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Sexually transmitted shigellosis

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Abstract

Shigellae can be transmitted through sexual contact, especially among gay, bisexual, and other men who have sex with men (gbMSM). The dynamics and factors contributing to sexual transmission of shigellosis are not yet fully understood. *Shigella* spp. are intestinal pathogens with a low infectious dose, making them more likely to spread through sexual contact. Asymptomatic carriage may also contribute to its transmission through sexual activity. Recommendations for prevention of sexual transmission of shigellosis include less risky sexual behavior, the use of protective measures, thorough cleaning of sex toys, and good personal hygiene. If a partner has diarrhea, it is recommended to avoid sexual contact during and for at least 1 to 2 weeks after the symptoms have resolved, and to refrain from oral–anal contact for 4 to 6 weeks. Globally, the burden of shigellosis is highest in low- and middle-income countries, particularly among young children. In high-income countries, international travelers and gbMSM are considered the main risk groups for shigellosis. Raising awareness about the possible sexual transmission of shigellosis among at-risk groups is necessary. Increasing awareness among clinicians about the potential for sexual transmission of shigellosis is vital to ensure appropriate counseling and patient management.

Keywords: antibiotic resistance, genotyping, prevention, public health control strategies, sexually transmitted shigellosis, shigellosis

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Introduction

Sexually transmitted infections (STIs) are infections that spread primarily through sexual contact, including vaginal, anal, and oral sexual activities as well as the use of sex toys. Some STIs can also be transmitted from mother to child during pregnancy, childbirth, and breastfeeding. More than 30 different bacteria, viruses, and parasites can be transmitted through sexual contact, the most common being syphilis, gonorrhea, chlamydia, trichomoniasis, and four viral infections: human papillomaviruses, herpes simplex virus, hepatitis B virus, and HIV. In addition to the "classic" STIs, other pathogens such as *Shigella* spp., *Neisseria meningitidis*, mpox, hepatitis A, and the Ebola virus can be transmitted via sexual contact, although this is not their primary mode of transmission. These infections associated with sexual activity pose additional challenges for STI prevention and control (1).

Pathogen

Shigellae are invasive bacteria that are genetically very similar to *Escherichia coli*. Modern phylogenetic approaches have shown that they are essentially specific pathovars of *E. coli* with distinctive metabolic and antigenic properties. Shigellae are polyphyletic; members of several different *E. coli* clones probably acquired the ancestor of the present-day invasion plasmid, followed by adaptation to the plasmid, convergent evolution, acquisition of other mobile genetic elements, deletions, or mutations affecting gene function (such as loss of motility), resulting in invasive bacteria with the ability to spread from cell to cell. This process appears to have occurred independently several times with different *Shigella* spp. founders, resulting in the different *Shigella* lineages. Despite these phylogenetic findings, they are traditionally classified in a separate genus, *Shigella*, which is divided into four species based on their metabolic and antigenic characteristics: *Shigella dysenteriae*, *Shigella flexneri*, *Shigella boydii*, and *Shigella sonnei* (2, 3).

Pathogenesis

Shigellae are invasive bacteria that invade the epithelial cells of the mucosa in the distal ileum and colon, where they cause acute inflammation with microabscesses and subsequent ulceration of the mucosa. Infection with shigellae usually remains limited to the mucosa; positive blood cultures in patients with shigellosis are rare. Virulence factors enable the bacteria to attach to the mucosa, invade and multiply within cells, and spread laterally to neighboring cells. This invasiveness is due to a large virulent plasmid (pINV) that encodes several virulence factors, such as proteins known as invasion plasmid antigens (Ipa), which facilitate cell entry, along with a type III secretion system. Shigellae also produce exotoxins with enterotoxic activity. Although invasiveness is the primary pathogenic mechanism, the production of enterotoxins likely contributes to the development of watery diarrhea in the early stages of infection. *S. dysenteriae* serotype 1 also produces a protein cytotoxin called Shiga toxin, which has cytotoxic and enterotoxic effects, in addition to other biological effects (4).

Clinical presentation

The incubation period typically ranges from 1 to 4 days but may extend to as long as 8 days in some cases. Clinically, shigellosis can present with a range of symptoms. The first signs are usually fever, malaise, loss of appetite, and sometimes vomiting, followed by acute watery diarrhea within a few hours; in otherwise healthy individuals, these symptoms generally resolve within a few days. In severe cases, symptoms worsen and lead to bacterial dysentery

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or dysenteric diarrhea, with visible blood and mucus in the stool due to bacterial invasion of the mucosa of the distal ileum, colon, and rectum, often accompanied by abdominal pain and intestinal cramps. Rarely, extraintestinal complications may also occur, including febrile seizures, septicemia, keratoconjunctivitis, acute immune complex glomerulonephritis, post-infectious irritable bowel syndrome and reactive arthritis, and hemolytic uremic syndrome.

During the recovery phase of shigellosis, shigellae may continue to be shed in the stool for several weeks (usually 1 to 4), although long-term carriage is rare (4, 5). Asymptomatic infections are possible, especially in individuals that have previously had shigellosis (5).

The severity of the symptoms varies depending on the specific *Shigella* species causing the infection. *S. dysenteriae* characteristically produces bacterial dysentery. Infections with *S. boydii* and *S. flexneri* are also associated with more severe clinical presentation, and *S. sonnei* typically causes watery diarrhea (4, 5).

Diagnostics

During acute diarrhea, shigellae are typically present in high enough quantities to yield positive stool cultures. To maximize the likelihood of successful cultivation, it is crucial to collect stool samples as soon as possible after symptom onset. Whenever possible, transport media for stool samples (e.g., Cary–Blair) should be used to preserve shigellae viability because the bacteria degrade rapidly under unfavorable conditions. Stool samples should be delivered to the microbiology laboratory within 2 hours and plated immediately; if this is not possible, the stool sample should be refrigerated to improve the survival of shigellae. The stool sample is cultured on differential and selective media for *Salmonella* and *Shigella* (e.g., xylose lysine desoxycholate agar, XLD) (6, 7).

Molecular syndromic diagnostics for bacterial diarrhea include detection of specific virulence genes (typically *ipa*H) common to both *Shigella* spp. and enteroinvasive *E. coli* (EIEC). Stool samples are preferred for molecular diagnostics. Flocked rectal swab in transport medium can also be used in the diagnostics of diarrhea when stool specimens are not available; however, rectal swabs should be analyzed using molecular diagnostic tests because the sensitivity for this approach is lower (8). If a molecular test for *Shigella*/EIEC yields a positive result, stool samples should be cultured to obtain the shigellae because molecular testing does not provide information on the specific species or its antimicrobial susceptibility.

Presumptive shigellae colonies on plates are identified based on their metabolic characteristics using biochemical tests. Identification via mass spectrometry using the matrix-assisted laser desorption/ionization time-of-flight (MALDI-TOF) technique is still not feasible because it cannot reliably distinguish between the mass spectra of *Shigella* spp. and *E. coli*. Definitive species and serotype determination requires serotyping, identifying O antigens with specific antisera using slide agglutination (6).

Antibiotic susceptibility testing is crucial because resistance to multiple antibiotics is increasingly common. This resistance is especially common in countries where shigellosis is endemic (notably in developing regions) and in cases of sexually transmitted shigellosis (4, 5).

Treatment and prevention

In most patients, shigellosis is mild, but more severe cases require oral or intravenous fluid and electrolyte replacement. Antibiotic treatment is used in shigella dysentery; it reduces the duration of fever and diarrhea by 1 to 2 days and also shortens the duration of fecal shedding, thus reducing the risk of person-to-person transmission (4, 5). Fluoroquinolones or azithromycin are the first-line antibiotics for empirical treatment; if the infection occurred in Asia or other regions with a known high frequency of antibiotic resistance, ceftriaxone may also be considered. Specific therapy is based on in vitro antibiotic susceptibility results and includes fluoroquinolones, ceftriaxone, trimethoprim/sulfamethoxazole, or azithromycin (4, 9).

In the past, shigellae strains were highly susceptible to antibiotics such as ampicillin, chloramphenicol, trimethoprim/sulfamethoxazole, and nalidixic acid. However, resistant strains—particularly to fluoroquinolones, azithromycin, and third-generation cephalosporins—are becoming increasingly common, including multidrug-resistant strains (10). In 2024, the World Health Organization included fluoroquinolone-resistant shigellae as a high-priority pathogen on its list of bacterial pathogens of public health importance to guide research, development, and strategies to combat antimicrobial resistance (11).

Because shigellosis is highly contagious, strict hygiene measures are crucial to prevent its spread. For hospitalized patients, isolation is recommended (e.g., standard and in some cases—such as extensively drug-resistant shigellae—contact precautions). Preventive strategies for secondary transmission can vary significantly but often include advising patients—and sometimes their contacts—to avoid attending daycare, school, or work until a negative microbiological test result is obtained. In addition, individuals are discouraged from visiting public swimming pools or spas and from handling or preparing food for others until fully recovered (4, 5, 12).

Epidemiology

All *Shigella* species are pathogenic to humans and other primates, with humans being the only natural reservoir (5). The prevalence of specific *Shigella* species varies by region. Shigellae are endemic in countries with temperate and tropical climates. In higher-income countries, *S. sonnei* is the predominant species, whereas *S. flexneri* is more common in low- and middle-income countries (LMICs). Infections with *S. boydii* and *S. dysenteriae* are less common; *S. boydii* is mainly found in the Indian subcontinent, and *S. dysenteriae* is endemic in sub-Saharan Africa and South Asia (13).

The primary source of infection is symptomatic patients, although asymptomatic carriers can also spread the disease. The infectious dose is very low; studies in healthy volunteers have shown that as few as 10 *S. dysenteriae* serotype 1 bacteria or 180 *S. flexneri* or *S. sonnei* bacteria are sufficient for symptomatic infection, making shigellosis one of the most contagious forms of bacterial diarrhea. The reasons for such a low inoculum are not entirely clear; one factor could be their ability to survive low gastric pH because shigellae are more resistant to the effects of low gastric pH than *Salmonella* (14).

Transmission primarily occurs via the fecal–oral route, often via contaminated hands. After the symptoms have subsided, patients may continue to shed shigellae for several weeks. Secondary transmission to contacts is common, depending on the age of those involved, and it is more frequent when younger children are infected. In environments with poor hygiene, such as closed communities and institutions, the disease can spread rapidly (4, 5). A recent study has noted a rise in shigellosis cases among people that experience homelessness (15). Epidemics are frequently linked to contaminated food and water, with foodborne spread potentially related to inadequate hygiene among food handlers and the presence of flies (5). Shigellae are responsible for approximately 5% to 18% cases of traveler's diarrhea (13). Shigellae can also be transmitted through sexual contact (16).

Shigellosis occurs worldwide and in all seasons, affecting all age groups, with the highest burden of disease among children 1 to 4 years old in LMICs (5, 17). In Europe, according to the 2022 annual epidemiological report on shigellosis from the European Centre for Disease Prevention and Control (ECDC), 30 European Union/European Economic Area (EU/EAA) countries reported 4,149 confirmed cases, which corresponds to an overall notification rate of 1.5 cases per 100,000 population. This rate is comparable to pre-pandemic levels because the incidence of shigellosis significantly decreased during the COVID-19 pandemic. The highest notification rate in 2022 was observed in children under age 5, followed by the 25- to 44-year-old age group, which showed a pronounced gender imbalance. Among men 25 to 44 years old, the notification rate was 2.7 cases per 100,000 population, with a male-to-female ratio of 1.7:1. The gender disparity is believed to be linked to sexual transmission of shigellosis among gay, bisexual, and other men who have sex with men (gbMSM) (18). Species-level identification was available for 82.8% of the reported cases. The majority of shigellosis cases were caused by *S. sonnei* (62.3%) and *S. flexneri* (33.5%). Data on antimicrobial susceptibility were reported by only six countries (*S. sonnei* 671 isolates, *S. flexneri* 136 isolates), limiting the ability to draw conclusions regarding the prevalence of antimicrobial resistance in shigellae across the EU/EEA. Resistance to ampicillin was 80.6% in *S. sonnei* and 91.2% in *S. flexneri*. Resistance to cefotaxime was 49.5% in *S. sonnei* and 6.7% in *S. flexneri*, whereas resistance to ceftazidime was 2.7% in *S. sonnei* and 5.2% in *S. flexneri*. Resistance to ciprofloxacin was 31.9% in *S. sonnei* and 40.2% in *S. flexneri*. According to the ECDC shigellosis report, this resistance pattern in *S. sonnei* was connected with a travel-related outbreak of extended-spectrum beta-lactamase (ESBL)-producing *S. sonnei* ST152 infections.

Sexual transmission of shigellosis

The first descriptions of sexually transmitted shigellosis were published in the 1970s (19). A basic PubMed search for the term "sexually transmitted shigellosis" or "sexually transmitted *Shigella*," all article types (Fig. 1), shows few publications per year in the 1980s, a handful of publications in the 1990s, and a steady increase from 2000 onward, especially in the last decade.

A number of reports were published in the mid-2010s noting a growing gap between adult male and female case rates, strongly suggesting male-to-male sexual transmission as well as an increase in sexually transmitted shigellosis among gbMSM caused by *S. flexneri* 3a, *S. flexneri* 2a, or *S. sonnei* in a number of countries (20–24). One study also highlighted the intercontinental spread of macrolide-resistant *S. flexneri* serotype 3a through sexual transmission (25). Sexual transmission of variants of the intercontinentally transmitted ciprofloxacin-resistant *S. sonnei* has also been reported (26).

In January 2022, the United Kingdom Health and Security Agency (UKHSA) reported an increase in infections with extensively drug-resistant *S. sonnei*, followed by the first ECDC rapid alert on rising cases of sexually transmitted shigellosis among gbMSM in Europe. Cases occurred both sporadically and in outbreaks, and they were usually caused by *S. flexneri* and *S. sonnei* (27). Shigella spp. strains transmitted among gbMSM often show resistance to multiple antibiotics, with clusters of multidrug-resistant *Shigella* spp. being increasingly reported (28–30). Resistance to oral antibiotics such as ciprofloxacin and azithromycin was common, whereas resistance to third-generation cephalosporins was less frequent (31, 32). In July 2023, the ECDC issued a new epidemiological update on the spread of extensively drug-resistant *S. sonnei* in Europe. Genotyping has confirmed both national and international clusters, especially among gbMSM. These strains were resistant to first- and second-line treatments: fluoroquinolones, trimethoprim-sulfamethoxazole, and third-generation cephalosporins, as well as azithromycin, significantly limiting treatment options (33).

The dynamics and factors contributing to the sexual transmission of shigellosis remain unclear. Intestinal pathogens with low infectious doses are likely to be more easily transmitted through sexual contact, especially when there is an increased risk of fecal contamination, such as in oral–anal contact, fisting, and the use of sex toys (16). Asymptomatic transmission is also thought to contribute to sexual transmission of shigellosis (34, 35). Data on the prevalence of bacterial pathogens in the general asymptomat-

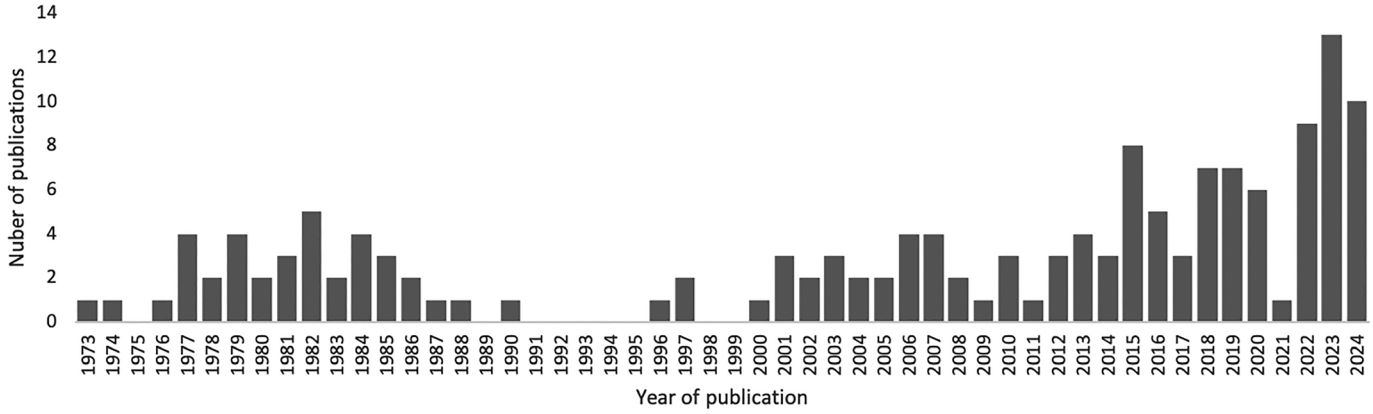

Figure 1 | Number of publications per year according to a basic search in Pubmed for the term "sexually transmitted shigellosis" or "sexually transmitted *Shigella*," all article types.

ic adult population, using sensitive molecular methods, are limited. In a Dutch study using molecular methods, no cases of *Shigella* spp. or EIEC were found in a control (asymptomatic) group of 1,195 individuals (36). In a smaller Slovenian study on the usefulness of molecular methods for the diagnosis of infectious diarrhea in adults, *Shigella* spp. / EIEC and *Giardia* spp. were detected in one individual among 154 asymptomatic adults (prevalence of 0.6%); the cultivation of *Shigella* spp. in this case was not successful (37). A 2024 meta-analysis of six studies from Australia, the Netherlands, and the United Kingdom on the prevalence of bacterial enteric pathogens in asymptomatic gbMSM using molecular methods found that 1.1% of 3,766 gbMSM had *Shigella* spp. in their stool (95% confidence interval [CI]: 0.7%–1.7%) (38). Bacterial enteric pathogens were detected more frequently in asymptomatic gbMSM on HIV pre-exposure prophylaxis (PrEP), those living with HIV, gbMSM reporting more than five new sexual partners in the past 3 months, and those engaging in insertive oral–anal contact and group sexual encounters, compared to gbMSM that tested negative for bacterial enteric pathogens (39, 40).

Risk factors for shigellosis in gbMSM include urban residence, HIV patients with low CD4+ counts, high viral loads (> 100,000 copies per mL), untreated HIV, HIV PrEP use, use of dating apps, recreational drug use and chemsex, multiple casual sex partners, oral–anal sexual practices, and concurrent STIs (16).

Sexually transmitted shigellosis is more commonly caused by strains resistant to first-line oral antibiotics such as macrolides and fluoroquinolones as well as third-generation cephalosporins, which can lead to failure of empiric treatment. A recent review found that resistance to antibiotics is generally higher in sexually transmitted shigellosis than in travel-associated cases (41). In 2015, the intercontinental spread of macrolide-resistant *S. flexneri* serotype 3a was described (25). In recent years, reports from various countries have been published describing clusters of patients with *Shigella* spp. resistant to third-generation cephalosporins and fluoroquinolones; of particular concern is the spread of extensively drug-resistant *Shigella* spp. with ESBL encoded by $bla_{\text{CTX-M-27}}$ (28, 42, 43). Infections with extensively drug-resistant *Shigella* spp. may lead to hospitalization because only parenteral antibiotics such as carbapenems remain as a treatment option for more severe infections caused by *Shigella* spp. that are resistant to fluoroquinolones, azithromycin, and third-generation cephalosporins (30).

Prevention of sexual transmission of shigellosis

Since the first report on sexually transmitted shigellosis, the importance of prevention through multimedia awareness campaigns targeting the at-risk gbMSM subpopulation has been emphasized (19). To prevent sexual transmission of shigellosis, safe sexual practices, use of protective measures such as condoms and gloves, thorough cleaning of sex toys and implements, and good personal hygiene before and after sexual contact are recommended in addition to standard measures to prevent secondary spread. If a partner has diarrhea, it is recommended to avoid sexual contact during and for at least 1 to 2 weeks after the symptoms have subsided, and to refrain from oral–anal contact for 4 to 6 weeks (33, 44).

Raising awareness of the possibility of sexually transmitted shigellosis among at-risk populations—especially gbMSM—and appropriate preventive measures is essential. Patients diagnosed with shigellosis should discuss the risk of sexual transmission with their healthcare provider; patient management should follow the same guidelines as for newly diagnosed STIs in these cases (33, 44).

Surveillance of sexually transmitted shigellosis and clinician awareness

The ECDC highlights the importance of raising clinician awareness about the rising incidence of sexually transmitted shigellosis, which can occur both domestically and during travel. Particular attention should be given to the possibility of sexual transmission in young men that have no history of travel or have not recently traveled to regions where shigellosis is common. For these patients, it is important to obtain a sexual history, perform testing for other STIs, and consider referring the patient to a sexual health clinic for further evaluation and care (27). In some countries, the epidemiological assessment of shigellosis patients includes a question regarding potential sexual transmission (for adult patients only) as part of the patient questionnaire, which aids surveillance efforts (45, 46).

Cases of shigellosis must be reported to the public health authorities. Microbiology laboratories are advised to monitor the antibiotic resistance of *Shigella* spp. and send resistant isolates to national reference centers for genotyping (e.g., whole genome sequencing). This allows the comparison of *Shigella* spp. sequences with international databases, facilitating the tracking of multidrug-resistant clones and reporting to the ECDC to strengthen public health control strategies (27).

Conclusions

Shigellosis, a bacterial infection caused by *Shigella* spp., can be transmitted through sexual contact, particularly among gbMSM. Although the dynamics and contributing factors for sexual transmission of shigellosis are not yet fully understood, its incidence has been steadily rising, especially over the past decade. Of particular concern is the international spread of multidrug-resistant shigellae, posing challenges to treatment and containment efforts. In high-income countries, international travelers and gbMSM are considered the main risk groups for shigellosis. Raising awareness among at-risk populations about the potential for sexual transmission is essential for limiting its spread. Equally important is increasing awareness among clinicians to ensure timely diagnosis and effective management of sexually transmitted shigellosis, as well as comprehensive counseling of patients regarding STIs. Microbiology laboratories are advised to refer resistant *Shigella* spp. isolates to national reference centers for genotyping. This allows the comparison of *Shigella* spp. sequences with international databases, providing better insights into ongoing transmission pat-

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