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# Prevalence and Antimicrobial Resistance Patterns of *Shigella* in Ethiopia: A Review

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## Abstract

*Shigella* is a non-motile, rod shaped, nonspore forming, and non-lactose fermenting facultative anaerobic Gram-negative bacterium that causes bacillary dysentery or also known as shigellosis. It is endemic throughout the world and it is among the most common causes of bacterial diarrheal diseases. Globally, it is estimated that shigellosis causes about 1,100,000 deaths per year, two-thirds of the patients being children under 5 years of age. The disease is transmitted faeco-orally, the commonest modes being person-to-person contact and contaminated food and water. Infected food handlers can spread the disease Flies can breed in infected faeces and contaminate food. It is a disease of overcrowding, insanitary conditions and poor personal hygiene, and affects mostly children of developing countries. The treatment of Shigellosis has currently become more challenging due to the emergence of drug resistant species and associated with a variety of biological, pharmacological and societal variables with the worst combinations in low and middle income countries. Multidrug-resistant *Shigella* significantly varies from area to area of the world in relation with the practice of widespread use of antimicrobial agents. There is an increasing burden of *Shigella* infection and *Shigella* is becoming resistance to the commonly prescribed antimicrobial drugs in Ethiopia like chloramphenicol, Amoxicillin and tetracycline. Therefore, initiating and scale up of performing drug susceptibility test for each shigellosis case, create awareness and educate the community.

## Article Info

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## Keywords

*Shigella*, Ethiopia, Antimicrobial resistance, community

## Introduction

*Shigella* is a non-motile, rod shaped, nonspore forming, and non-lactose fermenting facultative anaerobic Gram-negative bacterium that causes bacillary dysentery or also known as shigellosis (Al-Haddad, 2011). Shigellosis is an acute invasive enteric infection often characterized by abdominal pain, fever and bloody diarrhea (dysentery).

Shigellosis is caused by *Shigella* species. However, three predominant strains are responsible for majority of shigellosis cases, *S. sonnei*, *S. flexneri* 2a and *S. dysenteriae* type 1. Of these, *S. sonnei* is encountered mostly in industrialized countries, *S. flexneri* 2a in developing countries and *S. dysenteriae* type 1 is the only epidemic as well as pandemic strain. They are pathogenic primarily due to their ability to invade intestinal epithelial cells. Shigellosis is a global human health problem. It is the most important cause of bloody diarrhea worldwide, especially in developing countries with substandard hygiene and poor quality of water supplies (Niyogi, 2005). Shigellosis as a global human health problem is more severe than other forms of gastroenteritis. It is endemic throughout the world and it is among the most common causes of bacterial diarrheal

diseases. Globally, it is estimated that shigellosis causes about 1,100,000 deaths per year, two-thirds of the patients being children under 5 years of age (Moezardalan *et al*., 2003).

The disease is transmitted faeco-orally, the commonest modes being person-to-person contact and contaminated food and water. Infected food handlers can spread the disease Flies can breed in infected faeces and contaminate food. It is a disease of overcrowding, insanitary conditions and poor personal hygiene, and affects mostly children of developing countries (Sur *et al*., 2004). Shigellosis typically evolves through several phases and manifestations of *Shigella* infection vary with the infecting species, the age of the host, the presence of risk factors and the specific immune status of the host. The incubation period is 1 to 4 days, but may be as long as 8 days with *S. dysenteriae* (Niyogi, 2005).

The emerging of multi drug resistance is becoming a serious problem in the treatment of shigellosis. An increment of multidrug resistance to shigellosis is equivalent to a widespread uncontrolled use of antibiotics in developing countries. This emergency of drug resistance calls for the rational use of eff ective drugs and underscores the need for alternative drugs to treat infections caused by resistant strains (Bhattacharya *et al*., 2005). Studies have been carried out in different parts of Ethiopia at different times to document the epidemiology of and drug Susceptibility pattern of *Shigella* species. Even though there are researches, there is no summarized prevalence data of this bacterial infection and its drug Susceptibility pattern in Ethiopia and/or it is not enough. The objective of this paper is to review the prevalence and antimicrobial resistance patterns of *shigella* isolates conducted in Ethiopia.

## Evolution of shigellosis

*Shigella* was discovered in 1896 by a Japanese scientist, Dr Kiyoshi Shiga as bacteria causing dysentery in humans and primates (Niyogi, 2005; CDC, 2013). *Shigella flexneri* was described by Dr Simon Flexner in 1900. *Shigella boydii* was first isolated in India 1931 and was described by American bacteriologist and epidemiologist, Mark Frederick Boyd while *Shigella sonnei* was first isolated in 1904, but it was in 1915 that its pathogenicity was recognized by Dr Carl Olaf Sonnei(Washington *et al*., 2006; Todar, 2010) and it was in 1950 that the Congress of the International Association of Microbiologists *Shigella* Commission adopted as the generic name *Shigella* and that species subgroups be designated A (*Shigella dysenteriae*), B (*S. flexneri*), C (*S. boydii*) and D (*S. sonnei*). Based on 16S rRNA sequencing, *Shigella*is from gamma Proteobacteria in the family *Enterobacteriaceae* phylum (Washington *et al*., 2006).

## Shigellosis and pathogenesis

*Shigella* causes disease by invading and replicating in cells lining the colonic mucosa. Epithelial cells of the colonic mucosa are the primary targets used by these bacteria and a key step in the pathogenesis of *Shigella* infection. Ray *et al.,* (2009) elucidate the process of cell infection to be aided by the bacterial DNA encoding a number of plasmid and chromosomal proteins that assisted in adhesion of bacterial cells to epithelial cells with subsequent invasion through the M cells. M cells are specialized epithelial cells which continuously sample material from the gut lumen and deliver them to the underlying mucosal lymphoid tissue, where immune responses can be initiated. This in turn facilitates transportation of bacteria (Winkler *et al*., 2007).

*Shigella* infection is characterized by degeneration of the epithelium and inflammation of the lamina propria resulting in desquamation and ulceration of the mucosa with eventual leakage of blood and mucus into the lumen of the intestine. During infection, water absorption by the colon is negligible and this exacerbates diarrhoea. It is possible that prostaglandin interactions induced by the inflammatory response to bacterial invasion contribute to intestinal electrolytes and fluid movement resulting in colitis and diarrhoea (Todar, 2010; Romero *et al*., 2011). Central to its mechanism of virulence, *Shigella* expresses a type III secretion system (T3SS) that is responsible for the conveyance of a series of bacterial effectors into host cells, aimed at diverting host cellular processes that result in direct bacterial colonization and subsequent dissemination within the mucosal epithelium via subjugation of the host inflammatory response (Todar, 2010).

## Health impact of shigellosis

*Shigella* is highly adapted to human as the only known natural hosts and incidences of shigellosis have been reported worldwide. According to Ram *et al.,* (2008), the average world annual incidences are estimated to be 80-165 million cases with 99% occurring in developing countries. About 1.1 million people die from *Shigella* infection each year of which 60% occur in children below 5 years of age. In endemic areas of the developing

world, shigellosis is predominantly a pediatric disease (Mandomando *et al*., 2007). The urban impoverished communities globally are hardest hit due to overcrowding, substandard sanitation, hygiene and lack of clean water. Institutions such as day-care centers, prisoners, military recruits and travellers are especially at high risk.

In developed countries shigellosis occurs erratically as outbreaks, while in developing countries reported incidences are probably 20 times more than in developed countries, yet a significant number of cases go unreported (WHO, 2009). *S. flexneri*, the most frequently isolated species worldwide and accounts for 60% of cases in the developing countries, *S. sonnei* causes 77% of cases in the developed world as compared to 15% of cases in the developing countries while *S. dysenteriae* causes epidemics of dysentery particularly in confined populations like camps or schools (WHO, 2009). *Shigella* species is one of the eight dangerous drug resistance bacteria. Worldwide, there are 700,000 deaths as a result of antimicrobial resistance (AMR) every year according to 2016 WHO report. The experts suggest that this figure will rise to 4.2 million in Africa and 10 million globally by 2050, if nothing is done (Woolhouse *et al*., 2017).

## Antimicrobial resistance

## Global trends of antimicrobial resistance

Antibiotic resistance becomes a critical public health problem around the globe in recent years. Antibiotic resistance is a natural phenomenon that occurs whenever antibiotics are in use. However, there are human behaviors that contribute to the rapid development and spread of bacterial antibiotic resistance. According to UNICEF/WHO (2009) availability and use of broad spectrum antibiotic without prescriptions facilitate the development of resistance by *Shigella* species.

The treatment of Shigellosis has currently become more challenging due to the emergence of drug resistant species and associated with a variety of biological, pharmacological and societal variables with the worst combinations in low and middle income countries (Woolhouse *et al*., 2017). Multidrug-resistant *Shigella* significantly varies from area to area of the world in relation with the practice of widespread use of antimicrobial agents (Woolhouse *et al*., 2017).

In the late 1980s, fluoroquinolones (norfloxacin, ciprofloxacin and ofloxacin) were introduced and were found to be very effective in the treatment of shigellosis cases including those caused by multi-drug resistant *S. dysenteriae* type 1 strain (Sur *et al*., 2004). Recent outbreak investigations in India and Bangladesh showed high level of resistance even to norfloxacin, ciprofloxacin and ofloxacin (Sarkar *et al*., 2003). A case–control study to characterize the epidemiology of bloody diarrhea in rural western Kenya reported that 80% of the bacterial pathogens isolated were *Shigella* species of which approximately 49% was caused by *S. flexneri* (Brooks *et al*., 2006). Shigellosis is also an important cause of infectious diarrhea in Iran (Moezardalan *et al*., 2003), mostly community acquired, caused mainly by *S. flexneri* and *S. dysenteriae*.

## Antimicrobial resistance in Ethiopia

Antimicrobial resistance is a global problem in general, but it might be more severe in Ethiopia where there is lack of rigorous regulations, but there is easy access of antimicrobials for purchase of people without prescription and incomplete treatment courses as the result of patient non-compliance. There have been studies conducted in Ethiopia on shigellosis (Table 1) which suggest an increase in the antimicrobial resistance of *shigella* to commonly used antimicrobials (Moges, 2009; Atsebaha *et al*., 2015; Wondwossen *et al*., 2018; Addisu and Mengistu, 2019; Getachew *et al*., 2014; Getnet and Haimanot, 2014; Yeshiwodim *et al*., 2015; Kahsay *et al*., 2008; Gebremichael *et al*., 2018; Berhanu *et al*., 2006).

Abebe (2001) and Mengistu *et al*., (2014) reported that 77, 17 *Shigella* strains were isolated from 384, 382 stool samples. Among the isolates 40.3%, 29.5% were caused by *S. flexneri* respectively. High prevalence of *Shigella* species (16.9%) was also reported from South Ethiopia (Kahsay *et al*., 2008). Similarly, a study conducted in northwest Ethiopia (Gondar) from 2006 to 2008 showed that *Shigella* species was isolated from 7.5 % (90 isolates) of the total 1,200 stool specimens. The study also indicated that *S. flexneri* was the most frequently isolated species which constituted 72.2 % of *Shigella* isolates (Moges, 2009). Other studies in Gondar also showed that *Shigella* species were the most frequently identified etiological agents for diarrhea (Kahsay *et al*., 2008). In addition, a study conducted by Tesfaye *et al*., 2014, Michael *et al*., 2019 and Gebremichael *et al*., 2018 reported the majority of isolate is *S. flexneri*, *S. dysenteriae* and *S. sonnei* respectively (Table 2).

**Table.1** Antimicrobial resistance profiles of *Shigella* isolates in Ethiopia

| Year | Location | Specimen | No. of sample Tested | Prevalence No. (%) | MDR | Predominant serogroup Isolated | Common resistance pattern | Maximum drug resisted No. | References |
|---|---|---|---|---|---|---|---|---|---|
| 2000 | Jimma | Stool | 384 | 77(20.1) | 66 (85.7) | *Serogroup B(flexneri)* | TET, AMP, SXT, CHL, CF,CB | 10 | Abebe (2001) |
| 2001-2005 | Gondar | Stool | 2891 | 214(7.4) | 188 (87.8) | - | COT, AMP, TET,CHL | 6 | Gizachew *et al.*, (2006) |
| 2003/4 | Gondar | Stool | 391 | 29(7.42) | 28(96.6) | - | AMP,CHL,TET | 9 | Berhanu *et al.*, (2006) |
| 2005 | Gondar | Stool | 384 | 65(16.9) | 53(81.5) | - | AMP,TET,SXT,CHL | 6 | Kahasay *et al.*, (2008) |
| 2006/7 | Gondar | Stool | 384 | 60(15.6) | 48(80) | - | AMP,TET,SXT, CHL | 6 | Kahasay *et al.*, (2011) |
| 2007 | Harar | Stool | 244 | 17(6.7) | - | - | TET, AMP, AMX | 5 | Ayalu *et al.*, (2011) |
| 2006-2008 | Gondar | Stool | 1200 | 90(7.5) | 71(79) | *Serogroup A (S. dysenteriae)* | AMP,TET,COT,CHL | 7 | Moges (2009) |
| 2009 | Bahir Dar | Stool | 215 | 32(14.9) | 32(100) | - | S,AMP,TET,AMX,COT,CF ,CHL | 9 | Getachew *et al.*, (2011) |
| 2011 | Hawassa | Stool | 158 | 11(7) | 11(100) | *Serogroup B(flexneri)* | AMP,TET,ERY,CRO,AMX | 7 | Mulatu *et al.*, (2014) |
| 2011 | Harar | Stool | 384 | 56(14.6) | 46(82.1) | - | TET,AMP,COT,CHL | 5 | Habtamu *et al.*, (2014) |
| 2011/12 | Mekelle | Stool | 260 | 18(6.9) | 16(88.9) | *Serogroup D (S. sonnei)* | AMP,TET,CHL,COT | 6 | Gebremichael *et al.*, (2018) |
| 2011/12 | Butajira | Stool | 382 | 17(4.5) | 9(56.25) | *Serogroup D (S. sonnei)* | TET,AMP,SXT | 7 | Getachew *et al.*, (2014) |
| 2012 | Jimma | Stool | 260 | 6(2.3) | 6(100) | - | AMP,COT,AMX | 5 | Getnet and Haimanot (2014) |
| 2012 | Addis Ababa | Stool | 253 | 23(9.1) | 20 (87) | - | AMP,AUG,SXT | 8 | Yeshiwodim *et al.*, (2015) |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| 2014 | Gondar | Stool | 372 | 17(4.57) | 16(94.1) | *Serogroup B(S. flexneri)* | AMP,TET,AMX,SXT,CF,KAN,GEN | 9 | Tesfaye *et al.,* (2014) |
| 2014 | Mekelle | Stool | 216 | 15(6.9) | 12(80) | - | AMX,COT,CIP,NOR,GEN | 8 | Atsebaha *et al.,* (2015) |
| 2014 | Jimma | Stool | 176 | 2(1.1) | 2(100) | - | COT,NAL,AMP,TET | 4 | Tesfahun *et al.,* (2016) |
| 2015 | Arbaminch | Stool | 376 | 10(3) | - | - | CLR,AMX,AMC | 10 | Mohammedaman and Alemu (2016) |
| 2015/16 | Debre Markos | Stool | 220 | 5(2.3) | - | - | AMP,CHL,TET | 5 | Abeba *et al.,* (2018) |
| 2015/16 | Nekemte | Stool | 422 | 9(2.1) | 3(33.3) | - | AMX,GEN,CHL | 4 | Alemayehu and Mulissa (2018) |
| 2015/16 | Harar | Stool | 417 | 6(1.4) | 6(100) | - | CHL,COT,TET | 8 | Dadi *et al.,* (2018) |
| 2016 | Wegera | Stool | 225 | 5(2.2) | - | - | TET,AMP,AMX,GEN | 6 | Hailemariam *et al.,* (2018) |
| 2016 | Robe/Goba | Stool | 422 | 18(4.3) | 18(100) | - | CHL,TET,DOX,AMX | 4 | Addisu and Mengistu (2019) |
| 2016/17 | Wolkite | Stool | 170 | 4(2.4) | 3(75) | - | AMP,AMX | 2 | Temesgen *et al.,* (2019) |
| 2017 | Arbaminch | Stool | 167 | 8(4.8) | 5(62.5) | - | AMP,ERY,CHL | 7 | Gemechu *et al.,* (2018) |
| 2017 | Dire Dawa | Stool | 218 | 6(2.8) | 2(10.5) | - | AMP,AMX,CHL,TET | 5 | Gizaw *et al.,* (2019) |
| 2017 | SNNP | Stool | 204 | 17(8.3) | 15(88.2) | - | AMP,GEN,SXT,CHL | 6 | Wondwossen *et al.,* (2018) |
| 2017 | Adama | Stool | 232 | 22(9.5) | - | *Serogroup A (S. dysenteriae)* | AMP,TET,CIP | 7 | Bedada *et al.,* (2019) |
| 2018 | Gondar | Stool | 257 | 26(10.1) | 10(38.5) | *Serogroup A (S. dysenteriae)* | AMP,SXT,TET,AMX | 8 | Michael *et al.,* (2019) |
| 2018 | Gondar | Stool | 272 | 29(10.7) | 17(58.6) | - | AMX,CHL,TET,SXT | 7 | Amare *et al.,* (2019) |

AMP: Ampicillin; AMX-CAL: amoxicillin-clavulinic acid; CHL: chloramphinicol; CF: cephalothin; CIP: ciprofloxacin; GEN: gentamycin; CLR: clarithromycin; KAN: kanamycin; CEF: cefaclor; CRO: ceftriaxone; NOR: norfoxacillin; DOX: doxycycline; S: streptomycin; SXT (COT): trimethoprim-sulfamethoxazole; TET: tetracycline; CB: carbenicillin; AUG: augumentin; ERY: erythromycin; MDR: multiple drug resistance.

**Table.2** *Shigella* resistance patterns for each antibiotic tested in some selected reports in Ethiopia

| Authors | Antibiotics tested | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | AMP (%) | AMX (%) | CIP (%) | TET (%) | CHL (%) | CRO (%) | GEN (%) | SXT (%) |
| Abebe (2001) | 29.9 | - | - | 36.4 | 59.7 | - | 98.7 | 67.5 |
| Moges (2009) | 78.9 | - | 2.2 | 90 | 67.8 | 0 | 12.2 | 84.6 |
| Tesfaye *et al.,* (2018) | 94.1 | 88.2 | 0 | 88.2 | 17.6 | - | 41.2 | 58.8 |
| Atsebaha *et al.,* (2015) | 100 | 86.7 | 6.7 | - | 46.7 | - | 13.3 | 66.7 |
| Michael *et al.,* (2019) | 61.5 | 34.6 | 0 | 65.4 | 7.7 | 3.9 | 23.1 | 38.5 |
| Amare *et al.,* (2019) | - | 93.1 | - | 89.7 | 44.8 | - | - | 41.4 |
| Gizaw *et al.,* (2019) | 83.3 | 100 | - | 50 | 66.7 | 0 | 0 | 16.7 |
| Wondwossen *et al.,* (2018 | 82.4 | - | 17.6 | - | 47.1 | 17.6 | 76.5 | 64.7 |
| Getnet and Haimanot (2014) | 100 | 100 | 0 | - | 16.7 | 0 | 0 | 100 |
| Getachew *et al.,* (2014) | 47.1 | - | 5.9 | 82.4 | 29.4 | 0 | 17.6 | 76.5 |
| Ayalu *et al.,* (2011) | 100 | 100 | - | 70.6 | 29.4 | - | 0 | - |
| Mulatu *et al.,* (2014) | 63.6 | 100 | 0 | 54.5 | 9.1 | 54.5 | 27.3 | 0 |
| Dadi *et al.,* (2018) | 33.3 | - | 0 | 83.3 | 50 | 16.7 | 33.3 | 66.7 |
| Abeba *et al.,* (2018) | 100 | 100 | 0 | 80 | 80 | 0 | 0 | 20 |
| Kahasay *et al.,* (2011) | 80 | - | 8.3 | 85 | 48.3 | - | 10 | 76.7 |
| Kahasay *et al.,* (2008) | 81.5 | - | 9.2 | 87.7 | 50.8 | - | 10.7 | 75.4 |
| Habtamu *et al.,* (2014) | 94.6 | - | 0 | 96.4 | 53.6 | - | 21.4 | 73.2 |
| Gizachew *et al.,* (2006) | 79.9 | - | 8.9 | 86 | 52.8 | - | 7.9 | 73.4 |
| Gebremichael *et al.,* (2018) | 88.9 | - | 0 | 77.8 | 55.6 | 0 | 27.8 | 55.6 |
| Hailemariam *et al.,* (2018) | 100 | 100 | - | 60 | 40 | 0 | 60 | 40 |
| Getachew *et al.,* (2011) | 93.8 | 75 | 0 | 93.8 | 53.1 | - | 18.8 | 62.5 |
| Yeshiwodim*et al.,* (2015) | 95.7 | 91.4 | 4.3 | - | 21.7 | 4.3 | 17.4 | 52.2 |

AMP: ampicillin; AMX: amoxicillin; CIP: ciprofloxacin; TET: tetracycline; CHL: chloramphenicol; CRO: ceftriaxone; GEN: gentamicin; SXT(COT): trimethoprim-sulfamethoxazole; - (not tested); 0(susceptible).

## Conclusion and recommendation

This review paper indicates there is an increasing burden of *Shigella* infection and *Shigella* is becoming resistance to the commonly prescribed antimicrobial drugs in Ethiopia like ampicillin, Amoxicillin, chloramphenicol and tetracycline. Therefore, initiating and scale up of performing drug susceptibility test for each shigellosis case, create awareness and educate the community about not to use drugs unless it is prescribed.

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