

Epidemic dysentery



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Epidemics of bloody diarrhoea are currently sweeping through Africa, resulting in the deaths of many adults and children. This *DD* supplement looks at what causes epidemic dysentery and provides guidelines on preventing and managing epidemics.



Marc Schlossman/Panos Pictures

People in refugee camps with limited water and sanitation facilities are particularly at risk of infection during a dysentery epidemic.

Dysentery – bloody diarrhoea – is one of the most dangerous types of diarrhoea. In general, it is more severe and more likely to result in death than other forms of acute diarrhoea.

Large scale outbreaks (epidemics) of dysentery are a particular threat to public health. The death rate can be as high as 15 per cent, and health care services are severely stretched during epidemics. Even when correctly treated, about 5 per cent of people with dysentery can die during an epidemic.

The bacterium responsible for epidemic dysentery is *Shigella dysenteriae* type 1 (Sd1)¹. *S. dysenteriae* is one of four species of *Shigella*. The others are *Shigella flexneri*, *Shigella sonnei* and *Shigella boydii*. These species are usually less dangerous than Sd1 and they do not cause large epidemics.

Disease caused by Sd1 tends to be more common in infants, and elderly and malnourished people. Mortality is also highest in these groups.

Since Sd1 was first identified late last century, extensive epidemics have been reported in Africa, Asia and Latin America.

An epidemic of Sd1 in Latin America between 1969 and 1973 was responsible for more than 500,000 cases of dysentery and 20,000 deaths.

Recently, a series of epidemics has been affecting countries in eastern, central and southern Africa including Rwanda, Burundi, Malawi, Zambia, Zimbabwe, Swaziland and Mozambique. There are no reliable data yet on the situation in Tanzania, Zaire and Angola, but it is likely that Sd1 epidemics are also present in those countries. Political upheaval in Burundi in October and November 1993 caused more than 650,000 people to flee to neighbouring Tanzania and Rwanda, taking dysentery with them. Sd1 is one of the main causes of death in refugee camps in countries who share a border with Burundi.

Clinical features

The main clinical sign of infection with Sd1 is bloody diarrhoea. Other symptoms can include abdominal cramps, fever, or severe pain during defecation. However, bloody diarrhoea during a dysentery epidemic is the only sign needed for diagnosing

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DEFINITIONS

- **dysentery** bloody diarrhoea
- **epidemic dysentery** large-scale outbreaks of bloody diarrhoea, almost always caused by *Shigella dysenteriae* type 1 (Sd1)
- **endemic dysentery** a normal incidence of bloody diarrhoea, caused by a range of organisms including *Shigella*.
- **Shigella** a genus of bacteria with four species – *Shigella dysenteriae*, *Shigella flexneri*, *Shigella boydii* and *Shigella sonnei*. *Shigella* causes the most serious episodes of bloody diarrhoea
- **shigellosis** infection caused by one of the *Shigella* species, often (but not always) associated with bloody diarrhoea

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infection with Sd1.

Most cases are uncomplicated and self-limiting, lasting up to seven days. But dysentery caused by Sd1 can also result in severe complications including persistent diarrhoea (diarrhoea lasting 14 days or more), septicaemia (blood poisoning), rectal prolapse, and haemolytic-uraemic syndrome (HUS). HUS is a serious condition affecting the kidneys and blood clotting system.

Transmission

Sd1 is extremely virulent – only a few bacteria need to be swallowed to cause illness. As few as 10–100 organisms can cause disease in adults. Because of this, Sd1 is thought to be transmitted often through direct person to person contact. For example, a person with faecally-contaminated hands can pass Sd1 bacteria to another person's hands. The second person may swallow the germs and become ill with dysentery. (This is unlike cholera which is mainly spread through contaminated food or water, and is rarely spread by direct person to person contact.) Sd1 can also be transmitted through contaminated food and water.

Epidemics tend to occur during hot,

humid and rainy seasons, although this is not always the case. In epidemic areas, up to one-third of the population can be infected, especially in crowded areas with inadequate sanitation, poor hygiene and limited supplies of safe water.

Other causes of dysentery

While large-scale outbreaks of dysentery are almost always caused by *Shigella dysenteriae* type 1, other types of bloody diarrhoea are often present (endemic) in communities without reaching epidemic proportions.

The most severe episodes of endemic dysentery are caused by other species of *Shigella* – *Shigella flexneri*, *Shigella boydii* and *Shigella sonnei*. Other pathogens causing endemic dysentery in children include: *Campylobacter jejuni*, invasive strains of *Escherichia coli*, non-typhoid *Salmonella* strains and *Entamoeba histolytica*.

This DD supplement concentrates on *Shigella dysenteriae* type 1 because it is the cause of epidemic dysentery.

Guidelines available soon

WHO has recently revised its guidelines for the control of epidemics due to *Shigella dysenteriae* type 1. WHO recognises that current knowledge regarding Sd1 is

incomplete and further research is needed. But much can be done to reduce the incidence and deaths caused by Sd1.

The guidelines describe the disease, its clinical features and epidemiology, and propose strategies for control and prevention. They provide the basis for the information in this supplement, especially pages 3–4.

The guidelines also explain steps health managers and workers faced with dysentery epidemics can take to organise and use their resources effectively.

The material for this supplement was written by Dr Ronald Waldman, CDR, WHO, CH-1211 Geneva 27, Switzerland with assistance from Dr Olivier Fontaine and Dr Leila Richards.

1. *The cause of epidemic dysentery during the last half of this century has always been Shigella dysenteriae type 1 (Sd1) with one possible exception – an outbreak of epidemic dysentery in Swaziland in 1992 where E. coli O157 was reported as the cause; however Sd1 and Vibrio cholerae were also present in the population.*

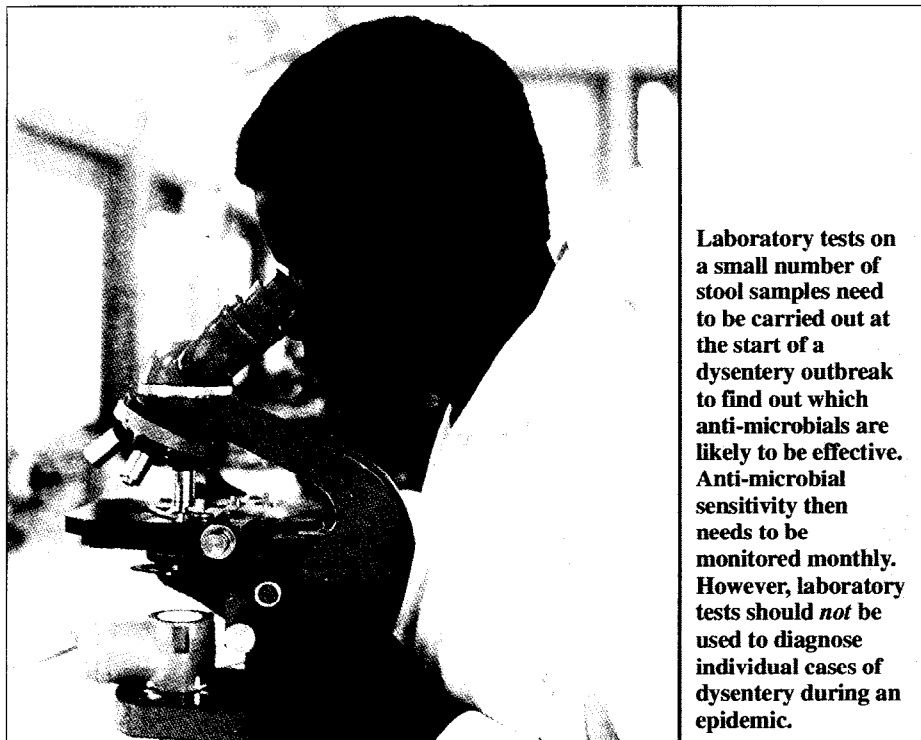
The new WHO guidelines will be available in mid 1994. Write to: CDR, WHO, CH-1211 Geneva 27, Switzerland.

For more information about treating endemic dysentery caused by Shigella, see the shigellosis supplement in DD44.

A comparison of epidemics of dysentery and cholera

	EPIDEMIC DYSENTERY	CHOLERA
Causative organism	<i>Shigella dysenteriae</i> type 1	<i>Vibrio cholerae</i> O1 or <i>Vibrio cholerae</i> O139
Related organisms	Other shigella species; <i>C. jejuni</i> , certain <i>E. coli</i> , and <i>E. histolytica</i> can cause bloody diarrhoea, but not epidemics	Other <i>V. cholerae</i> can cause profuse watery diarrhoea, but not epidemics
Infective dose (The number of organisms that need to be swallowed before a person becomes ill)	10–100 organisms	1,000–1,000,000 organisms
Clinical features	bloody diarrhoea, sometimes with fever abdominal cramps pain on defecation	profuse watery diarrhoea dehydration
Complications	persistent diarrhoea septicaemia rectal prolapse haemolytic-uraemic syndrome	severe dehydration leading to shock (circulatory collapse) electrolyte imbalance
Treatment	antibiotics save lives feeding is crucial rehydration may be necessary	rehydration saves lives antibiotics shorten illness in severely ill patients
Antibiotics of choice (according to sensitivity patterns)	nalidixic acid cotrimoxazole ampicillin	doxycycline tetracycline cotrimoxazole
Ages affected	all	all
Transmission	person to person food water	food water
Attack rates	up to 50 per 1,000 population	1–20 per 1,000 population
Case-fatality rate in untreated patients	10–20%	40%
Case-fatality rate in treated patients	about 5%	less than 1%

Detecting and managing an epidemic of Sd1



Chevalier/WHO

Laboratory tests on a small number of stool samples need to be carried out at the start of a dysentery outbreak to find out which anti-microbials are likely to be effective. Anti-microbial sensitivity then needs to be monitored monthly. However, laboratory tests should *not* be used to diagnose individual cases of dysentery during an epidemic.

■ SURVEILLANCE

Outbreaks of dysentery can only be detected early if a system for observing and reporting disease has been established. This is called disease surveillance.

A simple case record (showing the date, name, age and address of each patient; the clinical diagnosis; and the treatment provided) should be kept at every health facility, and information from case records should be reported regularly to the local health authorities. This would ensure that outbreaks of epidemics were detected early.

An epidemic should be suspected if there is a rapid increase in the daily or weekly number of cases of bloody diarrhoea, or if increased deaths from bloody diarrhoea are reported in a community.

When an epidemic is suspected, health workers should immediately notify their supervisors and request assistance from them.

■ LABORATORY ANALYSIS

When epidemic dysentery has been reported, all efforts should be made to confirm the cause (which, in almost all cases, will be *Shigella dysenteriae* type 1) by laboratory tests.

The role of the laboratory during an epidemic of dysentery is two-fold: to confirm the diagnosis and to establish which drugs the organism responds to.

For these purposes, only a small number (e.g. 10–15 samples every 3–4 weeks) of stool samples need to be collected, transported and tested.

Special care needs to be taken in transporting stool samples containing *S. dysenteriae* type 1 from outlying areas to central laboratories. This is because Sd1 organisms die quickly if they are not stored correctly. Samples need to be kept in a special medium for transportation and refrigerated. The guidelines available soon from WHO contain more information

about appropriate supplies and equipment for laboratories and how to transport stool specimens.

The laboratory should not be used to diagnose all cases of dysentery during an epidemic. Once the organism causing the epidemic has been established, and an effective anti-microbial to treat it identified, all cases of bloody diarrhoea should be treated with that anti-microbial.

■ PRIORITY TREATMENT

The treatment for epidemic dysentery is anti-microbial drugs. Early treatment shortens the duration of illness and reduces the risk of serious complications and death. Ideally, all people with blood in their stools should be given anti-microbial treatment. Unfortunately, during an epidemic effective drugs may not be available for *all* patients with dysentery. It may be necessary to reserve treatment for those who are most likely to die if they are not treated.

Those most at risk are of dying from dysentery are:

- children less than two years old
- elderly people
- patients who are obviously malnourished
- patients with complications such as dehydration or fever.

To avoid situations where treatment with drugs needs to be restricted, WHO will assist national authorities to identify less expensive sources of effective drugs.

■ CHOOSING AN EFFECTIVE ANTI-MICROBIAL

Choosing the appropriate anti-microbial drug is not always easy. Over the last few years, the organism causing epidemic dysentery – *S. dysenteriae* type 1 – has become increasingly resistant to a variety of drugs. In some instances, only expensive or less widely available drugs are effective.

Wherever possible, laboratory tests should be done to find out which drugs Sd1 responds to (called establishing anti-microbial sensitivity). Drugs to which Sd1 bacteria are resistant in the laboratory should never be used to treat patients. Even if Sd1 responds to a drug in a laboratory, the drug still needs to be assessed for clinical effectiveness in patients.

The WHO guidelines suggest the following course of action:

- **When laboratory confirmation or information about anti-microbial sensitivity is NOT available**, the anti-microbial drug of choice is currently nalidixic acid. It is low-cost, has few side effects, and is widely available in most countries.

- When laboratory confirmation or information about anti-microbial sensitivity is possible, three key anti-microbials – nalidixic acid, ampicillin and trimethoprim-sulphamethoxazole (cotrimoxazole) – should be tested for sensitivity.

- When laboratory tests show that strains are resistant to nalidixic acid, ampicillin and cotrimoxazole, other anti-microbials such as pivmecillinam (amdinocillin pivoxil), ciprofloxacin and norfloxacin are likely to be effective against Sd1. However, these drugs are very expensive, so are not suitable for widespread use during an epidemic.

- Ineffective anti-microbials Sd1 has been consistently resistant to a wide variety of other anti-microbials, including sulphonamides, streptomycin, tetracyclines and chloramphenicol. These should not be used unless Sd1 has clearly been shown to be sensitive to them.

A number of other drugs have never been shown to be effective in patients, despite laboratory tests sometimes showing Sd1 bacteria are sensitive to them. These include: furazolidone, gentamicin, and cephalosporins.

MAIN POINTS

- The case definition of dysentery is diarrhoea with visible blood in stools.
- A dysentery epidemic should be suspected if there is a rapid increase in the number of cases, or deaths in a community from bloody diarrhoea.
- The cause of epidemic dysentery is almost always *Shigella dysenteriae* type 1.
- Anti-microbial treatment is required. Carry out laboratory tests on a small number of stool samples to verify the cause of dysentery and establish what drugs the organism is sensitive to. If testing is not possible, nalidixic acid is the anti-microbial of choice.
- All patients with dysentery should have their fluid intake increased and continue feeding. Patients should be monitored for dehydration, and given oral rehydration therapy if necessary.

When Sd1 is resistant to all available anti-microbials, cases of dysentery should be managed with supportive therapy alone – oral rehydration and appropriate feeding (see page 6).

MONITORING IMPROVEMENT

When a drug is effective, obvious improvement – increased appetite, decreased number of stools, less blood in the stool, less fever, and less abdominal pain – normally occurs within 48 hours.

Patients who do not show signs of improvement 48 hours after the start of treatment should be examined again. Treatment should change to an alternative anti-microbial to which Sd1 is likely to respond. This is because laboratory tests of anti-microbial sensitivity are not 100 per cent accurate. Failure of treatment does not mean that the illness is caused by another organism, and drugs for other organisms such as amoebiasis should not be given.

LONGER TERM MEASURES

After an epidemic has subsided, surveillance should continue to ensure that occasional cases of shigellosis are promptly detected and treated.

Efforts should be made to improve personal and domestic hygiene, water supplies and sanitation facilities to try to prevent further epidemics (see prevention, page 6).

Preparations should be made for dealing with epidemics at both national and district level. If further epidemics occur, control measures should be taken rapidly and efficiently.

Action to prepare for epidemics should include the following:

- supplies of oral rehydration salts, intravenous fluids and anti-microbials should be available at district and health facility level (see list of supplies below)
- laboratories should be equipped and staff trained to identify the cause of dysentery and to find out anti-microbial sensitivity patterns
- health care workers should be trained in case management of epidemic dysentery.

The experience gained during the course of one epidemic of dysentery should be used to strengthen the capacity of national diarrhoeal disease programmes to deal with all forms of diarrhoea.

Drug dosages for treating dysentery caused by Sd1

ANTI-MICROBIAL	ADULTS	CHILDREN
Ampicillin	500mg 4 times daily for 5 days	25mg/kg 4 times daily for 5 days (maximum 2g)
Trimethoprim-Sulphamethoxazole (TMP/SMX)	TMP 160mg and SMX 800mg* 2 times daily for 5 days	TMP 5mg/kg and SMX 25mg/kg 2 times daily for 5 days (do not exceed the adult dose)
Nalidixic acid	1g 4 times daily for 5 days	55mg/kg per day Divide into 4 separate doses and give 4 times daily for 5 days (maximum 4g)

*TMP/SMX is a fixed-combination product: the ratio of TMP to SMX is always 1:5.

Clinic supplies for 100 persons with dysentery

TREATMENT SUPPLIES	100 bars soap 30 boxes soap powder for washing clothes 2 bottles cleaning solution (2% chlorine or 1–2% phenol)
REHYDRATION SUPPLIES	100 packets ORS (for 1 litre per patient) 10 litres Ringer's lactate solution 5 scalp vein sets
ANTI-MICROBIALS	1600 x 1gm tablets of nalidixic acid Other anti-microbials may be needed instead of nalidixic acid, depending on local drug sensitivity
OTHER SUPPLIES	1 large container for drinking water 5 x 1 litre bottles for mixing ORS solution 10 glasses for drinking 5 teaspoons

Refugee crisis spreads disease

Studies in one of the African countries worst hit by the current epidemics – Burundi – have indicated possible risk factors for becoming ill with dysentery.

Over the last twelve years the east African state of Burundi has experienced regular annual outbreaks of dysentery, peaking in the rainy seasons (September to December).

In 1992 and 1993 particularly severe epidemics swept across the country. In 1992, almost 80,000 cases were reported – a national incidence of 14.2 cases per 1,000 people.

This year, a refugee crisis has increased the severity of the epidemic. As a result of political and social upheaval, hundreds of thousands of Burundians have fled to neighbouring Rwanda, Tanzania and Zaire, and spread the epidemic to refugee camps. WHO and the United Nations High Commission for Refugees are working with other relief organisations to coordinate a response to the epidemic.

Three studies in Burundi between 1990 and 1993 show that epidemic dysentery is a serious problem with high mortality rates, and that a rapidly changing pattern of sensitivity to drugs makes treatment difficult.

In 1990, Ries et al. collected 189 stool samples from patients in Gitega province with bloody diarrhoea. An organism causing dysentery was isolated in 125 samples. Of these, 66 per cent were *Shigella dysenteriae* type 1 (Sd1), and a further 25 per cent were other *Shigella* species.

Sd1 strains were resistant to nalidixic acid, ampicillin, cotrimoxazole, tetracycline and chloramphenicol. The only drugs Sd1 responded to – ciprofloxacin, pivmecillinam and ceftriaxone – were expensive and were not available in large quantities at short notice.

A community survey of 9,300 inhabitants in the same province was conducted by Birmingham et al. in February to September 1992. The incidence of bloody diarrhoea during the epidemic was found to be 13.9 per 1,000 people. Incidence increased with age. Possible risk factors for becoming ill included: use of a cloth rag for anal cleansing following defecation; recent loss of weight; and little or no schooling.

In March 1993, Murray et al. followed up 775 patients who had reported to health facilities in Muramvya province with bloody diarrhoea in the previous six

months. Seven per cent of patients (including many who had received treatment) had died. The median interval between the onset of symptoms and death was 13 days.

The researchers also collected 133 stool samples, 35 per cent of which yielded Sd1. Significantly, the resistance pattern to drugs had changed.* While Sd1 was still resistant to ampicillin and cotrimoxazole, it was now sensitive to nalidixic acid, as well as the more expensive drugs found to be effective before – ciprofloxacin, pivmecillinam and ceftriaxone. This meant that nalidixic acid was the clear drug of choice.

The Burundi studies show that anti-microbial sensitivity patterns can change rapidly. Therefore, active laboratory monitoring systems need to be established before the onset of an epidemic. The studies also indicate that much more needs to be found out about risk factors and transmission.

Adapted from Manirankunda, L et al., 1993. The epidemiology of bacillary dysentery in Burundi. Bulletin Epidémiologique du Burundi, July–September.

**Although the studies were done in different provinces, there is no reason to suggest that sensitivity patterns would differ from province to province during a nationwide epidemic.*



Dysentery is a major cause of death in refugee camps on the Burundi-Rwanda border

Marc Schlossman/Panos Pictures

South Asia also affected by Sd1

During a south Asian epidemic in 1976, *Shigella dysenteriae* type 1 spread from south India to Sri Lanka. Sd1 is now endemic in Sri Lanka, with epidemics occurring periodically.

Problems of civil unrest and migration of refugees from 1985–1992 led to a sharp increase in cases of dysentery, with the number of cases more than tripling (from 79 cases per 100,000 people, to 245 per 100,000) in this period.

Lack of awareness about the way the disease spreads has been a major factor in epidemic transmission. A study in the town of Galle showed that while more than 90 per cent of the adult population used toilets, the stools of about 70 per cent of children were

disposed of outside, in open pits or simply left on exposed ground.

The anti-microbial sensitivity pattern has changed considerably between epidemics. In the first epidemic between 1976 and 1978, Sd1 was sensitive to nalidixic acid. During the next epidemic (1978–9) Sd1 continued to respond to nalidixic acid. However, between 1982 and 1989, Sd1 was found to be resistant to nalidixic acid, but was sensitive to other drugs at various times, including pivmecillinam.

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Supportive treatment is vital

In addition to life-saving anti-microbial treatment, all patients with dysentery caused by Sd1 need to drink more and to continue normal feeding.

■ INCREASING FLUIDS

It is crucial for people with dysentery to drink more liquids (including plain water) in order to prevent dehydration.

Readily available home fluids (such as yoghurt drinks; water in which a cereal has been cooked; unsweetened tea; green coconut water; and fresh, unsweetened fruit juice) are good choices. If possible, dysentery patients should also be given a fluid that contains salt, e.g. salted soup, salted rice fluid or oral rehydration fluid.

Patients with dysentery should be assessed regularly for signs of dehydration. The key signs are: increased thirst; restlessness; irritability; and loss of skin elasticity (when the skin is pinched and released it does not flatten immediately). If dehydration becomes severe, a patient may become lethargic or unconscious and be unable to drink.

If the patient shows signs of dehydration, they should be given rehydration fluid immediately. There are three main types of rehydration fluid: oral rehydration salts (ORS) solution, sugar-salt solution (SSS) and cereal-based solutions. Oral rehydration fluid should be given at a steady rate in small amounts. Children under two years old should receive at least ¼–½ cup of rehydration fluid after each loose stool. Older children should receive at least ½–1 cup. Children over 10 years old should drink as much rehydration fluid as they want.

For more information about oral rehydration therapy, see DD52.

■ CONTINUING FEEDING

Continuing to give nutritious food to people with dysentery is very important. A major complication of dysentery is weight loss and rapid worsening of nutritional status. This is because people with dysentery often have reduced appetites, yet their bodies need more nutrients than usual in order to fight infection, repair tissue damage, and replace nutrients lost during diarrhoea.

Even when patients survive dysentery, resultant malnutrition may increase their vulnerability to other life-threatening illnesses.

In general, the same foods should be given during dysentery as those a patient eats when he or she is well. Meals may need to be given in smaller amounts more frequently, and carers should gently but

persistently encourage people with dysentery to eat. If possible, food rich in potassium such as spinach, avocado pears, bananas and coconut water should be given.

Even if patients are well fed, they may have lost weight and be malnourished after the dysentery episode is over. Providing an extra meal every day for two weeks can help to restore lost weight.

If infants with dysentery are normally breastfed, mothers should continue to breastfeed them frequently. If infants under four months old normally receive other foods in addition to breastmilk, these should also be continued during an episode of dysentery and mothers encouraged to breastfeed frequently. However, after the dysentery episode health workers should find time to encourage these mothers to practise exclusive breastfeeding until their infants are at least four months old.

For more information about feeding during diarrhoea, including when diarrhoea lasts 14 days or more, see DD53.



WHO/UNICEF/NIKONRAT

Eating well can ensure a good recovery after a dysentery attack.

Prevention strategies

Like other forms of diarrhoea, Sd1 infection is spread through human faeces. When people become infected with Sd1, they excrete large numbers of Sd1 organisms in their stools. If germs from these stools come into contact with food or water, other people can swallow them and become infected. In addition, Sd1 bacteria are so infectious that sufficient organisms to cause disease can be spread from one person's hands to another's.

The only proven ways of preventing infection and transmission of all forms of *Shigella* are handwashing with soap and breastfeeding. Methods for preventing other forms of diarrhoea are also likely to reduce transmission of Sd1, although there is no research showing this. These methods include: the promotion of commercial and household food hygiene; the provision of adequate supplies of clean water for drinking; and the safe disposal of human faeces.

Handwashing

Thorough handwashing with soap appears to be the single most effective way to prevent transmission of all forms of *Shigella*. The key times for handwashing are: after defecation;

after cleaning a child who has defecated or after disposing of their stools; and before preparing or eating food. Health care workers should wash their hands before and after examining each patient and before giving ORS or food to a patient.

It is now known that people are more likely to wash their hands if they have easy access to a plentiful supply of water. Water for washing and drinking should be stored in different containers.

If soap is not available, ash or mud can be used. If possible, in areas affected by dysentery epidemics, soap should be distributed to families who cannot afford it.

After handwashing, hands should be dried with a clean cloth or left to dry naturally in the air. Hands should not be dried with a dirty cloth.

Breastfeeding

Breastfed infants are much less likely to get dysentery than other infants. If breastfed infants do get dysentery, their illness is likely to be much milder than in infants who are not breastfed.

For more information about prevention, see DDs 54, 45 and 44.