Bacterial Enteric Infections

Updated: January 26, 2026
Reviewed: March 16, 2026

Epidemiology

HIV-associated alterations in mucosal immunity or intestinal integrity and treatment with acid-suppressive agents may increase the risk of enteric bacterial infections. Rates of Gram-negative bacterial enteric infections are at least 10 times higher among adults with HIV than in the general population, but these rates are lower among people with HIV who are treated with antiretroviral therapy (ART).[1] The risk of bacterial diarrhea varies according to CD4 T lymphocyte (CD4) cell count and is greatest in individuals with clinical AIDS or CD4 counts <200 cells/mm^3.

The bacteria most frequently isolated by culture from adults with HIV in the United States are *Shigella*, *Campylobacter*, and nontyphoidal *Salmonella* spp. (particularly *Salmonella enterica* serotypes Typhimurium and Enteritidis).[1-6] Diarrheagenic *Escherichia coli*, particularly enteroaggregative *E. coli*, may contribute to the burden of diarrheal disease,[7] but their role is understood poorly because reporting to public health systems is not required. Data on *Helicobacter pylori* infection in the setting of HIV infection are limited and do not suggest excess risk in people with HIV.[8] Resistance to clinically relevant antimicrobials is common or increasing among many bacterial enteric pathogens, with multi- or extensively drug-resistant shigellosis linked to global outbreaks among men who have sex with men.[9-13]

Clostridioides difficile–associated infection (CDI) is common in people with HIV[14]; in addition to traditional risk factors, such as exposure to a health care facility or to antibiotics, data[15] suggest that low CD4 count (<50 cells/mm^3) is an independent risk factor. Incidence of community-onset CDI is increasing, and clinicians also should consider CDI in the evaluation of outpatient diarrheal illnesses in people with HIV.

Other enteric infections that may cause diarrhea—such as *Mycobacterium avium* complex (MAC), cytomegalovirus, and various protozoa—are discussed elsewhere in these guidelines.

As with bacterial enteric infections in people without HIV, the probable source for most bacterial enteric infections in people with HIV is ingestion of contaminated food or water.[16] Sexual activity with the potential for direct or indirect fecal-oral exposure also increases risk of infections, especially with *Shigella*[3,17-20] and *Campylobacter*.[21,22]

Clinical Manifestations

Three major clinical syndromes of infection are associated with Gram-negative enteric bacteria among people with HIV:

- Self-limited gastroenteritis;
- Severe and prolonged diarrheal disease, potentially associated with fever, bloody diarrhea, and weight loss; *and*
- Bacteremia associated with extra-intestinal involvement, with or without concurrent or preceding gastrointestinal (GI) illness.[6,23,24]

Severe community-associated diarrhea often is defined as six or more loose stools (loose stool is defined as defecated material that takes the shape of a container) per day with or without other signs of systemic illness, such as fecal blood, orthostatic hypotension, or fever. In people with HIV, the risk of more profound illness increases with the degree of immunosuppression but risk diminishes with ART.[1,4,5,16,24,25] Relapses in infection with *Salmonella* and other Gram-negative bacterial enteric pathogens after appropriate treatment have been well documented in people with HIV.[16,26,27] As in other populations, CDI can cause a variety of syndromes, from watery diarrhea to toxic megacolon.[15]

Although enteric pathogens can be associated with clinical proctitis (e.g., pain with defecation, tenesmus, bloody discharge),[28] other infections that may be transmitted during intimate contact (e.g., *Chlamydia trachomatis* including lymphogranuloma venereum, *Neisseria gonorrhoeae,* herpes simplex virus, *Treponema pallidum,* and mpox) more commonly cause this syndrome, especially in those with relevant exposures (e.g., condomless receptive anal intercourse).[29] Proctocolitis with diarrhea caused by sexually transmitted infections (STIs) is less common but may occur; relevant exposures should be queried.[30]

Diagnosis

Assessment of patients with diarrhea should include a complete exposure history (i.e., ingestion of contaminated food or water, including through recreational exposure to water, sexual history or other fecal-oral exposures, animal/pet exposures, travel-related exposures, exposure to antibiotics or chemotherapies, use of acid-suppressing medications, or recent hospitalization); a medication review, because diarrhea is a common side effect of some ART and antibiotics; quantification of the diarrheal illness by stool frequency, consistency, volume, duration, and presence of blood; and associated signs and symptoms, such as presence and duration of fever. Physical examination should include measurement of temperature and assessment of intravascular volume and nutritional status.

The diagnosis of Gram-negative bacterial enteric infection is established through cultures of stool and blood or stool molecular methods (i.e., culture-independent diagnostic tests [CIDTs]), ideally before antibiotics are given. Although stool molecular methods rapidly diagnose enteric infections, stool cultures are required to obtain phenotypic antibiotic sensitivity testing for isolated enteric pathogens and may also be helpful during outbreak investigations to identify the source. Thus, the Centers for Disease Control and Prevention (CDC) recommends reflex stool cultures and antibiotic sensitivity testing for specimens with positive CIDT reports given increasing resistance detected in enteric bacterial infections.[31] Because incidence of bacteremia associated with *Salmonella* gastroenteritis is high in people with HIV—particularly those with advanced disease—blood cultures should be obtained from any patient who has diarrhea and fever. For shigellosis, blood cultures may be helpful but are less likely to be positive than in salmonellosis.[24]

Other infections for which people with HIV are at risk, albeit at a lower rate, are non-*jejuni,* non-*coli Campylobacter* spp.—such as *C. fetus, C. upsaliensis,* and *C. lari*—and the enterohepatic *Helicobacter* spp. (*H. cineadi* and *H. fennelliae)*, which were described originally as *Campylobacter* spp. Blood culture systems typically will grow these bacteria, but they are unlikely to be identified on routine stool cultures performed by most laboratories because growing these fastidious organisms requires special stool culture conditions.

The diagnosis of CDI can be made only through careful selection of the correct population for testing and a correlation of clinical and laboratory findings. Populations at risk for *C. difficile* diarrhea include individuals who recently received or currently are receiving antibiotics (including antimicrobial prophylaxis) or cancer chemotherapy, those who have been hospitalized in the past 4 to 6 weeks (or currently are hospitalized), those who reside in long-term care facilities, those with CD4

counts <200 cells/mm^3, those taking acid-suppressive medications, and those with moderate-to-severe community-acquired diarrhea.[32] Only people with diarrhea (defined as three or more loose stools in 24 hours) should be tested for CDI to limit detection of asymptomatic colonization, and only stool samples that take the shape of the container (i.e., diarrheal) should be tested.[33] Detection of either the *C. difficile* toxin B gene (using nucleic acid amplification testing [NAAT]) or the *C. difficile* toxin B protein (using an enzyme immunoassay [EIA]) is required for diagnosis. Current EIAs suffer from low sensitivity, whereas polymerase chain reaction (or PCR) assays have high sensitivity and can detect asymptomatic carriers. Glutamate dehydrogenase (GDH) antigen enzyme immunoassays, which detect an antigen common to *C. difficile* strains, whether or not toxigenic, must be combined with a second confirmatory test for stool *C. difficile* toxin B.[34,35] Based on the criteria above (i.e., symptoms meet the definition of diarrhea, and the stool sample is diarrhea, taking the shape of the container), Infectious Diseases Society of America (IDSA) and Society for Healthcare Epidemiology of America (or SHEA) guidelines for CDI support using an NAAT alone or a multiple-step algorithm (e.g., GDH plus toxin B assay) versus an EIA alone for *C. difficile* testing.[35]

Endoscopy generally should be reserved for patients in whom stool culture, microscopy, *C. difficile* toxin B assay, and blood culture fail to reveal an etiology or in whom treatment for an established diagnosis fails. Endoscopy with biopsy may be required for diagnosing etiologies other than bacterial enteric infections—including cryptosporidiosis, microsporidiosis, cytomegalovirus, or MAC gastroenteritis—and noninfectious causes of GI symptoms.

Clinicians should remain alert to the possibility of STIs. In patients with relevant exposures and symptoms of proctitis or proctocolitis, diagnostic evaluation and treatment for STIs are recommended.[36]

Preventing Exposure

Multiple epidemiologic exposures can place people at risk for enteric illnesses. The most common are ingestion of contaminated food or water and fecal-oral exposures. Health care providers can help patients avoid illness by educating them about transmission risk factors. The clinical condition and CD4 count of a person with HIV can help the provider determine what prevention recommendations are most appropriate. People with HIV with CD4 counts <200 cells/mm^3 or a history of AIDS-defining illness[37] are at the greatest risk of enteric illnesses; however, excess risk of undetermined magnitude or duration may persist in those with lesser degrees of immune impairment, including individuals treated with ART.

Individuals in the community should be advised to wash their hands regularly with soap and water or alcohol-based cleansers to reduce the risk of enteric infection **(AIII).** To prevent enteric infections, soap and water are preferred over alcohol-based cleansers, which do not kill *C. difficile* spores and are active only partially against norovirus and *Cryptosporidium* **(AIII).** People with HIV should be advised to wash their hands with soap and water after potential contact with human feces (e.g., through defecation, sexual exposures, cleaning feces from infants, contact with a person who has diarrhea), after handling pets or other animals, after gardening or other contact with soil, and before preparing food and eating **(AIII).** In addition to handwashing, use of barriers (e.g., condoms, dental dams, gloves) can reduce exposure to feces when engaging in sex practices such as anal sex and oral-anal contact **(AIII).**[28,36,38] Avoiding sex while any partner has diarrhea may further reduce risk of transmission. Travelers to relevant locations may be counseled on food and water hygiene (see the CDC Travelers' Health webpage).[39]

Preventing Disease

Recommendations for Preventing Bacterial Enteric Infections
• Immunizations (e.g., against *Salmonella serotype* Typhi) should be recommended in advance of travel to relevant locations (see Immunizations for Preventable Diseases in Adults and Adolescents With HIV in the Adult and Adolescent Opportunistic Infection Guidelines) (AIII).
• Antimicrobial prophylaxis to prevent bacterial enteric illness is not routinely recommended, including for travelers (AIII).
• In rare cases—such as for immunosuppressed travelers (depending on their level of immunosuppression, the region of travel, and the trip's duration)—antimicrobial prophylaxis with rifaximin or azithromycin should be offered (CIII).
• Because of toxicity associated with fluoroquinolone use (e.g., CDI, tendinitis) and increasing rates of antimicrobial resistance among enteric bacterial pathogens outside of the United States, routine use of fluoroquinolones for prophylaxis is discouraged (AIII).
• During pregnancy, azithromycin is the preferred agent for prophylaxis (BIII).

Key: CDI = *Clostridioides difficile*–associated infection

Antimicrobial prophylaxis to prevent bacterial enteric illness **is not routinely recommended,** including for travelers **(AIII).** Prophylactic antimicrobial treatment can elicit adverse reactions, promote the emergence of resistant organisms, and increase the risk of CDI. In rare cases, however, antimicrobial prophylaxis (e.g., with rifaximin or azithromycin) should be considered—such as for immunosuppressed travelers, depending on their level of immunosuppression, the region of travel, and the trip's duration **(CIII).**[40,41] In addition, immunizations, (e.g., against *Salmonella* serotype Typhi), should be recommended in advance of travel to relevant locations (see Immunizations for Travel in the Immunizations section of the Adult and Adolescent Opportunistic Infection Guidelines) **(AIII).**

For people with HIV already taking trimethoprim-sulfamethoxazole (TMP-SMX) (e.g., for *Pneumocystis jirovecii* pneumonia prophylaxis), TMP-SMX may offer limited protection against traveler's diarrhea.[42] During pregnancy, azithromycin would be the preferred agent for prophylaxis **(BIII).** Clinicians should be aware of concerns about fluoroquinolone safety. Given increased recognition of fluoroquinolone toxicities, as well as increasing rates of antimicrobial resistance among enteric bacterial pathogens outside the United States, routine use of fluoroquinolones for prophylaxis is discouraged **(AIII).**[43]

Treating Disease

Recommendations for Treating Bacterial Enteric Infections
General Considerations When Managing Patients With Bacterial Enteric Infections
• Oral or IV rehydration therapy (if indicated) should be given to patients with diarrhea (AIII).
• Antimotility agents should be avoided if concern about inflammatory diarrhea, including CDI, exists (BIII).
• Diagnostic fecal specimens should be obtained before initiation of empiric antimicrobial therapy.
• If a pathogen is identified in stool, antibiotic susceptibilities should be performed to confirm and inform antibiotic choice given increased reports of antibiotic resistance. Reflexively culturing the stool of patients diagnosed using PCR-based methods can facilitate antibiotic susceptibility testing among these patients.

- Risk of a bacterial enteric infection increases as CD4 count declines, with the greatest risk in people with CD4 counts <200 cells/mm^3. Risk of bacteremia also increases with decreasing CD4 count. If no clinical response occurs after 3 to 4 days of therapy, consider follow-up stool culture with antibiotic susceptibility testing and other methods to detect enteric pathogens (e.g., toxin assays, molecular methods), alternative diagnosis, antibiotic resistance, or drug–drug interactions (BIII).
- Effective ART may reduce the frequency, severity, and recurrence of bacterial enteric infections.

Empiric Treatment of Bacterial Enteric Infections (Pending Diagnostic Studies and Antimicrobial Resistance Testing)

For People With HIV and CD4 >500 cells/mm^3, With 1–2 Days of Loose Stools Without Fever or Blood

- Oral hydration; no further work-up and no treatment is needed.

For People With HIV and CD4 Count 200–500 cells/mm^3, With Diarrhea Severe Enough to Compromise Quality of Life or Ability to Work

- Azithromycin 500 mg PO daily for 5 days (BIII), *or*
- Ciprofloxacin 500–750 mg PO every 12 hours for 5 days (BIII)

For People With HIV and Severe Disease (e.g., people with CD4 count <200 cells/mm^3 or concomitant AIDS-defining illnesses), With Clinically Severe Diarrhea (**≥6 liquid stools/day or bloody stool and/or accompanying fever or chills)**

- Hospitalization for inpatient diagnostic evaluation and IV antibiotics
- Ceftriaxone 1–2 g IV every 24 hours (BIII) until antimicrobial susceptibility is available, then treatment can be changed based on sensitivity results.
    - If *Campylobacter* or *Shigella* bacteremia is suspected, a carbapenem is preferred for empiric therapy (BIII).

Duration of Therapy

- Therapy and its duration should be adjusted depending on stool microbiology results and antibiotic sensitivity testing. See recommendations for specific bacteria below. If no pathogen is identified and the patient recovers quickly, 5 days of therapy is recommended.

Other Considerations

- MSM may be at increased risk for antibiotic-resistant enteric infections.
- Diarrhea is a common illness of international travelers. Antimicrobial resistance among enteric bacterial pathogens outside the United States is common. Clinicians should consider the possibility of resistant infections when prescribing empiric antibiotic therapy for travelers with HIV while traveling or upon return to the United States, particularly among travelers to South and Southeast Asia or Africa.
- For patients with persistent diarrhea (>14 days) but no other severe clinical signs (e.g., dehydration, blood in stool), antibiotic therapy can be withheld until a diagnosis is confirmed. Noninfectious etiologies of persistent diarrhea (e.g., inflammatory bowel disease) also can be considered in the differential diagnosis (BIII).
- Azithromycin should not be used to treat bacteremia.
- Before susceptibilities are known, empiric IV ceftriaxone is recommended, unless *Campylobacter* or *Shigella* are suspected/identified; however, given the rise of antimicrobial resistance in enteric pathogens, updated outbreak information, local susceptibility patterns, and travel history should always be considered.

Treating Nontyphoidal Salmonellosis

All people with HIV and salmonellosis should receive antibiotic treatment because of the increased risk of bacteremia (by 20- to 100-fold) and mortality (by as much as sevenfold) compared with people without HIV (AIII).

For Invasive Disease (Suspected or Confirmed) Pending Susceptibilities

- Ceftriaxone 1–2 g IV every 24 hours pending susceptibilities (BIII)

Preferred Therapy for Nontyphoidal *Salmonella* Gastroenteritis With or Without Bacteremia (If Susceptible)

- Ciprofloxacin 500–750 mg PO (or 400 mg IV) every 12 hours (AIII)

Alternative Therapy (If Susceptible)

- Levofloxacin 750 mg (PO or IV) every 24 hours (BIII), *or*
- Moxifloxacin 400 mg (PO or IV) every 24 hours (BIII), *or*
- Trimethoprim 160 mg/sulfamethoxazole 800 mg (PO or IV) every 12 hours (BIII), *or*
- Ceftriaxone 1–2 g IV every 24 hours (BIII)

Duration of Therapy for Gastroenteritis Without Bacteremia

- If CD4 count ≥200 cells/mm^3: 7–14 days (BIII)
- If CD4 count <200 cells/mm^3, minimum of 2 weeks (may extend to up to 6 weeks if with severe disease) (BIII)

Duration of Therapy for Gastroenteritis With Bacteremia

- If CD4 count ≥200 cells/mm^3: 14 days; longer duration if bacteremia persists or if the infection is complicated (e.g., metastatic foci of infection are present) (BIII)
- If CD4 count <200 cells/mm^3: 2–6 weeks (BIII)

Secondary Prophylaxis

- The role of long-term secondary prophylaxis for patients with recurrent bacteremia or gastroenteritis is not well established. Clinicians must weigh the benefit against the risks of long-term antibiotic exposure (BIII). Antibiotic choices for secondary prophylaxis are the same as for primary treatment and are dependent on the sensitivity of the *Salmonella* isolate.
- HIV suppression with ART is expected to decrease the risk of recurrent illnesses.
- Clinicians should be aware that recurrence may represent development of antimicrobial resistance during therapy.

Indication

- Patients with recurrent bacteremia (BIII), *or*
- Patients with recurrent gastroenteritis (with or without bacteremia) with CD4 count <200 cells/mm^3 and severe diarrhea (BIII)

Discontinuing Secondary Prophylaxis

- After resolution of *Salmonella* infection and response to ART with sustained viral suppression and CD4 count >200 cells/mm^3, secondary prophylaxis likely can be discontinued (CII).

Treating Shigellosis

Therapy should be considered because it may slightly shorten the duration of illness and help prevent spread of the infection to others (AIII); however, antibiotic selection should be guided by the results of antibiotic susceptibility testing. Because antimicrobial resistance of *Shigella* spp. is increasing and limited data demonstrate that antibiotic therapy limits transmission, antibiotic treatment may be withheld in people with HIV and CD4 >500 cells/mm^3 whose diarrhea is mild or resolves before culture confirmation of *Shigella* infection (CIII).

In Severely Ill Patients R**equiring** Empiric Parenteral Therapy While Awaiting Susceptibility

- Initiate a carbapenem until antimicrobial susceptibilities are available (BIII).

Preferred Therapy (If Susceptible)

- Ciprofloxacin 500–750 mg PO (or 400 mg IV) every 12 hours if MIC <0.12 μg/mL for 5–10 days (AIII)

Alternative Therapy (If Susceptible)

- Levofloxacin 750 mg (PO or IV) every 24 hours if MIC <0.12 ug/mL for 5–10 days (BIII), *or*
- Trimethoprim 160 mg/sulfamethoxazole 800 mg PO or IV every 12 hours for 5–7 days (BIII), *or*
  - Note: TMP-SMX is not recommended for bacteremia.
- Azithromycin 500 mg PO daily for 5 days (BIII), *or*
  - Note: Azithromycin is not recommended for bacteremia (AIII).
- Ceftriaxone 1–2 g IV every 24 hours (BIII)

Duration of Therapy

- Gastroenteritis: 5–7 days (AIII) (except ciprofloxacin [5 to 10 days] and azithromycin [5 days])
  - 7–10 days of therapy may be reasonable in patients who are severely immunosuppressed with poor clinical response to antibiotics.
- Bacteremia: ≥14 days (BIII)
- Recurrent infections: up to 6 weeks (BIII)

Chronic Maintenance or Suppressive Therapy

- Not recommended for first-time *Shigella* infections (BIII)

Treating Campylobacteriosis

- Optimal treatment is poorly defined, and multidrug resistance may occur.
- Antimicrobial therapy should be modified based on susceptibility reports.

Mild Disease If CD4 Count >200 cells/mm^3

- If diarrhea resolves before culture confirmation of *Campylobacter* infection, antibiotic treatment can be withheld (CIII). If symptoms persist for more than several days, consider antibiotic therapy (CIII).

Mild-to-Moderate Disease

- *Preferred Therapy (If Susceptible)*
  - Azithromycin 500 mg PO daily for 5 days (BIII) (not recommended for bacteremia [AIII]), *or*
  - Ciprofloxacin[b] 500–750 mg PO (or 400 mg IV) every 12 hours for 7–10 days (BIII) (if susceptible)
- *Alternative Therapy (If Susceptible)*
  - Levofloxacin[c] 750 mg PO or IV every 24 hours (BIII)

Bacteremia

- Ciprofloxacin[a] 500–750 mg PO (or 400 mg IV) every 12 hours for ≥14 days (BIII) (if susceptible) plus an aminoglycoside (BIII) to limit the emergence of antibiotic resistance

Duration of Therapy

- Gastroenteritis: 7–10 days (except azithromycin, which is 5 days) (BIII)
- Bacteremia: ≥14 days (BIII)
- Recurrent disease: 2–6 weeks (BIII)

Chronic Maintenance or Suppressive Therapy

- Not recommended for first-time *Campylobacter* infections (BIII)

[a] The rate of fluoroquinolone resistance in the United States is increasing (29% resistance in 2018 among *C. jejuni* isolates). Third-generation cephalosporins are not reliably active, and use of alternative cell-wall active agents, such as carbapenems, may be necessary in severely ill people requiring empiric parenteral therapy until antimicrobial susceptibilities return.

Treating *Clostridioides difficile*–Associated Infection

Preferred Therapy (Severe or Nonsevere CDI[c])

- Fidaxomicin 200 mg PO two times per day for 10 days (AI)

Alternative Therapy

- Vancomycin 125 mg PO four times per day for 10 days (AI)
- For severe, life-threatening CDI, see *C. difficile* and references for additional information.

Alternative Therapy for Nonsevere CDI[b]

- If neither fidaxomicin nor vancomycin is available: metronidazole 500 mg PO three times per day for 10 days (CI)

Note: Based on clinical trials, vancomycin is superior to metronidazole for therapy of CDI (discussed in text).

Recurrent CDI

- Use of fidaxomicin over oral vancomycin is recommended, in agreement with the 2021 IDSA CDI Guidelines, as it has a greater likelihood for a sustained clinical response at 30 days (AI).
- Vancomycin is an acceptable option (see IDSA Guideline for tapered and pulsed regimens) (AI).
- In patients with CD4 counts >200 cells/mm^3 , FMT may be considered after three CDI episodes (i.e., an initial and two recurrent episodes) (CIII).

[b] Severe CDI: white blood cell count ≥15,000 cells/mL or serum creatinine concentrations >1.5 mg/dL; nonsevere CDI: white blood cell count <15,000 cells/mL and serum creatinine concentrations <1.5 mg/dL

Treating Bacterial Enteric Infections During Pregnancy

- Based on their safety profile, expanded-spectrum cephalosporins (such as ceftriaxone and cefotaxime) or azithromycin should be the first-line therapy for bacterial enteric infections during pregnancy if antimicrobials are required, depending on the organism and the results of susceptibility testing (BIII).
- Other commonly prescribed antimicrobials during pregnancy include vancomycin and metronidazole. Fidaxomicin, quinolones, and TMP-SMX should be prescribed using shared decision-making.
- Quinolones can be used for bacterial enteric infections during pregnancy if indicated by susceptibility testing or failure of first-line therapy (BIII).
- TMP-SMX use in the first trimester should be avoided, if possible, because of an association with an increased risk of birth defects, specifically neural tube, cardiovascular, and urinary tract defects (BIII). As soon as possible during the first trimester of pregnancy, or when anticipating or attempting conception, any administration of TMP-SMX should be supplemented with 4 mg/day of folic acid (BIII).

Key: ART = antiretroviral therapy; CD4 = CD4 T lymphocyte; CDI = *Clostridioides difficile*–associated infection; FMT = fecal microbiota therapy; IDSA = Infectious Diseases Society of America; IV = intravenously; MIC = minimum inhibitory concentration; MSM = men who have sex with men; PCR = polymerase chain reaction; PO = orally; TMP-SMX = trimethoprim-sulfamethoxazole

Empiric Therapy

In most situations, treatment of diarrheal disease in people with HIV does not differ significantly from that in immunocompetent individuals. Decisions on therapy are based on an assessment of diarrhea severity and hydration status. Patients should be informed of the importance of maintaining hydration and be given oral or intravenous (IV) rehydration, if indicated **(AIII).** Because diarrheal disease can produce temporary malabsorption or lactose intolerance, consuming a bland diet and avoiding fat, dairy, and complex carbohydrates are likely to be useful and are therefore recommended **(BIII).** The effectiveness and safety of probiotics or antimotility agents have not been studied adequately in people with HIV who have diarrheal illnesses.[44] Antimotility agents should be avoided if concern about inflammatory diarrhea, including CDI, exists **(BIII).**

After obtaining stool samples for diagnostic evaluation, the initiation and duration of empiric antimicrobial therapy depend on the patient's CD4 count and clinical appearance. If stool samples are obtained, antibiotic susceptibility testing should be performed to confirm and inform antibiotic choice. For example, in patients with CD4 counts >500 cells/mm^3 who have had 1 to 2 days of loose stools without fever or blood, no further work-up and no treatment other than oral rehydration may be required. However, a short course of antibiotics (e.g., ciprofloxacin for 5 days **[BIII]**) may be indicated in people with HIV and CD4 counts of 200 cells/mm^3 to 500 cells/mm^3 who have diarrhea severe enough to compromise quality of life or ability to work. Patients with severe disease (advanced HIV disease [i.e., CD4 counts <200 cells/mm^3 or concomitant AIDS-defining illness] and clinically severe diarrhea [i.e., ≥6 liquid stools per day or bloody stools or a lower number of liquid stools per day but accompanied by fever or chills concerning for invasive bacterial disease]) should undergo inpatient diagnostic evaluation to determine the etiology of the diarrheal illness and receive parenteral antimicrobial treatment **(AIII).** In stable patients, empiric therapy with oral ciprofloxacin **(BIII)** or azithromycin **(BIII)** is recommended, particularly if the infection is not associated with international travel. However, even in the United States, all patients should have careful follow-up since rates of resistance to ciprofloxacin and (to a lesser extent) azithromycin in common enteric pathogens are substantial, and therefore treatment failure may occur. In patients with severe disease, treatment with empiric IV ceftriaxone is recommended until antimicrobial susceptibility results are available **(BIII).** Given the rise of antimicrobial resistance in enteric pathogens, however, updated outbreak information, local susceptibility patterns and travel history always should be considered. For example, if *Campylobacter* or *Shigella* bacteremia is suspected, a carbapenem is preferred for empiric therapy **(BIII).**

Therapy should be adjusted based on the results of the diagnostic work-up. For diarrhea that is persistent (i.e., lasting >14 days) in the absence of other clinical signs of severity—such as bloody stool or dehydration—antibiotic therapy can be withheld and directed therapy initiated once a diagnosis is confirmed. Noninfectious etiologies of persistent diarrhea (e.g., inflammatory bowel disease) also should be considered in the differential diagnosis **(BIII).**

International travel: Diarrhea is one of the most common illnesses affecting international travelers. Antimicrobial resistance among enteric bacterial pathogens outside the United States is an important public health problem. For example, traveler's diarrhea caused by fluoroquinolone-resistant *C. jejuni* in South and Southeast Asia or Africa is common.[45,46] Clinicians should consider the possibility of a resistant infection when prescribing empiric therapy for travelers with HIV who experience diarrhea or a syndrome consistent with a systemic infection while traveling or upon returning to the United States, given reports of multidrug-resistant *Enterobacteriaceae* acquisition during travel.[47-51]

Pathogen-Specific Therapy

Nontyphoidal *Salmonella* Species

Immunocompetent hosts who do not have HIV often do not require antibiotic treatment for *Salmonella* gastroenteritis (typically caused by nontyphoidal *Salmonella* spp.) because the condition is usually self-limited, and treatment may prolong the carrier state. In contrast, all people with HIV and salmonellosis should be treated **(AIII),** even though no clinical trials have compared antimicrobial therapy with placebo. Notably, HIV infection increases the risk of *Salmonella* bacteremia 20 to 100 times and mortality as much as seven times compared to people who do not have HIV.[25,52]

The treatment of choice for susceptible nontyphoidal *Salmonella* spp. infection is a fluoroquinolone **(AIII).** Ciprofloxacin is the preferred agent **(AIII).**[53] Other fluoroquinolones—such as levofloxacin and moxifloxacin—are recommended as alternatives to ciprofloxacin **(BIII).** Although they have not been well evaluated in clinical trials, they likely would be effective in treating salmonellosis in people with HIV. Depending on antibiotic susceptibility, alternatives to the fluoroquinolones include TMP-SMX or expanded-spectrum cephalosporins, such as ceftriaxone **(BIII).** Fluoroquinolone resistance in nontyphoidal *Salmonella* spp. appears to be increasing, with preliminary CDC data showing genetic markers of fluoroquinolone resistance among 20% of 39,065 nontyphoidal *Salmonella* spp. isolates tested in the United States in 2024.[10] In agreement with IDSA guidelines, the Panel on Guidelines for the Prevention and Treatment of Opportunistic Infections in Adults and Adolescent With HIV (the Panel) recommends ceftriaxone over ciprofloxacin if invasive disease is suspected or confirmed, at least until susceptibilities return **(BIII).**[10,53]

The optimal duration of therapy for HIV-related nontyphoidal *Salmonella* infection has not been defined. For patients with CD4 counts ≥200 cells/mm^3 who have mild gastroenteritis without bacteremia, 7 to 14 days of treatment is recommended **(BIII).** For the same patients with bacteremia, 14 days is appropriate provided clearance of bacteremia is documented. Longer treatment is recommended if bacteremia persists or if the infection is complicated (i.e., if metastatic foci are present) **(BIII).**

For any patients with advanced HIV disease (CD4 count <200 cells/mm^3) and *Salmonella* infection, a minimum of 2 weeks of antibiotics, with extension up to 6 weeks in severe disease or bacteremia, is often recommended **(BIII).**[54]

People with HIV and *Salmonella* bacteremia, which typically occurs in those with advanced HIV disease, should be monitored clinically for recurrence after treatment **(BIII).** As people with HIV age, it is also important to remember that rates of invasive *Salmonella* infections increase with age in each age group beyond infancy.[55,56] Recurrence may present as bacteremia or as an anatomically localized infection, including intra-abdominal, endothelial, urinary tract, soft tissue, bone and joint, lung, or meningeal foci. Secondary prophylaxis should be considered for patients with recurrent *Salmonella* bacteremia **(BIII),** and it also might be considered for patients with recurrent gastroenteritis (with or without bacteremia) and in those with CD4 counts <200 cells/mm^3 with severe diarrhea **(BIII).** The value of this secondary prophylaxis has not been established and must be weighed against the risks of long-term antibiotic exposure. Recurrent *Salmonella* bacteremia constitutes an AIDS-defining illness,[37] and HIV suppression with ART appears to decrease the risk of recurrent illnesses.

In patients whose *Salmonella* infection is resolved and who have responded to ART with sustained viral suppression and CD4 counts >200 cells/mm^3, secondary prophylaxis for salmonellosis likely

can be discontinued **(CII).** Clinicians also should be aware that recurrence may indicate development of antimicrobial resistance during therapy.

Shigella Species

Therapy for *Shigella* infections should be considered because it may slightly shorten the duration of illness and help prevent transmission to others **(AIII);** however, because antimicrobial resistance of *Shigella* spp. is increasing and limited data demonstrate that antibiotic therapy limits transmission, antibiotic treatment may be withheld in people with HIV and CD4 >500 cells/mm^3 with mild symptoms or whose diarrhea is resolving before culture confirmation of *Shigella* infection **(CIII).** When treatment is offered, antibiotic selection should be guided by the results of antibiotic susceptibility testing.[49,57-60]

Preferred treatment for susceptible shigellosis is a fluoroquinolone, preferably ciprofloxacin, for 5 to 10 days **(AIII)** with levofloxacin serving as an alternative **(BIII).** Importantly, preliminary CDC data estimate 66% of *Shigella* spp. isolated among the general U.S. population in 2024 harbored genetic markers of resistance to ciprofloxacin.[10] Although current Clinical and Laboratory Standards Institute criteria categorize *Shigella* isolates with a ciprofloxacin minimum inhibitory concentration (MIC) of 0.12 µg/mL and 0.25 µg/mL as susceptible and a MIC of 0.5 µg/mL as intermediate, these isolates typically harbor a fluoroquinolone resistance gene or mutation. Until the clinical significance of these findings can be determined, alternative antibiotics should be considered to treat patients whose isolates have ciprofloxacin MICs ≥0.12 µg/mL **(BIII).**[61] In general, automated antimicrobial susceptibility test panels do not have doubling dilutions that span the MIC range to determine susceptibility to ciprofloxacin based on the CDC recommendation of ≤0.06. As such, a clinically validated manual antimicrobial susceptibility testing method, such as reference broth microdilution, or a gradient diffusion method would be required to confirm susceptibility at the lower MIC range. Ciprofloxacin-resistant *S. sonnei* and *S. flexneri* infections in the United States are associated with international travel, homelessness, and men who have sex with men (MSM); ciprofloxacin-resistant shigellosis among MSM appears to be acquired predominantly within the United States rather than during travel.[49]

Depending on antibiotic susceptibilities, in stable patients without concern for bacteremia, azithromycin (5 days) or TMP-SMX (5–7 days) may be alternatives **(BIII).** Azithromycin has not been evaluated in people with HIV and shigellosis, and the therapy suggested is extrapolated from limited data in immunocompetent hosts.[61] Azithromycin susceptibility testing is not widely available in clinical laboratories but can be performed by many state public health laboratories. Preliminary CDC data estimate 32% of *Shigella* spp. isolated among the general U.S. population in 2024 harbored genetic markers of resistance to azithromycin.[10] Azithromycin-resistant *Shigella* spp. infections in MSM with HIV have been reported.[62-64]

Multidrug resistance is common among shigellae, and clinicians should be aware that rates of infections caused by extensively drug-resistant *Shigella* strains (strains resistant to azithromycin, ciprofloxacin, ceftriaxone, trimethoprim-sulfamethoxazole, and ampicillin) are increasing in the United States and abroad.[13,65] Therefore, while IV ceftriaxone is recommended therapy for susceptible *Shigella*, in severely ill people requiring empiric parenteral therapy, carbapenems can be initiated before antimicrobial susceptibilities are available **(BIII).**

Treatment for people with *Shigella* bacteremia is less well defined, but extending treatment to at least 14 days is recommended **(BIII).** Azithromycin **is not recommended** for treatment of *Shigella* spp. bacteremia **(AIII).** Chronic suppressive or maintenance therapy **is not recommended** for first-time *Shigella* infections **(BIII).** Recurrent infections can occur, particularly in individuals with CD4

counts <200 cells/mm^3, in which case, extending antimicrobial therapy for up to 6 weeks is recommended **(BIII).** Because of *Shigella's* extremely low infectious dose, patients with shigellosis should be counseled about transmission prevention. As with *Salmonella* infections, suppression of HIV replication with ART is expected to decrease the risk of recurrent shigellosis.

Campylobacter Species

The optimal treatment of campylobacteriosis in people with HIV is poorly defined, and multidrug resistance might occur.[66,67] Culture and testing for the antibiotic susceptibility of *Campylobacter* isolates is recommended **(BIII).** In the United States in 2017 and 2018, 28% of *C. jejuni* isolates were resistant to ciprofloxacin, 2.4% were resistant to erythromycin, and 1.4% were resistant to ciprofloxacin, erythromycin, and drugs from at least three of the five remaining classes (extensively drug-resistant [XDR]); among *C. coli* isolates, 40% of isolates were resistant to ciprofloxacin, 11% were resistant to erythromycin, and 7.8% were XDR.[9]

For people with mild disease and CD4 counts >200 cells/mm^3, therapy should be withheld unless symptoms persist for more than several days **(CIII).** For mild-to-moderate campylobacteriosis, initiating therapy with azithromycin for 5 days or a fluoroquinolone—such as ciprofloxacin—for 7 to 10 days (if the organism is sensitive) is recommended **(BIII).** Azithromycin has not been evaluated in people with HIV and campylobacteriosis, and the therapy suggested is extrapolated from limited data in immunocompetent hosts.[45] Azithromycin susceptibility testing, however, is not widely available in clinical laboratories but can be performed by many state public health laboratories. *Campylobacter* bacteremia should be treated for at least 14 days using a fluoroquinolone if the isolate is sensitive **(BIII).**[68] Adding a second active agent—such as an aminoglycoside—may be prudent in patients with bacteremia to limit the emergence of antibiotic resistance **(BIII).** Third-generation cephalosporins are not reliably active, and use of alternative cell-wall active agents, such as carbapenems, may be necessary in severely ill people requiring empiric parenteral therapy until antimicrobial susceptibilities return. Antibiotic choice should be guided by antibiotic susceptibility tests. Azithromycin **is not recommended** for treatment of *Campylobacter* bacteremia **(AIII).** Chronic suppressive or maintenance therapy **is not recommended** for first-time *Campylobacter* infections in people with HIV **(BIII).** However, recurrent infections can occur, particularly in people with CD4 counts <200 cells/mm^3. In recurrent disease, extending the length of antimicrobial therapy for 2 to 6 weeks is reasonable **(BIII).** As with *Salmonella* infections, suppression of HIV replication with ART is expected to decrease the risk of recurrent *Campylobacter* spp. infections.[69]

Clostridioides difficile

No randomized controlled trials have been conducted for CDI therapy in people with HIV. Available data suggest that people with HIV respond to treatment of CDI similarly to people without HIV.[14] Thus, treatment of CDI in people with HIV is the same as in people without HIV. Guidelines and subsequent updates for treatment of CDI have been published[35,70] and should be consulted for further information.

Treatment of an Initial Episode of Clostridioides difficile*–Associated Infection*

Four randomized clinical trials, all conducted in the general population (two identical studies with approximately 60% hospitalized patients; two studies restricted to hospitalized patients),[71-74] have revealed that, when compared with oral vancomycin, fidaxomicin increased the likelihood of a sustained clinical response of CDI (at 28 days) in the initial therapy of CDI (relative risk [RR] 1.16; 95% confidence interval [CI], 1.09–1.24).[70] Fidaxomicin was equivalent to oral vancomycin in initial clinical cure, serious adverse events, and all-cause mortality. Given these data, the 2021 IDSA CDI

Clinical Practice Guideline update[70] for adults suggests treatment with fidaxomicin rather than oral vancomycin for initial CDI, whether CDI is severe or nonsevere. Fidaxomicin remains very expensive but should be considered in people with HIV and CDI, if available **(AI).** Oral vancomycin is also an acceptable option for initial CDI **(AI).** Earlier multicenter, randomized, double-blind studies identified that oral vancomycin is superior to metronidazole for treatment of CDI.[75,76] Thus, metronidazole is to be considered as an alternative drug for CDI therapy only if fidaxomicin or vancomycin are unavailable and CDI is nonsevere (white blood cell count <15,000 cells/mL and serum creatinine concentrations <1.5 mg/dL) **(CI).**[35]

Treatment of Recurrent Clostridioides difficile–Associated Infection

Treatment of recurrent CDI is complex and, in part, defined by the specific circumstances of the patient with recurrent CDI and the number of prior CDI episodes. Brief guidance is provided here; the 2017 and 2021 IDSA CDI guidelines should be consulted for a full discussion of this topic.[35,70] Risk factors for CDI recurrence are age ≥65 years, history of CDI, compromised immunity, severe CDI, and certain virulent strains (ribotypes 027/078/244). Similar to an initial episode of CDI, and also based on the randomized clinical trials cited above,[71-74] the Panel recommends administering fidaxomicin instead of oral vancomycin to adults with recurrent CDI **(AI),** consistent with the 2021 IDSA CDI Clinical Practice Guideline update.[70] Fidaxomicin therapy increased the likelihood of a sustained clinical response for recurrent CDI at 30 days (RR 1.27; 95% CI, 1.05–1.54). For treatment of an initial CDI recurrence, fidaxomicin was equivalent to oral vancomycin in initial clinical cure, serious adverse events, and all-cause mortality. Vancomycin is also an acceptable option for recurrent CDI (see the IDSA Guideline for tapered and pulsed regimens) **(AI).**

Bezlotoxumab is a humanized monoclonal antibody against *C. difficile* toxin B approved for prevention of recurrent CDI in high-risk adults when used in conjunction with standard-of-care (SOC) antibiotic therapy. The 2021 IDSA CDI Clinical Practice Guideline update suggests use of bezlotoxumab as a cointervention, along with vancomycin as the SOC antibiotic, in patients with a history of CDI in the last 6 months or other risk factors for recurrence (i.e., age ≥65 years, compromised immunity, severe CDI, or certain virulent strains [ribotypes 027/078/244]).[70] However, data on the benefit of bezlotoxumab therapy when fidaxomicin is used as the SOC antibiotic are limited. Limited case reports suggest that fecal microbiota therapy (FMT) (i.e., fecal transplant) may be successful and safe for treating recurrent CDI in people with HIV.[77-79] However, it is important to note that complications of FMT, including transmission of enteric pathogens and antibiotic-resistant bacteria with deaths, have been reported.[80,81] In patients with CD4 counts >200 cells/mm^3, FMT for treatment of recurrent CDI may be considered after three total CDI episodes (initial and two recurrent CDI episodes) **(CIII).**[35,70] The effect of ART on recurrence of CDI is unknown, but ART initiation should follow standard guidelines, similar to other enteric infections (see the Special Considerations Regarding ART Initiation section below).

Special Considerations Regarding ART Initiation

ART initiation should follow standard guidelines. The presence of an enteric infection should not delay ART initiation **(AIII).** The presence of a diarrheal illness is relevant only in terms of a patient's ability to ingest and absorb ART. If recurrent enteric infections are documented or *Salmonella* bacteremia occurs, prompt initiation of ART should be considered regardless of CD4 count.

Monitoring of Response to Therapy and Adverse Events (Including Immune Reconstitution Inflammatory Syndrome)

Patients should be monitored closely for response to treatment, defined clinically by improvement in systemic signs and symptoms, resolution of diarrhea, and sterilization of infected tissues or body fluids, such as blood. Follow-up stool testing may be required when public health considerations and state policies dictate the need to ensure microbiologic cure, such as in health care or food service workers. Follow-up stool culture and antibiotic susceptibility testing should be considered for patients with incomplete clinical response to appropriate antimicrobial therapy. In patients with persistent or recurrent diarrhea despite therapy, clinicians should consider other enteric infections (including STIs; see the Diagnosis section above) in the context of the patient's immune status and exposures, as well as the possibility of *C. difficile* or the development of antimicrobial resistance **(BIII).**

Observational studies suggest that plasma drug concentrations in people with HIV may be decreased as a result of severe diarrhea or malabsorption.[82,83] Coadministration of fluoroquinolones with magnesium- or aluminum-containing antacids or with calcium, zinc, or iron should be avoided because these agents interfere with fluoroquinolone absorption **(AII).**[84] Although larger prospective studies are needed to determine the impact of severe diarrhea on antibiotic absorption, it is prudent to use IV antibiotics in clinically unstable patients **(AIII).**

Immune reconstitution inflammatory syndrome has not been described in association with treatment for typical bacterial enteric pathogens.

Preventing Recurrence

The pharmacologic approach to recurrent enteric infections is covered in the section on directed therapy for each bacterial species. As noted above, secondary prophylaxis should be considered for patients with recurrent *Salmonella* bacteremia **(BIII)** and, in some circumstances, for those with recurrent shigellosis **(BIII)** or campylobacteriosis **(BIII).**

Special Considerations During Pregnancy

During pregnancy, the diagnosis and management of bacterial enteric infections are the same as for other people with HIV, with several considerations. **Based on their safety profile, expanded-spectrum cephalosporins or azithromycin should be the first-line therapy for bacterial enteric infections during pregnancy if antimicrobials are required, depending on the organism and the results of susceptibility testing (BIII).**[85] Arthropathy has been noted in the offspring of animals treated with quinolones during pregnancy. However, studies evaluating quinolone use during pregnancy did not find an increased risk of birth defects or musculoskeletal abnormalities.[86-88] Thus, quinolones can be used for bacterial enteric infections if indicated by susceptibility testing or failure of first-line therapy, as listed above with a shared medical decision-making decision model in discussion with the patient **(BIII).** TMP-SMX use in the first trimester should be avoided, if possible, because of an association with an increased risk of birth defects, specifically neural tube, cardiovascular, and urinary tract defects **(BIII).**[89-91] However, a review of potential risks related to TMP-SMX use cites the low quality of current data and supports the use of TMP-SMX during pregnancy as clinically indicated.[92] As soon as possible during the first trimester of pregnancy, or when anticipating or attempting conception, any administration of TMP-SMX should be supplemented with folic acid 4 mg/day **(BIII).**[89,90,93] Neonatal care providers should be informed if maternal sulfa therapy was used near delivery because of the theoretical increased risk of

hyperbilirubinemia and kernicterus in the newborn. Because oral rifaximin and fidaxomicin are not absorbed systemically, they can be used during pregnancy. However, this should be a shared decision made between providers and patients ensuring that patients are made aware of the limited data about fidaxomicin in pregnancy **(BIII).**

Vancomycin and metronidazole are two antimicrobials that have been utilized in the perinatal period in the United States. Intravenous vancomycin has been utilized as intrapartum prophylaxis in the penicillin allergic patient colonized with group B streptococcus,[94] and minimal absorption is expected with oral therapy. Although vancomycin for enteric disease is recommended for use only in its oral formulation, which is not absorbed in meaningful concentrations from the gastrointestinal tract,[95] it should be noted that with IV use, vancomycin readily crosses the placenta.[96] A study of 10 infants evaluated after the second or third trimester for *in utero* exposure of maternal IV vancomycin therapy for serious staphylococcal infections found no hearing loss or renal toxicity attributed to vancomycin.[96] A review of metronidazole use in pregnancy for treatment of trichomoniasis or bacterial vaginosis found no increase in risk of birth defects.[97] Studies on the use of metronidazole for CDI in pregnancy were not found.

References

1. Sanchez TH, Brooks JT, Sullivan PS, et al. Bacterial diarrhea in persons with HIV infection, United States, 1992–-2002. *Clin Infect Dis*. 2005;41(11):1621-1627. Available at: http://www.ncbi.nlm.nih.gov/pubmed/16267735.

2. Newman KL, Newman GS, Cybulski RJ, Fang FC. Gastroenteritis in men who have sex with men in Seattle, Washington, 2017–-2018. *Clin Infect Dis*. 2020;71(1):109-115. Available at: https://www.ncbi.nlm.nih.gov/pubmed/31621824.

3. Verma A, Hine AM, Joelson A, et al. The influence of hospitalization and HIV severity on gastrointestinal PCR panel evaluation of HIV-related acute diarrhea in New York City: a retrospective, cross-sectional study. *Therap Adv Gastroenterol*. 2022;15:17562848221092593. Available at: https://www.ncbi.nlm.nih.gov/pubmed/35509422.

4. Chou YJ, Lin HW, Yang CJ, et al. Risk of recurrent nontyphoid Salmonella bacteremia in human immunodeficiency virus-infected patients with short-term secondary prophylaxis in the era of combination antiretroviral therapy. *J Microbiol Immunol Infect*. 2015. Available at: http://www.ncbi.nlm.nih.gov/pubmed/26316009.

5. Hung CC, Hung MN, Hsueh PR, et al. Risk of recurrent nontyphoid Salmonella bacteremia in HIV-infected patients in the era of highly active antiretroviral therapy and an increasing trend of fluoroquinolone resistance. *Clin Infect Dis*. 2007;45(5):e60-67. Available at: http://www.ncbi.nlm.nih.gov/pubmed/17682981.

6. Taramasso L, Tatarelli P, Di Biagio A. Bloodstream infections in HIV-infected patients. *Virulence*. 2016;7(3):320-328. Available at: https://www.ncbi.nlm.nih.gov/pubmed/26950194.

7. Huang DB, Mohanty A, DuPont HL, Okhuysen PC, Chiang T. A review of an emerging enteric pathogen: enteroaggregative Escherichia coli. *J Med Microbiol*. 2006;55(Pt 10):1303-1311. Available at: http://www.ncbi.nlm.nih.gov/pubmed/17005776.

8. Nevin DT, Morgan CJ, Graham DY, Genta RM. Helicobacter pylori gastritis in HIV-infected patients: a review. *Helicobacter*. 2014;19(5):323-329. Available at: https://www.ncbi.nlm.nih.gov/pubmed/24773336.

9. Ford L, Healy JM, Cui Z, et al. Epidemiology and antimicrobial resistance of Campylobacter infections in the United States, 2005–-2018. *Open Forum Infect Dis*. 2023;10(8):ofad378. Available at: https://pubmed.ncbi.nlm.nih.gov/37559755/.

10. Centers for Disease Control and Prevention. BEAM (Bacteria, Enterics, Ameba, and Mycotics) Dashboard. Atlanta, Georgia: U.S. Department of Health and Human Services. 2025. Available at: https://www.cdc.gov/ncezid/dfwed/BEAM-dashboard.html

11. Centers for Disease Control and Prevention. High blood lead levels in children consuming recalled cinnamon applesauce pouches. 2023. Available at: https://www.cdc.gov/han/2023/han00500.html

12. O'Flanagan H, Siddiq M, Llewellyn C, Richardson D. Antimicrobial resistance in sexually transmitted Shigella in men who have sex with men: a systematic review. *Int J STD AIDS*. 2023;34(6):374-384. Available at: https://pubmed.ncbi.nlm.nih.gov/36786731/.

13. Mason LCE, Greig DR, Cowley LA, et al. The evolution and international spread of extensively drug resistant Shigella sonnei. *Nat Commun*. 2023;14(1):1983. Available at: https://pubmed.ncbi.nlm.nih.gov/37031199/.

14. Revolinski SL, Munoz-Price LS. Clostridium difficile in immunocompromised hosts: a review of epidemiology, risk factors, treatment, and prevention. *Clin Infect Dis*. 2019;68(12):2144-2153. Available at: https://www.ncbi.nlm.nih.gov/pubmed/30281082.

15. Haines CF, Moore RD, Bartlett JG, et al. Clostridium difficile in a HIV-infected cohort: incidence, risk factors, and clinical outcomes. *AIDS*. 2013;27(17):2799-2807. Available at: http://www.ncbi.nlm.nih.gov/pubmed/23842125.

16. Angulo FJ, Swerdlow DL. Bacterial enteric infections in persons infected with human immunodeficiency virus. *Clin Infect Dis*. 1995;21 Suppl 1:S84-93. Available at: http://www.ncbi.nlm.nih.gov/pubmed/8547518.

17. Aragon TJ, Vugia DJ, Shallow S, et al. Case-control study of shigellosis in San Francisco: the role of sexual transmission and HIV infection. *Clin Infect Dis*. 2007;44(3):327-334. Available at: http://www.ncbi.nlm.nih.gov/pubmed/17205436.

18. Siddiq M, O'Flanagan H, Richardson D, Llewellyn CD. Factors associated with sexually transmitted Shigella in men who have sex with men: a systematic review. *Sex Transm Infect*. 2023;99(1):58-63. Available at: https://pubmed.ncbi.nlm.nih.gov/36283806/.

19. Mitchell HD, Thomson NR, Jenkins C, et al. Linkage of whole genome sequencing, epidemiological, and clinical data to understand the genetic diversity and clinical outcomes of Shigella flexneri among men who have sex with men in England. *Microbiol Spectr*. 2021;9(3):e0121321. Available at: https://www.ncbi.nlm.nih.gov/pubmed/34908501.

20. Mohan K, Hibbert M, Rooney G, et al. What is the overlap between HIV and shigellosis epidemics in England: further evidence of MSM transmission? *Sex Transm Infect*. 2018;94(1):67-71. Available at: https://www.ncbi.nlm.nih.gov/pubmed/28490580.

21. Quinn TC, Goodell SE, Fennell C, et al. Infections with Campylobacter jejuni and Campylobacter-like organisms in homosexual men. *Ann Intern Med*. 1984;101(2):187-192. Available at: http://www.ncbi.nlm.nih.gov/pubmed/6547580.

22. Kuhn KG, Hvass AK, Christiansen AH, Ethelberg S, Cowan SA. Sexual contact as risk factor for Campylobacter infection, Denmark. *Emerg Infect Dis*. 2021;27(4):1133-1140. Available at: https://www.ncbi.nlm.nih.gov/pubmed/33754996.

23. Tee W, Mijch A. Campylobacter jejuni bacteremia in human immunodeficiency virus (HIV)-infected and non-HIV-infected patients: comparison of clinical features and review. *Clin Infect Dis*. 1998;26(1):91-96. Available at: http://www.ncbi.nlm.nih.gov/pubmed/9455515.

24. Tobin-D'Angelo M, Oosmanally N, Wilson SN, Anderson EJ, Segler S, Poventud L. Shigella bacteremia, Georgia, USA, 2002-2012(1). *Emerg Infect Dis*. 2020;26(1):122-124. Available at: https://www.ncbi.nlm.nih.gov/pubmed/31855540.

25. Celum CL, Chaisson RE, Rutherford GW, Barnhart JL, Echenberg DF. Incidence of salmonellosis in patients with AIDS. *J Infect Dis*. 1987;156(6):998-1002. Available at: http://www.ncbi.nlm.nih.gov/pubmed/3680999.

26. Kristjansson M, Viner B, Maslow JN. Polymicrobial and recurrent bacteremia with Shigella in a patient with AIDS. *Scand J Infect Dis*. 1994;26(4):411-416. Available at: http://www.ncbi.nlm.nih.gov/pubmed/7984973.

27. Casado JL, Valdezate S, Calderon C, et al. Zidovudine therapy protects against Salmonella bacteremia recurrence in human immunodeficiency virus-infected patients. *J Infect Dis*. 1999;179(6):1553-1556. Available at: https://www.ncbi.nlm.nih.gov/pubmed/10228081.

28. Williamson DA, Chen MY. Emerging and reemerging sexually transmitted infections. *N Engl J Med*. 2020;382(21):2023-2032. Available at: https://www.ncbi.nlm.nih.gov/pubmed/32433838.

29. Thornhill JP, Barkati S, Walmsley S, et al. Monkeypox virus infection in humans across 16 countries - April-June 2022. *N Engl J Med*. 2022;387(8):679-691. Available at: https://www.ncbi.nlm.nih.gov/pubmed/35866746.

30. Arnold CA, Limketkai BN, Illei PB, Montgomery E, Voltaggio L. Syphilitic and lymphogranuloma venereum (LGV) proctocolitis: clues to a frequently missed diagnosis. *Am J Surg Pathol*. 2013;37(1):38-46. Available at: https://www.ncbi.nlm.nih.gov/pubmed/23095509.

31. Huang JY, Henao OL, Griffin PM, et al. Infection with pathogens transmitted commonly through food and the effect of increasing use of culture-independent diagnostic tests on surveillance--Foodborne Diseases Active Surveillance Network, 10 U.S. sites, 2012-2015. *MMWR Morb Mortal Wkly Rep*. 2016;65(14):368-371. Available at: https://www.ncbi.nlm.nih.gov/pubmed/27077946.

32. Pulvirenti JJ, Mehra T, Hafiz I, et al. Epidemiology and outcome of Clostridium difficile infection and diarrhea in HIV infected inpatients. *Diagn Microbiol Infect Dis*. 2002;44(4):325-330. Available at: http://www.ncbi.nlm.nih.gov/pubmed/12543536.

33. Kociolek LK, Gerding DN, Carrico R, et al. Strategies to prevent *Clostridioides difficile* infections in acute-care hospitals: 2022 update. *Infect Control Hosp Epidemiol*. 2023;44(4):527-549. Available at: https://www.ncbi.nlm.nih.gov/pubmed/37042243.

34. Mizusawa M, Carroll KC. The future of *Clostridioides difficile* diagnostics. *Curr Opin Infect Dis*. 2021;34(5):483-490. Available at: https://www.ncbi.nlm.nih.gov/pubmed/34524199.

35. McDonald LC, Gerding DN, Johnson S, et al. Clinical practice guidelines for Clostridium difficile infection in adults and children: 2017 update by the Infectious Diseases Society of America (IDSA) and Society for Healthcare Epidemiology of America (SHEA). *Clin Infect Dis*. 2018;66(7):987-994. Available at: https://www.ncbi.nlm.nih.gov/pubmed/29562266.

36. Workowski KA, Bachmann LH, Chan PA, et al. Sexually transmitted infections treatment guidelines, 2021. *MMWR Recomm Rep*. 2021;70(4):1-187. Available at: https://www.ncbi.nlm.nih.gov/pubmed/34292926.

37. Schneider E, Whitmore S, Glynn KM, et al. Revised surveillance case definitions for HIV infection among adults, adolescents, and children aged <18 months and for HIV infection and AIDS among children aged 18 months to <13 years--United States, 2008. *MMWR Recomm Rep*. 2008;57(RR-10):1-12. Available at: http://www.ncbi.nlm.nih.gov/pubmed/19052530.

38. de Vries HJC, Nori AV, Kiellberg Larsen H, et al. 2021 European guideline on the management of proctitis, proctocolitis and enteritis caused by sexually transmissible pathogens. *J Eur Acad Dermatol Venereol*. 2021;35(7):1434-1443. Available at: https://www.ncbi.nlm.nih.gov/pubmed/34057249.

39. Centers for Disease Control and Prevention. Healthcare resources. 2024. Available at: https://www.cdc.gov/c-diff/hcp/resources/?CDC_AAref_Val=https://www.cdc.gov/cdiff/clinicians/resources.html#cdc_listing_res2-resources

40. Advice for travelers. *Med Lett Drugs Ther*. 2019;61(1582):153-160. Available at: https://www.ncbi.nlm.nih.gov/pubmed/31599872.

41. Riddle MS, Connor BA, Beeching NJ, et al. Guidelines for the prevention and treatment of travelers' diarrhea: a graded expert panel report. *J Travel Med*. 2017;24(suppl_1):S57-S74. Available at: https://www.ncbi.nlm.nih.gov/pubmed/28521004.

42. Diptyanusa A, Ngamprasertchai T, Piyaphanee W. A review of antibiotic prophylaxis for traveler's diarrhea: past to present. *Trop Dis Travel Med Vaccines*. 2018;4:14. Available at: https://www.ncbi.nlm.nih.gov/pubmed/30455974.

43. Food and Drug Administration. FDA Drug Safety Communication: FDA updates warnings for oral and injectable fluoroquinolone antibiotics due to disabling side effects. 2018. Available at: https://www.fda.gov/drugs/drug-safety-and-availability/fda-drug-safety-communication-fda-updates-warnings-oral-and-injectable-fluoroquinolone-antibiotics.

44. Nwachukwu CE, Okebe JU. Antimotility agents for chronic diarrhoea in people with HIV/AIDS. *Cochrane Database Syst Rev*. 2008(4):CD005644. Available at: http://www.ncbi.nlm.nih.gov/pubmed/18843696.

45. Kuschner RA, Trofa AF, Thomas RJ, et al. Use of azithromycin for the treatment of Campylobacter enteritis in travelers to Thailand, an area where ciprofloxacin resistance is prevalent. *Clin Infect Dis*. 1995;21(3):536-541. Available at: http://www.ncbi.nlm.nih.gov/pubmed/8527539.

46. Tribble DR, Sanders JW, Pang LW, et al. Traveler's diarrhea in Thailand: randomized, double-blind trial comparing single-dose and 3-day azithromycin-based regimens with a 3-day levofloxacin regimen. *Clin Infect Dis*. 2007;44(3):338-346. Available at: http://www.ncbi.nlm.nih.gov/pubmed/17205438.

47. Kantele A, Laaveri T, Mero S, et al. Antimicrobials increase travelers' risk of colonization by extended-spectrum betalactamase-producing Enterobacteriaceae. *Clin Infect Dis*. 2015;60(6):837-846. Available at: http://www.ncbi.nlm.nih.gov/pubmed/25613287.

48. Barlow RS, Debess EE, Winthrop KL, Lapidus JA, Vega R, Cieslak PR. Travel-associated antimicrobial drug-resistant nontyphoidal Salmonellae, 2004-2009. *Emerg Infect Dis*. 2014;20(4):603-611. Available at: http://www.ncbi.nlm.nih.gov/pubmed/24655581.

49. Centers for Disease Control and Prevention. Importation and domestic transmission of *Shigella sonnei* resistant to ciprofloxacin — United States, May 2014–February 2015. *MMWR Morb Mortal Wkly Rep*. 2015;64(12):318-320. Available at: http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6412a2.htm?s_cid=mm6412a2_w.

50. Ruppe E, Andremont A, Armand-Lefevre L. Digestive tract colonization by multidrug-resistant Enterobacteriaceae in travellers: an update. *Travel Med Infect Dis*. 2018;21:28-35. Available at: https://www.ncbi.nlm.nih.gov/pubmed/29155322.

51. Tribble DR. Resistant pathogens as causes of traveller's diarrhea globally and impact(s) on treatment failure and recommendations. *J Travel Med*. 2017;24(suppl_1):S6-S12. Available at: https://www.ncbi.nlm.nih.gov/pubmed/28520997.

52. Cummings PL, Sorvillo F, Kuo T. Salmonellosis-related mortality in the United States, 1990-2006. *Foodborne Pathog Dis*. 2010;7(11):1393-1399. Available at: http://www.ncbi.nlm.nih.gov/pubmed/20617938.

53. Shane AL, Mody RK, Crump JA, et al. 2017 Infectious Diseases Society of America clinical practice guidelines for the diagnosis and management of infectious diarrhea. *Clin Infect Dis*. 2017;65(12):e45-e80. Available at: https://www.ncbi.nlm.nih.gov/pubmed/29053792.

54. Gordon MA, Banda HT, Gondwe M, et al. Non-typhoidal Salmonella bacteraemia among HIV-infected Malawian adults: high mortality and frequent recrudescence. *AIDS*. 2002;16(12):1633-1641. Available at: http://www.ncbi.nlm.nih.gov/pubmed/12172085.

55. Hsu RB, Tsay YG, Chen RJ, Chu SH. Risk factors for primary bacteremia and endovascular infection in patients without acquired immunodeficiency syndrome who have nontyphoid salmonellosis. *Clin Infect Dis*. 2003;36(7):829-834. Available at: https://www.ncbi.nlm.nih.gov/pubmed/12652381.

56. Nielsen H, Gradel KO, Schonheyder HC. High incidence of intravascular focus in nontyphoid Salmonella bacteremia in the age group above 50 years: a population-based study. *APMIS*. 2006;114(9):641-645. Available at: https://www.ncbi.nlm.nih.gov/pubmed/16948817.

57. Eikmeier D, Talley P, Bowen A, et al. Decreased susceptibility to azithromycin in clinical shigella isolates associated with HIV and sexually transmitted bacterial diseases, Minnesota, USA, 2012-2015. *Emerg Infect Dis*. 2020;26(4):667-674. Available at: https://www.ncbi.nlm.nih.gov/pubmed/32186495.

58. Hoffmann C, Sahly H, Jessen A, et al. High rates of quinolone-resistant strains of Shigella sonnei in HIV-infected MSM. *Infection*. 2013;41(5):999-1003. Available at: https://www.ncbi.nlm.nih.gov/pubmed/23852945.

59. Murray K, Reddy V, Kornblum JS, et al. Increasing antibiotic resistance in shigella spp. from infected New York City residents, New York, USA. *Emerg Infect Dis*. 2017;23(2):332-335. Available at: https://www.ncbi.nlm.nih.gov/pubmed/28098543.

60. Gharpure R, Friedman CR, Fialkowski V, et al. Azithromycin and ciprofloxacin treatment outcomes during an outbreak of multidrug-resistant Shigella sonnei infections in a retirement community-Vermont, 2018. *Clin Infect Dis*. 2022;74(3):455-460. Available at: https://www.ncbi.nlm.nih.gov/pubmed/33993224.

61. Centers for Disease Control and Prevention. CDC recommendations for diagnosing and managing Shigella strains with possible reduced susceptibility to Ciprofloxacin. 2017. Available at: https://stacks.cdc.gov/view/cdc/45183.

62. Heiman KE, Karlsson M, Grass J, et al. Notes from the field: Shigella with decreased susceptibility to azithromycin among men who have sex with men - United States, 2002–-2013. *MMWR Morb Mortal Wkly Rep*. 2014;63(6):132-133. Available at: http://www.ncbi.nlm.nih.gov/pubmed/24522098.

63. Hassing RJ, Melles DC, Goessens WH, Rijnders BJ. Case of Shigella flexneri infection with treatment failure due to azithromycin resistance in an HIV-positive patient. *Infection*. 2014. Available at: http://www.ncbi.nlm.nih.gov/pubmed/24488332.

64. Baker KS, Dallman TJ, Ashton PM, et al. Intercontinental dissemination of azithromycin-resistant shigellosis through sexual transmission: a cross-sectional study. *Lancet Infect Dis*. 2015;15(8):913-921. Available at: http://www.ncbi.nlm.nih.gov/pubmed/25936611.

65. Centers for Disease Control and Prevention. Increase in extensively drug-resistant shigellosis in the United States. 2023. Available at: https://emergency.cdc.gov/han/2023/han00486.asp

66. Gaudreau C, Rodrigues-Coutlee S, Pilon PA, Coutlee F, Bekal S. Long-lasting outbreak of erythromycin- and ciprofloxacin-resistant campylobacter jejuni subspecies jejuni from 2003 to 2013 in men who have sex with men, Quebec, Canada. *Clin Infect Dis*. 2015;61(10):1549-1552. Available at: https://www.ncbi.nlm.nih.gov/pubmed/26187024.

67. Greninger AL, Addetia A, Starr K, et al. International spread of multidrug-resistant Campylobacter coli in men who have sex with men in Washington state and Quebec, 2015-2018. *Clin Infect Dis*. 2020;71(8):1896-1904. Available at: https://www.ncbi.nlm.nih.gov/pubmed/31665255.

68. Fernandez-Cruz A, Munoz P, Mohedano R, et al. Campylobacter bacteremia: clinical characteristics, incidence, and outcome over 23 years. *Medicine (Baltimore)*. 2010;89(5):319-330. Available at: https://www.ncbi.nlm.nih.gov/pubmed/20827109.

69. Larsen IK, Gradel KO, Helms M, et al. Non-typhoidal Salmonella and Campylobacter infections among HIV-positive patients in Denmark. *Scand J Infect Dis*. 2011;43(1):3-7. Available at: https://www.ncbi.nlm.nih.gov/pubmed/20849366.

70. Johnson S, Lavergne V, Skinner AM, et al. Clinical practice guideline by the Infectious Diseases Society of America (IDSA) and Society for Healthcare Epidemiology of America (SHEA): 2021 focused update guidelines on management of Clostridioides difficile infection in adults. *Clin Infect Dis*. 2021;73(5):e1029-e1044. Available at: https://www.ncbi.nlm.nih.gov/pubmed/34164674.

71. Louie TJ, Miller MA, Mullane KM, et al. Fidaxomicin versus vancomycin for Clostridium difficile infection. *N Engl J Med*. 2011;364(5):422-431. Available at: https://www.ncbi.nlm.nih.gov/pubmed/21288078.

72. Cornely OA, Crook DW, Esposito R, et al. Fidaxomicin versus vancomycin for infection with Clostridium difficile in Europe, Canada, and the USA: a double-blind, non-inferiority, randomised controlled trial. *Lancet Infect Dis*. 2012;12(4):281-289. Available at: https://www.ncbi.nlm.nih.gov/pubmed/22321770.

73. Guery B, Menichetti F, Anttila VJ, et al. Extended-pulsed fidaxomicin versus vancomycin for Clostridium difficile infection in patients 60 years and older (EXTEND): a randomised, controlled, open-label, Phase 3b/4 trial. *Lancet Infect Dis*. 2018;18(3):296-307. Available at: https://www.ncbi.nlm.nih.gov/pubmed/29273269.

74. Mikamo H, Tateda K, Yanagihara K, et al. Efficacy and safety of fidaxomicin for the treatment of Clostridioides (Clostridium) difficile infection in a randomized, double-blind, comparative Phase III study in Japan. *J Infect Chemother*. 2018;24(9):744-752. Available at: https://www.ncbi.nlm.nih.gov/pubmed/29934056.

75. Johnson S, Louie TJ, Gerding DN, et al. Vancomycin, metronidazole, or tolevamer for Clostridium difficile infection: results from two multinational, randomized, controlled trials. *Clin Infect Dis*. 2014;59(3):345-354. Available at: http://www.ncbi.nlm.nih.gov/pubmed/24799326.

76. Zar FA, Bakkanagari SR, Moorthi KM, Davis MB. A comparison of vancomycin and metronidazole for the treatment of Clostridium difficile-associated diarrhea, stratified by disease severity. *Clin Infect Dis*. 2007;45(3):302-307. Available at: http://www.ncbi.nlm.nih.gov/pubmed/17599306.

77. Di Bella S, Gouliouris T, Petrosillo N. Fecal microbiota transplantation (FMT) for Clostridium difficile infection: focus on immunocompromised patients. *J Infect Chemother*. 2015;21(4):230-237. Available at: http://www.ncbi.nlm.nih.gov/pubmed/25703532.

78. Serrano-Villar S, Talavera-Rodriguez A, Gosalbes MJ, et al. Fecal microbiota transplantation in HIV: a pilot placebo-controlled study. *Nat Commun*. 2021;12(1):1139. Available at: https://www.ncbi.nlm.nih.gov/pubmed/33602945.

79. Kelly CR, Ihunnah C, Fischer M, et al. Fecal microbiota transplant for treatment of Clostridium difficile infection in immunocompromised patients. *Am J Gastroenterol*. 2014;109(7):1065-1071. Available at: https://www.ncbi.nlm.nih.gov/pubmed/24890442.

80. Food and Drug Administration. Fecal microbiota for transplantation: safety alert - risk of serious adverse events likely due to transmission of pathogenic organisms. 2020. Available at: https://www.fda.gov/safety/medical-product-safety-information/fecal-microbiota-transplantation-safety-alert-risk-serious-adverse-events-likely-due-transmission.

81. DeFilipp Z, Bloom PP, Torres Soto M, et al. Drug-resistant *E. coli* bacteremia transmitted by fecal microbiota transplant. *N Engl J Med*. 2019;381(21):2043-2050. Available at: https://www.ncbi.nlm.nih.gov/pubmed/31665575.

82. Gurumurthy P, Ramachandran G, Hemanth Kumar AK, et al. Malabsorption of rifampin and isoniazid in HIV-infected patients with and without tuberculosis. *Clin Infect Dis*. 2004;38(2):280-283. Available at: https://www.ncbi.nlm.nih.gov/pubmed/14699462.

83. Sahai J, Gallicano K, Swick L, et al. Reduced plasma concentrations of antituberculosis drugs in patients with HIV infection. *Ann Intern Med*. 1997;127(4):289-293. Available at: https://www.ncbi.nlm.nih.gov/pubmed/9265429.

84. U.S. Food and Drug Administration. Highlights of prescribing information. CIPRO® (ciprofloxacin hydrochloride) tablet, for oral use CIPRO® (ciprofloxacin), for oral suspension. 2021. Available at: https://www.accessdata.fda.gov/drugsatfda_docs/label/2021/019537s092lbl.pdf.

85. Bérard A, Sheehy O, Zhao J, Nordeng H. Use of macrolides during pregnancy and the risk of birth defects: a population-based study. *Pharmacoepidemiology and Drug Safety*. 2015;24(12):1241-1248. Available at: http://www.ncbi.nlm.nih.gov/pubmed/26513406.

86. Padberg S, Wacker E, Meister R, et al. Observational cohort study of pregnancy outcome after first-trimester exposure to fluoroquinolones. *Antimicrob Agents Chemother*. 2014;58(8):4392-4398. Available at: http://www.ncbi.nlm.nih.gov/pubmed/24841264.

87. Schaefer C, Amoura-Elefant E, Vial T, et al. Pregnancy outcome after prenatal quinolone exposure. Evaluation of a case registry of the European Network of Teratology Information Services

(ENTIS). *Eur J Obstet Gynecol Reprod Biol*. 1996;69(2):83-89. Available at: http://www.ncbi.nlm.nih.gov/pubmed/8902438.

88. Loebstein R, Addis A, Ho E, et al. Pregnancy outcome following gestational exposure to fluoroquinolones: a multicenter prospective controlled study. *Antimicrob Agents Chemother*. 1998;42(6):1336-1339. Available at: http://www.ncbi.nlm.nih.gov/pubmed/9624471.

89. Czeizel AE, Rockenbauer M, Sorensen HT, Olsen J. The teratogenic risk of trimethoprim-sulfonamides: a population based case-control study. *Reprod Toxicol*. 2001;15(6):637-646. Available at: http://www.ncbi.nlm.nih.gov/pubmed/11738517.

90. Hernandez-Diaz S, Werler MM, Walker AM, Mitchell AA. Folic acid antagonists during pregnancy and the risk of birth defects. *N Engl J Med*. 2000;343(22):1608-1614. Available at: http://www.ncbi.nlm.nih.gov/pubmed/11096168.

91. Hernandez-Diaz S, Werler MM, Walker AM, Mitchell AA. Neural tube defects in relation to use of folic acid antagonists during pregnancy. *Am J Epidemiol*. 2001;153(10):961-968. Available at: http://www.ncbi.nlm.nih.gov/pubmed/11384952.

92. Ford N, Shubber Z, Jao J, Abrams EJ, Frigati L, Mofenson L. Safety of cotrimoxazole in pregnancy: a systematic review and meta-analysis. *J Acquir Immune Defic Syndr*. 2014;66(5):512-521. Available at: http://www.ncbi.nlm.nih.gov/pubmed/24853309.

93. Bortolus R, Filippini F, Cipriani S, et al. Efficacy of 4.0 mg versus 0.4 mg folic acid supplementation on the reproductive outcomes: a randomized controlled trial. *Nutrients*. 2021;13(12). Available at: https://www.ncbi.nlm.nih.gov/pubmed/34959975.

94. Puopolo KM, Lynfield R, Cummings JJ, et al. Management of infants at risk for group B streptococcal disease. *Pediatrics*. 2019;144(2). Available at: https://publications.aap.org/pediatrics/article/144/2/e20191881/38546/Management-of-Infants-at-Risk-for-Group-B?autologincheck=redirected.

95. Rao S, Kupfer Y, Pagala M, Chapnick E, Tessler S. Systemic absorption of oral vancomycin in patients with Clostridium difficile infection. *Scand J Infect Dis*. 2011;43(5):386-388. Available at: https://www.ncbi.nlm.nih.gov/pubmed/21198337.

96. Bourget P, Fernandez H, Delouis C, Ribou F. Transplacental passage of vancomycin during the second trimester of pregnancy. *Obstet Gynecol*. 1991;78(5 Pt 2):908-911. Available at: http://www.ncbi.nlm.nih.gov/pubmed/1923224.

97. Sheehy O, Santos F, Ferreira E, Berard A. The use of metronidazole during pregnancy: a review of evidence. *Curr Drug Saf*. 2015;10(2):170-179. Available at: http://www.ncbi.nlm.nih.gov/pubmed/25986038.