van Daele S, Buylaert W, Vogelaers D. Communication in healthcare: a narrative review of the literature and practical recommendations. International journal of clinical practice. 2015 Nov:69(11):1257-67. doi: 10.1111/ijcp.12686