STRATEGIC RESPONSE TO
EPIDEMIC DYSENTERY
IN AFRICA

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EXECUTIVE SUMMARY

Over the last several decades, Africa has experienced epidemics of cholera and dysentery due to *S. dysenteriae* type 1. These epidemics have occurred in addition to the usual diarrheal diseases experienced in most developing countries. Both dysentery and cholera epidemics have affected large numbers of persons, have been associated with high case fatality rates, have affected all age groups and their control has exceeded the capabilities of the national health authorities. Furthermore, the dysentery epidemics have been due to strains resistant to nearly all antibiotics and the antibiotics which have been used were not the appropriate ones. While cholera epidemics have been scattered over most of the continent, the dysentery epidemic has involved a more limited number of countries in Central Africa.

This report supports the concept proposed by WHO that, because of the similarities in the transmission of the two diseases, and similarities in many of the control activities (logistics, training, IEC), a coordinated control program aimed at both diseases is appropriate. The similarities between the two should not however mask the differences in transmission, symptoms, case management, and clinical complications. Programs dealing with these epidemic diarrheal diseases will have to take into account the differences as well as similarities. Whatever strategies are developed should include preparations for the new strain of cholera (Bengal O139) which is now spreading more quickly than any previous cholera epidemic and will likely reach Africa during the next year.

Further, this report supports the concept that the control of epidemic diseases be carried out in a manner which builds on the strengths of the CDD program as well as building up the capability and stature of the program.

There is no single "magic bullet" which will control dysentery. In this regard dysentery is a more difficult problem to control than other diarrheal diseases. Several interventions can, however, decrease the risk of acquiring the infection and / or decrease the risk of complication or death and innovative strategies using these interventions are needed which involve the medical community as well as the private sector. Expectations of eradicating the disease or of lowering case fatality rates to < 1% are however unrealistic given current technologies and resources.

USAID, through its projects and through its funding of international organizations can play a key role in the control of epidemic dysentery in Africa. The specific activities with which USAID has a comparative advantage are shown on an activity grid.

Dysentery is different from "ordinary diarrhea" in that an epidemiologic surveillance and a laboratory system is needed to detect and track epidemics, and to monitor antibiotic resistance. This surveillance system would be greatly aided by the development of simple rapid tests to
avoid the need for building up basic laboratory services. Social science studies are needed to fully understand the modes of transmission and the culturally acceptable changes which could interrupt the spread of the agents. Behaviors such as water collection and storage, food preparation and storage, personal hygiene, and toilet habits are all important to control of these diseases.

The interrelation of these epidemic diseases with other common diseases are also important to understand. Especially important is the potential inter-relations between shigellosis and AIDS, malnutrition and malaria since these are all common in the same geographic areas.

Epidemic dysentery and cholera are major public health problems in Africa and the donors interested in health should take a more active role in assisting countries deal with them.
INTRODUCTION

Shigellosis, Critical Public Health Problem

Shigellosis, also known as bacillary dysentery, is an acute infectious enteritis of human and subhuman primates caused by bacteria of the genus *Shigella*. It usually causes frequent passage of small-volume, bloody mucoid stools, accompanied by abdominal cramps, and tenesmus (rectal pain). Life-threatening complications of shigella infection include hemolytic uremic syndrome (HUS), encephalopathies, colonic ulceration and perforation, shigellemia, toxic megacolon, intestinal stenosis and obstruction, persistent diarrhea, as well as severe malnutrition and wasting. Disease can result from infection with any one of the 4 known shigella species, *Sh. sonnei*, *Sh. flexneri*, *Sh. boydii* and *Sh. dysenteriae*. Each one of these species - with the exception of *Sh. sonnei* - can be grouped according to type-specific antigens into several serotypes [*Sh. dysenteriae* (10 serotypes), *flexneri* (6 serotypes), *boydii* (15 serotypes), and *sonnei* (1 serotype)]. Among all existing serotypes, only *Shigellae dysenteriae* type I (or Shiga’s bacillus) is known to be able to cause outbreaks of epidemic proportions.

Shiga epidemics exacerbate the already important burden of endemic shigellosis (due to other shigella species and other *Sh. dysenteriae* serotypes) in developing countries. It has been estimated that over 140 million cases and 576,000 deaths occur annually due to shigella infection in children under 5 years of age worldwide. Because of problems in clinical and laboratory diagnosis, these numbers are probably grossly underestimated. In endemic settings in developing countries *Sh. flexneri* is the predominant species. For reasons not entirely understood, *Shigellae dysenteriae* type I eventually goes from playing a minor role in the etiology of shigellosis to suddenly and dramatically becoming the dominant enteric pathogen of massive dysentery epidemics.

PART I - REVIEW OF SHIGA EPIDEMICS IN AFRICA

The Dimensions of the Problem in Africa

I. Reappearance of Major Shiga Epidemics in Central Africa

Epidemics of *Shigellae dysenteriae* type I (SD1, Shiga) have taken place in Central African countries for at least the last sixty five years. During a huge outbreak in the Belgian Congo (Zaire) in 1928-1932, shiga dysentery is known to have killed half of the cases in some
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of the affected areas.\textsuperscript{2} Eleven years later, it became an important problem in Zaire again, as well as in Rwanda and Burundi. This new epidemic moved through Central Africa affecting a population already devastated by the effects of the serious 1943-1945 famine. The reported case-fatality rate at that time approached 25 per cent.\textsuperscript{3} The next reports on Shiga outbreaks came from Somalia during 1963-1964\textsuperscript{4} and 1976.\textsuperscript{5} In November of 1979, 28 years after the last reported isolation of SD1 in Central Africa\textsuperscript{3}, a multi-resistant SD1 reappeared in a massive epidemic in North East Zaire.\textsuperscript{6} The SD1 epidemic then subsequently involved Rwanda (1981)\textsuperscript{7,8}, Burundi (1981)\textsuperscript{9}, Tanzania (1981)\textsuperscript{10}, Ethiopia (1983)\textsuperscript{11}, Zambia (1990)\textsuperscript{12}, and possibly Uganda, Central Africa Republic, Zimbabwe, Namibia and Angola. In 1992, Malawi and Mozambique also started reporting increasing cases of SD1 bacillary dysentery.\textsuperscript{13}

Genetic studies of SD1 strains isolated in Zaire and Rwanda in 1981 and 1982 showed great similarities with strains isolated in Somalia in 1976, suggesting that the outbreak in these countries, and probably in most of all the other Central Africa countries involved so far, may be epidemiologically related, reflecting the spread of a single \textit{Sh. dysenteriae} type 1 clone, whose plasmid content changed in response to antibiotic pressures.\textsuperscript{14,15}

A detailed summary of available country-specific shigellosis data with corresponding references are presented in Appendix 2.
Countries Affected By Dysentery Epidemics

- Zaire 79
- Rwanda 81
- Burundi 81
- Zambia 90
- Ethiopia 83
- Somalia 76
- Tanzania 81
- Mozambique/Malawi 92

Probable: CAR, Uganda, Angola, Namibia, and Zimbabwe

Confirmed:
II. Epidemiology of Current Central-Africa Epidemics

2.1. Shiga Dysentery Incidence and Attack Rates

In order to calculate shiga infection rates, one needs to detect not only cases of classical dysentery syndrome, but also mild and asymptomatic infections in the community that are not usually recognized by facility-based surveillance systems.

Both bacteriological and serological surveys can be used in prospective community-based studies to more accurately determine the incidence, prevalence, and risk factors for infection with SD1. To our knowledge, no such studies have been conducted in the countries currently affected by Shiga epidemics. Most estimates of incidence and prevalence in Central Africa relied on clinically apparent dysentery, and therefore represent the incidence of symptomatic disease and not necessarily the overall spread of SD1 infection in the community.

Data from non-Central African countries indicate that in hyper-endemic situations, the overall subsequent infection rates in contacts of SD1 cases are around 20-30 per cent, secondary case rates are around 10-13 per cent, and infection-to-case ratio are around 1.5:1 to 2.6:1 (about 10 out of 15-26 contacts developed symptomatic disease). In Bangladesh secondary infection rate was higher for the 0-4 age group (close to 31%). All children in this age group developed symptomatic illness (case-to-infection ratio of 1:1), requiring hospitalization in 25 per cent of the cases. Of the secondary infections, 34.7 per cent were symptomless, 55.3 per cent were mild to moderate and 10 per cent were severe enough to require hospitalization. For one hospitalized case there were seven symptomatic cases and 10 infections (i.e. infection-diarrhea-hospitalized ratio of 10:7:1).

It is important to keep in mind that SD1 epidemics in developing countries take place in settings with already high rates of endemic shigellosis. In 1954 a community-based bacteriological survey in Egypt showed that, in an endemic situation, the incidence of symptomatic shigella infections was 1 infection per person per year, and an equal number of asymptomatic shigella infections were detected so that the total incidence was 2 infections per person per year.

2.1.1. Absolute Numbers and Overall Rates:

At the beginning of the current Central Africa epidemic, the overall attack rate reported for Cyangugu, Rwanda was 5 per cent, and 6.4 per cent for Zaire (10.9, 8.8 and 3.0 per cent for women, men and children, respectively). In the 1979 epidemic in Zaire, it was estimated that over 100,000 persons were ill with SD1 dysentery, with 5-10,000 deaths nationwide. In
Ethiopia, a total of approximately 5,000 cases were reported during one month interval, and the overall attack rate was 7.3 per cent. In Zambia, from June 1990 to November 1991, there were close to 30,000 dysentery cases reported nationwide. In Burundi, the reported numbers have been much higher. There were 110,361 dysentery visits in 1991, 75,532 visits in 1992, and more than 50,000 since the beginning of 1993. This makes dysentery the fifth most common cause of health care visits in Burundi. The annualized national incidence rate of dysentery for 1991 in Burundi was estimated to be 16.3/1000 person. In other words, one in every 60 persons was affected in Burundi during 1991.

Since 1990, Burundi has received CDC assistance in the epidemiological investigation of SDI dysentery and the increased numbers reported since then may reflect in part improvements in disease surveillance. Increased awareness within the medical community, coupled with improved clinical and laboratory diagnosis, may partially explain why in 1991-92 the number of dysentery visits to health centers practically doubled compared to previous years. However, CDC experts believe that there has been a real increase in numbers above and beyond what could have been expected to have occurred with the introduction of a better surveillance system. An evidence for that was the presence of good patterns of health care use in this population. Most dysentery cases (76%) identified in a community survey in Burundi reported having seek care during their illness. Therefore, passive clinical surveillance relying on reports of dysentery visits to health facilities is believed to have identified the majority of symptomatic cases and accurately reflected the real increase of dysentery in this community.

2.1.2. According to Age:

While endemic shigellosis is a childhood disease, epidemic shigellosis can affect all age groups, but with higher incidence among adults.

In Rwanda, all age groups were affected, with the highest number of cases among the 20 to 24 year olds. Seventy two percent of the cases were over 18 years of age. In Zambia, the majority of patients with dysentery were also adults (over 14 years of age). The annualized rate of hospitalization for dysentery at the University Teaching Hospital was almost three times higher for adults than for children. Among adult cases, the 20-29 year age group had the most patients (46%). The proportions in the different age groups were similar for men and women. In Tanzania, a study of hospitalized dysenteric patients revealed that 63 per cent of the cases were over the age of 10 and that the most affected group was the 20-29 year olds (53%). In Ethiopia, the attack rate for children between 1 and 4 years of age was 22.6 per cent and 30.6 per cent for 25-44 year olds. In 1991, the mean age of persons reporting dysentery in Kibuye, Burundi was 35 years, with 77 per cent of cases over the age of 10 years. Incidence was approximately 15/1000 population at 1-4 years and as high as 78/1000 population among individuals over 60 years of age.
Predominance of epidemic disease among adults suggests recent introduction of the SD1 strain into a susceptible adult community, lowering of general hygiene and environmental sanitation, and/or increased vulnerability to infection among the most affected age group. Concentration of adult cases in the early twenties age group may also reflect increased risk of exposure to infection out-side household.

2.1.3. According to Sex:

The study of hospitalized cases in Rwanda showed a predominance of females in the age groups over 5 years, with the reverse being observed for children under 5 years of age. In Burundi, there was a significantly higher incidence among females (16/1000/yr) than males (11/1000/yr) in the 1991 epidemic, but no gender difference was observed during the 1992 epidemic. The inverse, although to a lesser degree, was observed in Zambia during the 1990-1991 epidemic, where male cases predominated (63%). However, gender-specific incidence rates were not reported for the Zambian epidemic. In Tanzania, males accounted for 61.4 per cent of all cases and prevailed in all age groups.

Gender differences in case rates seem to be country- or region-specific and may reflect distinct socio-cultural or occupational patterns within each country that could differentially increase the risk of exposure or the susceptibility to infection, or limit access to care.

2.1.4. Seasonality:

Changes in seasons seem to be an important factor leading to the rise in the number of cases during certain periods of the year. The situation in Burundi offers a good example of SD1 seasonality. Annual epidemics have been occurring during the rainy season - a higher number of cases occur from September through December - every year for the last 10 years. Isolation of SD1 from patients in Butare (southern Rwanda) between February 1985 and February 1987 also showed an endemic profile with seasonal variation, with peak incidence from September to March.

Epidemiological features in the environment are that lead to seasonal transmission are not well understood. It has been suggested that peaks during the rainy season may be related to increased contamination of water supplies, or seasonal worsening of nutritional status and increased susceptibility to infection.

In Calabar, Nigeria, peak isolation rates of endemic shigella species occur during the dry weather periods. This seasonal variation has been attributed to acute water shortage and use of water from contaminated streams during this period.
2.2. Mortality

2.2.1. Overall Rates and Ratios:

Dysentery mortality is associated with inadequate treatment. In the Zaire epidemic in the early eighties, the overall case-fatality rate (CFR) according to drug resistance patterns was: 2.0 - 2.4 per cent when effective treatment was in use and 4.6 per cent when antibiotic resistance developed. In Rwanda, the CFR dropped from around 10 per cent at the beginning of the epidemic to 2 per cent after the introduction of adequate antibiotic treatment. In Zambia, regional CFRs ranged from 0.4 to 10.8 per cent during 1991. However, these values were considered to be underestimated because not all deaths were being appropriately reported during the Zambian epidemic.

A community survey of recent history of dysentery indicated a CFR of 3.2 per cent in Burundi during 1991. Due to strong cultural reluctance of discussing death, these mortality data were believed to be underestimated by a factor of at least three. During the 1992 epidemic in this same country, a facility-based study revealed an overall case-fatality ratio of 7.2 per cent. Essentially, 1 out of 14 patients died, with most dying in the first 10-14 days. During 1991 and 1992, almost all detected cases in Burundi were being treated with drugs found to be ineffective against the epidemic strain, and this could have contributed to the high attendant mortality rate observed.

In the 1979 epidemic in Zaire, the overall mortality rate was 1.6 per 1000. In Zambia, the overall annualized rate of deaths from dysentery was 1.97 per 100,000 persons per year in 1991. A facility-based evaluation of cases occurring in November 1991 showed that 1 out of 25 deaths was due to dysentery. In Burundi, the annual dysentery mortality rate was estimated to be 1.53 per 1000 per year in 1991 and the proportional mortality rate due to dysentery was estimated to be 9.1 per cent. In other words, approximately 1 out of 11 deaths in Burundi during 1991 was due to dysentery.

2.2.2. According to Age:

CFRs were higher in children and older adults. In Rwanda, attendant CFRs were of 12, 9 and 4 per cent in children, adult males, and adult females, respectively. In Zambia, attendant CFRs were of 15 per cent in children and of 4 per cent in adults. In Burundi, CFRs were high in all age groups, but especially so in the young children and older adults - 7.3 per cent for those less than 5 years of age, 5.8 per cent for those between 5 and 49 years, and as high as 18.5 per cent for individuals over 50. Zaire was the only country that reported lower CFR for children in comparison to adults (1.9 vs 2.5 per cent).

In Zaire, mortality rates were around 2.5 per 1000 for adults and 0.5 per 1000 for
Epidemic Dysentery in Africa

children. In Zambia, fatality rates were highest among children below 4 years (5.5 per 100,000 per year). In Burundi, the largest proportion of deaths also occurred in the youngest age-group.

2.2.3. According to Sex:

In Zaire, CFRs were comparable among males and females (2.8 vs 2.5 per cent). In Rwanda, CFR in adult males were higher than in adult females (9 vs 4 per cent).

In Zambia, death rates per 100,000 population per year were higher among men than among women (2.36 vs 1.59). Among men, the highest rate was among the 20-29 (3.34/100,000/yr) and 40-49 year-olds (3.64/100,000/yr). Among women, the highest rates were among those 50 to 59 years (4.78/100,000/yr).

2.4. Risk Factors

Current knowledge on SDI epidemiology is incomplete and needs further investigation. All studies of risk factors for dysentery in Central Africa were conducted as case-control investigations, and were therefore selective for typical bloody diarrhea cases in the studied areas. No study investigated rates of asymptomatic infection, case-to-infection ratio, risk factors for secondary spread of shigellosis, risk factors for development of clinically apparent illness, and characteristics of asymptomatic carriers. In the future, it would be of interest to determine who is at a greater risk of becoming clinically ill after initial exposure to SDI.

2.4.1. Risk Factors for Disease:

In Burundi, a case-control study of patients seeking care for bloody diarrhea during November 1990 identified that among shigellosis cases, those with SDI infection were more likely to have had contact with a person with dysentery and to have recently taken an antibiotic. SDI patients did not differ significantly from non-dysenteric controls, except for history of diarrhea in the previous months, and recent contact with a person with dysentery. There were no differences in regards to the number of persons in the household, diet, water source, and
amount of water available for the family. Approximately 25 per cent of individuals in dysenteric households (aside from index case) also reported dysentery.

In February 1992, a community case-control study identified factors associated with past history (last six months) of dysentery in Burundi. Individual risk factors included being of older age, being female, using a cloth rag for anal cleansing after defecation, weight loss before disease onset, and no education. No association was observed for activities such as going to the market, travelling, receiving visitors, or taking care of a sick person. Significant household risk factors identified included carrying water in an open bucket, not having soap in the house, and not washing hands before preparing food. Households with and without dysentery were comparable in regards to indicators for crowding, socio-economic and sanitary status.

In 1992, a facility-based case-control study in Zambia identified the following individual risk factors for dysentery: eating food (meat and vegetables) purchased from the market or street vendor, having a family member with dysentery, and having recent contact with a person with dysentery. There were no significant differences regarding frequency of market visits, number of meals eaten outside of the home or number of meals from a common plate. Households risk factors included: food preparer with history of dysentery, drinking water obtained by hand-dipping a cup in wide-mouth water container rather than by pouring it out, having open latrines, and sharing latrines with other households. No significant differences were found in other aspects of hygiene such as availability of soap and toilet paper, wash water volume, distance to water source or cleanliness of latrine.

In addition, studies conducted outside Africa have identified lack of breastfeeding as an important risk factor for symptomatic shigellosis in children.

2.4.2. Risk Factors for Severity:

Mortality has been higher among the very young and the very old. In Burundi, the largest proportion of deaths occurred in the youngest age-group. The probability of survival 100
days after the onset of dysentery was significantly lower for the oldest age group than for the other age groups. Individuals who waited longer than one day before going to the clinic were less likely to survive than individuals who went to the clinic earlier. That may reflect the effect of delayed treatment or other underlying conditions associated with increased risk of death, such as low socio-economic status. There was no association between completeness of treatment and survival. In Zambia, many of the deaths in the Southern region were reported to be among fishermen. This was the only reference regarding occupational risk factors in the available literature for Central Africa SD1 epidemics.

The influence of nutritional status in infection incidence and outcome was not reported in most studies of the Central Africa epidemics. Nonetheless, studies in other developing settings have identified an association between isolation of shigella and the presence of malnutrition. Nutritional status was also found to affect chronicity of established illness. Malnourished individuals may develop chronic relapsing disease extending over months. In addition, shigella infection itself may cause severe malnutrition which is strongly associated with mortality.

### 2.4.3. Shigellosis and the HIV epidemic

As of mid 1993, the World Health Organization estimates that over 8 million adult HIV infections have occurred in sub-Saharan Africa. Of this total, about half to two-thirds have been in East and Central Africa. HIV prevalence among adults in major urban areas may exceed 20 per cent, but varies widely according to risk groups.

All Central African countries currently affected by Shiga epidemics also face an increasing AIDS burden. The cumulative number of AIDS cases reported to the WHO for some countries in Central Africa are presented in Table 1. The extent to which shigellosis and HIV infection interact in these countries needs to be further evaluated. Diarrhea is common in individuals infected with HIV and may occur in up to 90 per cent of patients with AIDS. Although the association of shigellosis and HIV has not been fully explored in developing countries, it is reasonable to expect that HIV positive individuals in shigella endemic and epidemic countries would be at a higher risk of acquiring shigella infection and developing symptomatic disease with potentially more severe progression. In one study in Kenya, about 30 per cent of nosocomial diarrhea in HIV positive individuals (free of shigella infection on admission) were due to salmonella or shigella infections, in contrast to 15 per cent in HIV negative individuals. Twenty-eight per cent of adults with nosocomial diarrhea had a diagnosis of HIV infection. In Burundi, weight loss prior to dysentery onset was a significant risk
factor for SD1 shigellosis. Even though weight loss is certainly a non-specific finding, it could, among other things, represent a marker for HIV wasting syndrome.

A study in Kenya showed that shigella antimicrobial multiresistance was a serious problem among AIDS patients. In such patients, selection of or increased vulnerability to multiresistant strains may be due to their intense drug use as a result of AIDS.

### 2.5. Sources and Routes of Transmission

Shigellosis is transmitted by fecal-oral contamination directly through person-to-person contact (direct [hand-to-hand-to-mouth] or indirect [hand-to-fomites/food/water-to-mouth]), or indirectly through fecal contamination of food (foodborne) and water sources (waterborne), and houseflies (fly-borne). A variation on the mechanism of fecal-oral transmission has been demonstrated among homosexual men engaging in anal-oral sex practices.

**Person-to-person** contact seems to be the most important mode of shigellosis transmission. It is believed that this may also be the case during epidemic circumstances. In Central Africa, direct transmission within families, hospitals and institutions apparently played a more important role than other modes of transmission. However, a combination of direct (hand-to-mouth) and indirect contamination (waterborne, foodborne spread) is likely to have occurred in the settings where inadequate hygiene and sanitary patterns prevailed. The relative importance of housefly in comparison with other routes of transmission is not known, but is likely to be minimal.

Shigella species are potentially the most communicable of bacterial pathogens. Shiga's bacillus are transmitted very efficiently through the fecal-oral route. The infectious dose for symptomatic infection of shigella can be as low as 10 organisms. Large numbers of shigella ($10^5$ to $10^8$/g) are present in stools of clinical cases and healthy carriers ($10^2$/g). This level of

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</table>

* 1991 rate.
contamination combined with conditions which facilitate the spread of the inoculum needed to initiate the infection may explain the rapid involvement of large numbers of individuals in communities with poor sanitary conditions and hygienic practices. Most risk factors identified in Central Africa were related to person-to-person spread.

However, low secondary attack rates (SAR) were detected in Central Africa epidemic despite the postulated efficient person-to-person spread. Household transmission was estimated to account for only 7 per cent of the cases in Burundi. A similar rate was identified in a study in Dhaka, Bangladesh where only 13.3 per cent of the contacts of SD1 index patients developed symptomatic illness.

One possible explanation for low SAR is the presence of certain individual host characteristics associated with differential susceptibility to overt SD1 infection among members of the same household (e.g. hypochlorhydria and blood group type O are known risk factors for cholera). Household contacts may also differ in their levels of acquired immunity due to past exposure to SD1 and other shigella species (cross-immunity) and therefore respond differently against the new infection.

Finally, SAR does not reflect the actual person-to-person spread of SD1 among family members. To fully evaluate the role of direct contact as a mode of transmission in Central Africa, the proportion of secondary asymptomatic infection, the case-to-infection ratio, and the distinction between concurrent and secondary cases would need to be investigated.

Institutionalized populations also play an important role in SD1 spread. In Zambia, the first outbreak apparently started in one prison and spread to other custodial institutions through transferring of infected prisoners. The epidemic eventually found its way to the community at large, but the mechanism involved in this process was not fully investigated. In Tanzania, close to 11 per cent of hospitalized cases appeared to have become infected in institutions such as schools, prisons and work places; and 1 per cent were nosocomial infections.

Another point of uncertainty in SD1 epidemiology is the mechanism through which the organism maintains itself in the community for long inter-epidemic periods. It is not clear why after almost 30 years of absence, SD1 has emerged again as the most common cause of shigellosis in Central Africa. The role of environmental contamination in the maintenance of SD1 disease in the community is not well understood, and the possibility of a viable, but non-culturable form in the environment needs to be investigated.
III. Obstacles for Control

3.1. Obstacles for Reduction in Morbidity

Inadequate sanitary infra-structure, poor standards of personal hygiene, lack of early recognition of the epidemic, inadequate laboratory facilities, supplies and expertise, and inappropriate antibiotic treatment, along with lack of appropriate understanding of the epidemiology of \textit{Sh. dysenteriae} type 1 infections and its interaction with humans have led to rapid and massive uncontrolled spread of SDI infection in Central Africa.

In Zaire, it took more than a year to identify the agent responsible for the 1979 epidemic.\textsuperscript{6} In Zambia, more than one year into the epidemic, there were still conflicting reports identifying the epidemic as due to \textit{Entamoeba hystolitica}, \textit{Shigellae dysenteriae} type 3 and \textit{E. coli}.\textsuperscript{12}

Bacteriological diagnosis and drug resistance characterization have not always been available at the regional level in quality-controlled surveillance laboratories. There has been a lack of clear guidelines on collection, storage and transport of specimens. For example, Salmonella-Shigella (SS) agar has been a commonly used culture medium in Central Africa. However, SS agar is too inhibitory to shigella, especially SDI, and should not be recommended unless it will be used together with other screening media.

Poor environmental and personal hygiene standards are key elements for the spread of Shiga's bacillus. Lack of information on behavior patterns and of individuals' perception and acceptability of preventive measures hinders the design and implementation of appropriate interventions.

3.2. Obstacles for Reduction in Mortality

Adequate treatment reduces considerably the length and the lethality of the disease.
Epidemic Dysentery in Africa

Delayed or incorrect antibiotic treatment is associated with higher case-fatality rates. It is not clear whether use of inappropriate antibiotic treatment in Shiga infection will just do no good or whether it will actually cause harm. Ineffective antibiotics against SD1 may aggravate the patient’s condition by altering the normal gut flora, allowing for more aggressive shigella growth. Incomplete therapy is also a major problem and may contribute to selection of resistant strains. In Burundi, 70 per cent of patients under 5 years of age received an incomplete course of treatment.25

An important feature of SD1 has been its rapid adaptation to changes in antimicrobial therapy. SD1 patients in Central Africa were initially treated with tetracycline which was replaced by trimethoprim-sulfamethoxazole during July of 1981. By September about 42% of Shiga strains from Zaire, Rwanda and Burundi were resistant to trimethoprim. In Zaire, 75 per cent of the strains were resistant to co-trimoxazole by October 1981. In July 1982, two-thirds of the strains were again sensitive to co-trimoxazole.6

Nalidixic acid (NA) was then introduced to combat resistant strains.15,38 In April 1982 the first NA-resistant strain was isolated in Kivu, Zaire15,39 and by July of 1985 the majority of the SD1 isolates in this area were NA resistant.40

Since 1984 nalidixic acid was recommended for systematic use in Rwanda. The first case of SD1 resistance to NA in Rwanda was seen in February 1985, and during the period from November 1985 to February 1987 thirty-one percent of the species isolated were NA resistant.26

In Burundi, SD1 isolates were resistant to chloramphenicol, tetracycline, sulfisoxazole, nalidixic acid, ampicillin, streptomycin, and trimethoprim-sulfamethoxazole.29 In contrast with 1991, 1993 isolates were susceptible to nalidixic acid.25

WHO has recommended that antibiotic therapy be administered only SD1 patients at higher risk of severe outcome, namely the very young, the very old, malnourished individuals and those with severe illness. Adults with mild disease should receive supportive therapy alone.41 This recommendation is specially important in areas where antimicrobial supplies are limited.

The new quinolones, such as ciprofloxacin and norfloxacin, may become the future drug of choice, since clinical tests seem to indicate their efficacy against dysentery.40 However, the cost of these drugs may be prohibitive. In addition, ciprofloxacin, norfloxacin and enoxacin have not been approved for use in children. Drugs not recommended for SD1 treatment include: aminoglycosides (kanamycin, gentamicin), amoxicillin, nitrofurans (furazolidone, nitrofurantoin, furoxone, furadantin, macrodantin), and erythromycin.
Table 2 - Drugs used during Central Africa *Shigella dysenteriae* type I epidemic and year resistance developed.

<table>
<thead>
<tr>
<th>YEAR</th>
<th>79</th>
<th>81</th>
<th>82</th>
<th>84</th>
<th>85</th>
<th>90-92</th>
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</tbody>
</table>

○ = Reversed resistance
✓ = Development of resistance
I = Recommended for use

Availability of treatment depends on adequate drug supplies and adequate distribution systems. An inadequate drug procurement system can lead to arrival of recommended antibiotic after the epidemic peak has already taken place. In Zaire, for example, it took 3 months to overcome administrative barriers, allocate funds, find a company able to produce quickly large amounts of nalidixic acid at reasonable cost, and finally deliver the drug to affected areas.6

In countries where cholera is also epidemic, there is the concern that drugs designated for the treatment of multi-resistant Shiga infection could be misused in the treatment of cholera cases. Wide-spread utilization of such drugs is bound to contribute to resistance development.

**IV. Current Involvement of Donor Organizations**

CDC has provided technical support for diagnosis and epidemiological investigations in
Epidemic Dysentery in Africa

Central Africa since 1990. In Burundi, the CDC collaboration has been very beneficial to the CDD program, University Teaching Hospital and Public Health laboratory staff. CDC has also offered support to improve surveillance and to establish a sentinel site system. It is also in the process of planning a demonstration trial of a community intervention based on health education and soap distribution. CDC has proposed that research priorities should include: a) Clinical trials to evaluate alternative drug therapies, b) Ways to strengthen national and regional health surveillance and laboratory-based surveillance to monitor trends and the spread of antimicrobial resistance, c) Decision models in case-management and preventive strategies, d) Procurement mechanisms to quickly respond to resistance pattern changes, e) Community-based trials to assess the feasibility and efficacy of preventive measures with initial focus on handwashing with soap, f) Perform additional epidemiological studies to better understand modes of transmission and risk factors for severity and death. (Personal Communication with Dr. Lisa Lee, CDC)

WHO has concentrated on epidemic preparedness efforts and training in case management within the cholera framework. WHO programme for control of diarrheal disease is in the process of developing guidelines for the control of epidemics due to Shigella dysenteriae type 1, and an initial draft is already being reviewed. WHO has also targeted the development of new and improved shigella vaccines.

UNICEF and the Red Cross have participated in current epidemic control through procurement of drugs in Zambia. UNICEF has been also working on planning health education campaigns.

In Zambia, PRITECH has been also involved in training courses and development of educational campaigns. The PRITECH representative has followed up the issue of initiating a widespread hand washing campaign with the deputy Minister of Health, head of the Health Education Unit, and the UNICEF representative. Letters have been drafted to several soap manufacturers to enlist their support. UNICEF’s social mobilization officer also developed plans to promote health messages through the local media. In Zambia, a number of intervention studies are planned in cholera-prone areas supported by NORAD, the Italian government, and UNICEF, but will take time to yield results.
PART II - LESSONS FROM OTHER REGIONS

What has been done?

I. Intervention Studies

Interventions aiming at environmental sanitation and improvement of personal and community hygiene have shown to have an impact on shigella morbidity. A few examples follow.

A study in Bangladesh showed that giving soap to contacts of shigellosis patients resulted in reduction in secondary infection rates from 32.4 per cent among controls to 10.1 per cent among the targeted population. Total reduction in secondary infection was of 67 per cent. The secondary symptomatic rate was 2.2 per cent in the study group and 14.2 per cent in the control group. The overall reduction in secondary case rate was 84 per cent. Reduction of Sh. dysenteriae type 1 secondary infection rate was considerably lower (33%), and presumed to be due to its greater virulence and smaller dose requirement for infection. Diarrhea due to other causes were also reduced by 37 per cent. The same study found that providing additional water was effective only when soap was provided as well. It was suggested that provision of soap and water could result in an 80 per cent reduction in hospitalization for shigellosis.17

A hand-washing intervention study in Burma also demonstrated that hand washing is effective in reducing the morbidity from dysentery. The diarrheal incidence was reduced by as much as 40 per cent.42

A water, sanitation and hygiene education intervention project in rural Bangladesh showed that children in the intervention area had 25 per cent fewer episodes of diarrhea than those in the control area.43

Stewart et al showed that by making potable water more available the frequency of shigella infections halves.44 A study in Libya also demonstrated reduction of shigella incidence rates after the introduction of a water treatment center.45

Control of houseflies have also shown to have an impact on the prevalence of carriage of shigella organisms, diarrhea, and mortality due to diarrheal disease among young children.46

Breast-feeding confers a high level of protection against shigellosis throughout the first 3 years of life, especially among malnourished children. Therefore, breast-feeding promotion has been also recommend as an important component of shigella control efforts.47,48
Interventions aiming at environmental sanitation, food and water safety and health education for changes in personal hygienic habits are bound to have an impact on disease morbidity and mortality. However, such interventions need to be affordable, feasible, introduced in response to country-specific situations, accepted at the community level, and have guaranteed sustainability in order to achieve any real lasting results. In Libya for example, a failure to adequately maintain a water treatment unit reverted achievements in reduction of shigella infection rates initially obtained in the intervention area.
PART III - RECOMMENDATIONS

Interventions for dysentery due to Sh. dysenteriae

I. Assumptions

In developing a set of recommendations for dysentery control in Africa, some assumptions must first be made.

a) There are several potential agencies and donors involved in the dysentery program including the World Health Organization, UNICEF, Red Cross, European Community, etc., and most importantly, the national ministries of health. To maximize the impact of the interventions, the agencies involved must coordinate their efforts to avoid unnecessary duplication and/or competition. Coordination will require coordination meetings to understand the strengths, priorities, and opportunities of each agency.

b) Country-specific interventions will be carried out in the individual countries, each of whom have national interests, cultural and behavioral differences. While the general policies for dysentery, especially related to sanitation and case management, may be generalizable, decisions about specific policies need to be defined for each country. USAID (and other relevant agencies) should be available to assist the MOH’s in development of these policies.

c) The shigella control activities will be carried out within the context of control of "epidemic diarrheal diseases" which includes both cholera and epidemic dysentery and these activities

Assumptions Concerning
Dysentery Program in Africa

I. The USAID dysentery activities will be coordinated with other agencies and donor groups, e.g. WHO, UNICEF, Red Cross, etc.

II. National Ministries of Health will develop policies for dysentery control for their own countries.

III. The national dysentery control activities will be carried out within the context of the national CDD program, and will include activities related to cholera control as well as dysentery.

IV. The choice of USAID sponsored dysentery activities will be those for which USAID is best suited and has a comparative advantage.

V. Projects funded by USAID will coordinate their activities.
should complement and build up the national CDD programs. Generally a program to control epidemic diarrheal diseases will include a central coordinating role for the national CDD manager, but it may also include officials from other ministries.

d) USAID is not able to assume responsibility for the entire range of shigella control activities, and it should therefore choose those aspects of the control strategy which 1) best matches its strengths, 2) are not being covered by other donor agencies, and 3) is less susceptible to rapidly changing political situations.

e) USAID will want to use its various offices and projects it supports to accomplish the goal of dysentery control in Africa. AID supports several projects wholly (BASICS, WASH, ADDR, etc) and contributes substantially to others (ICDDR,B, WHO/CDR/, etc) and has other mechanisms in the Office of Health for funding needed work. Many of these funding sources and offices may need to coordinate their activities, if the goal is to be reached.

II. Activity Grids

A range of activities should be included in the program to control epidemic diarrheal diseases and these are illustrated on the two activity grids. The first illustrates the activities usually associated with "disease control" while the second corresponds to those activities usually associated with "research." The distinction between control and research is not always a clear one, and in fact it will be critical for the control to interact with the research to define the problems they face.

It must be emphasized that, in contrast to watery diarrheal diseases, there are no simple solutions to the dysentery problem, and no model systems to emulate. Therefore, the control activities are based on the knowledge of what "should work" given the knowledge available about the organism and the disease. However, the relative importance of the components, their effectiveness and their relative cost effectiveness is untested.

When considering the costs involved, in some cases, it may be more appropriate to invest a small amount into research to determine on a small scale the most successful approach, prior to embarking on a large scale project. This is true for case management delivered through CHW's, for IEC, for epidemiological surveillance methods, as well as for the laboratory. In each of these, the trend should be to make the program as possible.
## ACTIVITY GRID FOR CONTROL OF EPIDEMIC SHIGELLOSIS IN AFRICA

<table>
<thead>
<tr>
<th>CONTROL MEASURES</th>
<th>USAID support</th>
<th>WHO/CDR</th>
<th>World Bank</th>
<th>UNICEF</th>
<th>Red Cross</th>
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### Notes:

1. Operational research regarding water and sanitation behaviors and feasibility of personal sanitation interventions. This should be coordinated between BASICS and WASH.
2. Technical assistance to help establish and maintain an ongoing surveillance system.
3. Assistance with case control and special epidemiological projects.
4. Assistance in developing and validating and using rapid diagnostic tests for shigellosis and cholera.
5. Assistance with upgrading overall laboratory support.
6. Assistance with capital expenditures and infrastructure for laboratories.
7. The IEC messages need to be coordinated with the epidemiologic studies to insure that behavioral messages are appropriate and will decrease risk.
8. Interaction with private sector will stress certain products which are appropriate for dysentery control: soap, chlorine, water storage containers, private water distributors, food distribution.
III. Specific Recommendations

3.1. Joint Strategy for Epidemic Diarrhea

In evaluating interventions for dysentery due to *S. dysenteriae* 1 in Africa, an initial consideration includes the decision to favor interventions with a broad public health benefit as opposed to a targeted benefit for dysentery alone. The World Health Organization has initiated the concept of "epidemic diarrhea" as the target for an intervention strategy, especially for Africa. Within the "epidemic diarrheas" they include diseases due to both *V. cholerae* and *S. dysenteriae* 1. Though the respective diseases are quite unique, these two agents go logically together for several practical reasons. They both occur in epidemics, affect all age groups, are transmitted through fecal-oral routes, and both have a high case fatality rate if not treated appropriately. Furthermore, many of the preventive interventions for one will also benefit the other. From a logistical and administrative viewpoint, coordinating committees can be of great usefulness in both, and clinical and laboratory surveillance is needed to detect and monitor the course of the epidemics and the antibiotic sensitivity patterns of the pathogens. Finally, many of the countries affected by one epidemic are also affected by the other, and a joint effort would seem most beneficial.

Hence, for many practical reasons an intervention strategy for shigellosis should include cholera. While favoring a joint strategy, certain differences must also be realized as shown on the table 3. Disease symptoms and clinical management of two are quite distinct, and the benefit of case management is relatively less with shigellosis. For example, good case management for cholera lowers the case fatality rates from 40% to < 1% using very simple intravenous and oral rehydration techniques. With dysentery, case fatality rates lowers the rate from 10-20% down to 5% with appropriate antibiotics and supportive care, but this care is considerably more expensive, and patients may develop unusual and chronic complications.
which are not seen in cholera. Training and improvement in case management is thus important for both, but expectations should be less and inputs will be higher with dysentery.

3.2. Development of Country Plans in Cooperation with the MOH and Other Donors

In development of "the strategy" for epidemic diarrhea, country differences must be understood, and plans must be made by decision makers within the country. USAID should provide consultants who are able to provide technical assistance on development of country plans.

3.3. Combination of Activities

There is no simple intervention for either of the epidemic diarrheal diseases. Both require a multi-component program to decrease risk, to treat those with illness, and to minimize the complications of the illnesses. As a format with which to develop a multi-component national strategy, a checklist was developed for cholera and has now been updated for its potential use with dysentery and epidemic diarrhea, with special reference for Africa. The components generally include a) Planning /Management/ Administration, b) Case management, c) Training of health professionals, d) Epidemiology/surveillance, e) Water and sanitation, f) Personal/family hygiene, g) Laboratory services, h) Logistics and supply, and i) Information, education, communication. The checklist does not provide a set of instructions about how to control epidemic diarrhea, but it does provide a framework for dialogue between consultants and ministry officials for jointly developing a plan which is country specific and specific to the resources and constraints of the society. (See Appendix 1 for details).

3.4. Target One Country or Small Area

While many countries have been affected by the dysentery epidemic, it may not be possible for AID to work in all. Since there are no examples of successful national strategies for epidemic diarrhea in Africa, it may be wise to start in one (or perhaps two), to develop such a strategy and to proceed in a step by step basis, and thereby build experience. The emphasis country should have: a) Interest by MOH for carrying out a strategy for shigellosis control, b) Research center which can collaborate with the MOH or NGO in evaluating strategies, c) Suitable laboratory facility for diagnosis, and d) Interest in collaborating with scientists/public health officials.

3.5. Research and Evaluation

USAID should support a certain range of research activities which will be directly useful
in Africa within the next 1 to 5 years. As shown on the activity grid, many of these are involved with clinical aspects or the social sciences and some are more basic and biomedical. The biomedical areas suggested will directly relate to control activities and should either clarify or simplify the control activities. Some of these relate to fairly simple adaptations to solve practical problems in the field and some address more complex research issues for eventual control of the problem.

Some biomedical research projects with immediate relevance to controlling the epidemic include the following. a) Development and appropriate use of rapid diagnostic tests a) for appropriate categorization of a case as a case of watery diarrhea or dysentery, b) for rapid detection of cholera and S. dysenteriae 1 infections in surveillance systems, and c) a simpler method for antibiotic sensitivity of the strains detected. These should be cheaper, faster, more adaptable to field conditions, and should yield more reliable information than the current methods, especially for dysentery. Development and appropriate use of rapid and inexpensive assays would improve clinical practice and avoid unnecessary antibiotics. c) Determination of the interaction of dysentery and HIV infection, especially in relation to response to treatment and in the potential of AIDS patients being a reservoir for infection. d) Identification of patients who will benefit from more aggressive nutritional rehabilitation. e) Identification of risk of inappropriate antibiotics.

In addition to these applied research projects with immediate application, the development of a shigella vaccine for S. dysenteriae 1 should be given high priority. Bacterial genetics has progressed rapidly in recent years such that a bivalent cholera-Shiga vaccine is within reach within 3 to 4 years. Such a vaccine would contain protective antigens of both bacteria, but it could be safely given by mouth, without need for injection. If such a vaccine were developed, a single oral vaccine could be given which could protect against both diseases. While a vaccine would not solve the root socio-economic causes of diarrheal diseases, it would prevent many deaths, especially in Africa where the case fatality rates of these epidemic diseases is so high.

Development of such a vaccine would require research funding by AID. However, if a mechanism were found for funding such a project in a cooperative manner between for profit and not-for-profit groups, a successful outcome would be highly likely at moderate cost. Though scientifically feasible given current technology, this type of vaccine development has not received high priority be pharmaceutical companies because they do not see sufficient profits from developing a vaccine to be used exclusively in poor countries. Within other US funding agencies, such a project is likely seen as "too applied" since it involves no new technology. Thus it tends to be too practical for NIH and too basic for AID, and without sufficient profit for private companies unless the R&D were funded from donor sources. If USAID were to fund such a project, a) the likelihood of success is high, b) one could easily monitor the progress of the vaccine’s development and c) the level of funding could be adjusted to the needs of its development.
## Epidemic Dysentery in Africa

### RESEARCH NEEDS IMPORTANT FOR CONTROL OF EPIDEMIC SHigellosis IN AFRICA

<table>
<thead>
<tr>
<th>Research Area</th>
<th>BASICS</th>
<th>ICDDR,B</th>
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<th>CDC</th>
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<tr>
<td>Water storage containers</td>
<td>(c Wash)</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Family water purification</td>
<td>1 (c Wash)</td>
<td>2</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Village water purification</td>
<td>(c Wash)</td>
<td></td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Development &amp; evaluation of diagnostics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Simple diagnostic test</td>
<td>1</td>
<td></td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Simple test for antibiotic resistance</td>
<td>1</td>
<td></td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Evaluation of simple test for fecal wbc's</td>
<td>1</td>
<td></td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Evaluation of test for invasive ameba</td>
<td>1</td>
<td></td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Development and evaluation of IEC</strong></td>
<td></td>
<td>(c Healthcom)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Evaluation of private sector initiatives</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vaccine development</td>
<td>1 (with private sector)</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
3.6. **Social Science Research**

Among the social science research projects with immediate relevance are the following.

a) Identification of country specific risk factors for transmission of *S. dysenteriae*.

b) Identification of acceptable methods for water purification, food storage.

c) Development of market strategies for promotion of appropriate use of soap.

d) Identification of constraints to access of medical care.

3.7. **Sponsor a Conference on Control of Dysentery in Africa**

As USAID’s strategy becomes more clear cut, increased awareness of the problem is needed by the donors, scientists, public health officials, and ministries. A major meeting bringing these groups together can highlight the opportunities for successful control of epidemic diarrhea, and can also be used to strengthen the concept of carrying out the programs within the context of the CDD program.
# Epidemic Dysentery in Africa

## Table 3 - Comparison Between Cholera and Epidemic Dysentery.

<table>
<thead>
<tr>
<th>FEATURE</th>
<th>CHOLERA</th>
<th>EPIDEMIC DYSENTERY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Causative organism</td>
<td><em>V. cholerae</em> serotype 01 and 0139</td>
<td><em>S. dysenteriae</em> serotype 1 (Shiga's bacillus)</td>
</tr>
<tr>
<td>Related bacteria</td>
<td>Non O 1 <em>V. cholerae</em> and other vibrios which can cause cases of diarrhea, but not epidemics.</td>
<td>Other geni and species of <em>shigellae</em> which can cause bacillary dysentery (shigellosis) but not epidemics. Other species include <em>flexneri</em>, <em>sonnei</em>, <em>boydii</em>, and other serotypes of <em>S. dysenteriae</em>.</td>
</tr>
<tr>
<td>Infectious inoculum</td>
<td>About $10^{3-6}$</td>
<td>About $10^{2-3}$</td>
</tr>
<tr>
<td>Bacterial toxins</td>
<td>CT (cholera toxin) plays a key role in pathogenesis and is similar to a toxin in enterotoxigenic <em>E. coli</em>.</td>
<td>Shiga toxin plays an ancillary role in pathogenesis and is similar to a toxin(s) in hemorrhagic <em>E. coli</em> (SLT-1 and SLT-2).</td>
</tr>
<tr>
<td>Colonization factors</td>
<td>Pili facilitate colonization (tcp pilus and mannose sensitive hemagglutinin MSHA).</td>
<td>None described, but colonizing factor seems likely.</td>
</tr>
<tr>
<td>Standard diagnosis</td>
<td>Culture using special media and reagents.</td>
<td>Culture requires special care since organism is fragile. Transport medium is especially critical. Stool microscopy (for wbc's) can be helpful.</td>
</tr>
<tr>
<td>&quot;Appropriate&quot; diagnostic tests</td>
<td>Rapid tests are available. Genetic tests are available as research tools.</td>
<td>Experimental tests (pcr) have been developed and could be adapted. Simple test is available for fecal white cells. Other rapid diagnostic tests could be developed rapidly.</td>
</tr>
</tbody>
</table>
### Comparison Between Cholera and Epidemic Dysentery (cont.)

<table>
<thead>
<tr>
<th>FEATURE</th>
<th>CHOLERA</th>
<th>EPIDEMIC DYSENTERY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical disease</td>
<td>Severe watery diarrhea, frequently progressing to severe dehydration. Never becomes persistent.</td>
<td>Colitis, with blood and mucus in the stool, usually with fever, severe abdominal cramps. May become persistent.</td>
</tr>
<tr>
<td>Primary complications</td>
<td>1. Dehydration and electrolyte imbalance. 2. Several others can occur but are rare.</td>
<td>1. Sepsis. 2. Toxic megacolon. 3. Hemolytic uremic syndrome (HUS). 4. Leukemoid reaction. 4. Persistent dysentery. 5. Malnutrition.</td>
</tr>
<tr>
<td>Treatment</td>
<td>1. Rehydration saves lives. 2. Antibiotics shorten illness.</td>
<td>1. Antibiotics saves lives. 2. Rehydration also needed in some cases. 3. Nutritional rehab may be critical.</td>
</tr>
<tr>
<td>Purpose of antibiotics</td>
<td>Decrease duration of illness.</td>
<td>1. Decrease case fatality rate. 2. Shorten illness and prevent complications.</td>
</tr>
<tr>
<td>Antibiotic of choice for sensitive organisms</td>
<td>Tetracycline, Cotrimoxazole, Furoxone.</td>
<td>Cotrimoxazole, Ampicillin, Naladixic acid.</td>
</tr>
<tr>
<td>Complication if wrong antibiotic is used</td>
<td>Probably none.</td>
<td>Could increase severity of disease. Theoretically, the cell wall active antibiotics could increase HUS even in sensitive strains.</td>
</tr>
<tr>
<td>Antibiotic sensitivity patterns</td>
<td><em>V. cholerae</em> O1 is frequently resistant to many antibiotics. New O139 strain has been tetracycline sensitive.</td>
<td>Variable but most are resistant. Nalidixic acid is still useful in many, but not all areas of Africa. Nearly all strains are resistant to cotrimoxazole and ampicillin. Newer quinolones and mecillinam are effective but expensive.</td>
</tr>
</tbody>
</table>
## Comparison Between Cholera and Epidemic Dysentery (cont.)

<table>
<thead>
<tr>
<th>FEATURE</th>
<th>CHOLERA</th>
<th>EPIDEMIC DYSENTERY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age group</td>
<td>All ages. Newly infected areas usually have predominance of adults.</td>
<td>All ages. Newly infected areas usually have predominance of adults.</td>
</tr>
<tr>
<td>Endemic areas have more cases among children</td>
<td></td>
<td>Endemic areas have more children.</td>
</tr>
<tr>
<td>Sex</td>
<td>Both sexes are affected. Young adult women tend to have higher rates in endemic areas, but men tend to have higher rates in newly infected areas.</td>
<td>Both sexes are affected. Young adult women tend to have higher rates in endemic areas, but men tend to have higher rates in newly infected areas.</td>
</tr>
<tr>
<td>Vehicles of transmission</td>
<td>1. Municipal water. 2. Food contaminated by food or fingers. 3. Water contaminated in house.</td>
<td>1. Contaminated food. 2. Person to person. 3. Contaminated water</td>
</tr>
<tr>
<td>Reservoirs of infection</td>
<td>Water and shell fish including plankton.</td>
<td>None known.</td>
</tr>
<tr>
<td>Non-human mammalian infections</td>
<td>Only humans.</td>
<td>Primates can become infected, but these are not likely to be important for human disease.</td>
</tr>
<tr>
<td>Attack rates</td>
<td>1-20 per 1,000 population.</td>
<td>Up to 50 per 1,000 population.</td>
</tr>
<tr>
<td>Proportion of Symptomatic Infections</td>
<td>25 per cent (2 per cent severe, 5 per cent moderate, and 18 per cent mild).</td>
<td>About 40-50 per cent (84 per cent mild and 16 per cent severe).</td>
</tr>
<tr>
<td>Case-to-infection ratio</td>
<td>About 1:7.</td>
<td>About 1:1.5 to 1:2.5.</td>
</tr>
<tr>
<td>Secondary infec. rate</td>
<td>About 20 per cent.</td>
<td>About 20-30 per cent.</td>
</tr>
<tr>
<td>Secondary case rate</td>
<td>About 11 to 50 per cent.</td>
<td>About 7-13 per cent.</td>
</tr>
<tr>
<td>Case fatality rate in severe untreated cases</td>
<td>About 40 per cent.</td>
<td>About 10-20 per cent.</td>
</tr>
<tr>
<td>Case fatality rate in severe treated cases</td>
<td>Less than 1 per cent.</td>
<td>About 5 per cent.</td>
</tr>
</tbody>
</table>
Comparison Between Cholera and Epidemic Dysentery (cont.)

<table>
<thead>
<tr>
<th>FEATURE</th>
<th>CHOLERA</th>
<th>EPIDEMIC DYSENTERY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interventions with potential for greater impact</td>
<td>Those aiming at water and food safety. (However, it also greatly benefits from improvements in personal hygienic standards).</td>
<td>Those aiming at changes in personal hygienic habits. (However, it also greatly benefits from improvements on water and food safety).</td>
</tr>
<tr>
<td>Vaccines available?</td>
<td>Old injectable not recommended.</td>
<td>None.</td>
</tr>
<tr>
<td>Vaccine Development</td>
<td>New oral killed and <em>V. cholerae</em> O1 live vaccines developed and under evaluation. <em>V. cholerae</em> O139 oral vaccines under development.</td>
<td>Oral vaccine feasible if targeted funding were available.</td>
</tr>
</tbody>
</table>
APPENDIX 1

CHECKLIST FOR EPIDEMIC DIARRHEA

In response to the dysentery epidemic in Africa, PRITECH prepared this checklist to assist with control of epidemic diseases. This was felt to be needed to provide guidance when formulating and reviewing national and regional control plans. The following checklist, adapted from a similar checklist developed for control of cholera in Latin America, was prepared following review of literature sources, of relevant publications from the World Health Organization and from clinical experience. The checklist is divided into a short Administrative Checklist which will likely be more useful for administrators who are tracking activities and a more detailed Technical Checklist which should be useful for planners who are attempting to organize the technical activities.

These checklists may be useful to several groups, including:

- Diarrhea epidemic coordinating committees in the process of developing a national plan for whom the checklists are intended to illustrate the various components of the plan.
- Consultants who are reviewing national plans for whom the checklist will serve as a reminder of the types of components and indicators one might expect to see in the plan.
- Health administrators for whom the checklist will assist in defining the types of technical assistance which might be appropriate for a country dealing with epidemics of dysentery or cholera.
- Students of public health for whom the checklist will serve to illustrate the multifaceted nature of cholera and epidemic dysentery.

The checklists assume that a coordination committee is responsible for epidemic diarrhea control activities, and that it must secure cooperation from various ministries, donor agencies, non-governmental organizations, and industry. The primary role of the committee is one of policy development, coordination, and monitoring. To carry out the work, most committees have representation from several agencies, disciplines and groups including the national diarrheal disease control program (CDD) (which we anticipate will be the lead agency), physicians, nurses, water/sanitation, communications, epidemiology, logistics, laboratory services, hospital administration, training, high-risk minority group(s), and tourism. Most country plans will cover the following major areas:

Planning /Management / Administration
Training of health professionals
Water and sanitation
Laboratory services
Information, education, communication

Case management
Epidemiology/surveillance
Personal/family hygiene
Logistics and supply
Not all the areas are equally important, and clear prioritization of efforts is necessary. Some activities are immediately life-saving, while others will have a longer-term benefit to the nation. Each of the activities need to have clear lines of responsibility so that it can be carried out with a minimum of duplication.

The checklist is formatted as a series of questions which allows the reviewer to determine what the situation is, and whether plans have been formulated. A national committee is unlikely to have answers for all the items on the technical checklist, and there is not necessarily a correct answer to all items. When information is not available, the first decision will be to determine if the information is important; if not, nothing further need be done. However, the lack of information may bring attention to an area which has been overlooked. The annotations which accompany each checklist provide some guidance as to appropriate directions, but each national committee must develop its own strategy most appropriate for the country.

Few of the activities or interventions suggested by the checklist have been evaluated in a systematic "scientific" manner; however, from all available evidence, the components described here are thought to be effective in control of epidemic diarrhea. The final list covers a number of activities which are often included in cholera plans but are controversial, of limited or no benefit, or are even detrimental.
## Administrative Checklist

<table>
<thead>
<tr>
<th>Plan exists?</th>
<th>Agencies involved</th>
<th>Last evaluation?</th>
</tr>
</thead>
</table>

### I. Planning/Management/ Administration
- National plan
- Coordination of implementation
- Donor coordination
- Program evaluation

### II. Case management
- Treatment standards
- Indicators developed
- Accessibility of care

### III. Training of health professionals
- Linked to CDD
- Training courses for MD’s
- Training for health workers
- Continuing education

### IV. Epidemiology/surveillance
- Definitions established
- Reporting system
- Rapid response team

### V. Water and sanitation
- Personal/family hygiene
- Municipal Water
- Non-Municipal Water
- Long Term Issues in Water and Sanitation
- Solid waste
- Excreta collection
- Excreta disposal and waste water treatment

### VI. Laboratory services
- Appropriate use of lab?
- Technical capabilities

### VII. Logistics and supply
- ORS, IV and antibiotics
- Supplies for remote areas
- Inventory system?

### VIII. Information, education, communication
- Strategy developed?

### IX. Avoidance of unnecessary activities
- no vaccine at borders
- no or limited prophylactic antibiotics
- avoiding unnecessary isolation
I. Planning /Management/ Administration

This list emphasizes the need for a national coordinating body to formulate a national strategy for control of epidemic diarrhea and to include important elements into the policy. It also emphasizes the need to garner the available resources both within the country and from outside donor sources, and to prepare realistic plans within available budgets.

1. Has a national coordination committee for epidemic diarrhea (CCED) been established?

2. What is the frequency of the CCED meetings?

3. Have a policy for epidemic diarrhea and plan been formulated?

4. Are appropriate antibiotics available at the DTC? What antibiotics are available? What was the lab and epidemiologic basis for the selection of the antibiotic used?

5. Does the policy have a stated and realistic goal?

6. Do the goals have measurable outcomes?

7. Have communication channels been established with regions / districts / municipalities to report cases and permit a smooth flow of information?

8. Has communication been established between the national CCED and WHO CDR?

9. Has communication been established between the national CCED and possible donors and outside technical resources?

10. What is the relation of the CCED to the CDD program; will the CCED enhance the CDD program?

11. Have economic aspects of the control plan been considered? If the budget must be cut, what activities will be scaled back or eliminated?
II. Case Management

CASE MANAGEMENT I. Items which indicate the medical system’s ability to give life-saving care during an epidemic.

Treatment of epidemic diarrhea patients depends on the ability of trained health persons to administer rehydration rapidly, and on the capability of patients to reach and have access to proper care. For those living in remote areas, rapid response may be needed to bring care to the patients -- thus improving accessibility.

1. Have specific hospitals and diarrheal treatment centers (DTC) been clearly identified as being available for epidemic diarrhea treatment?

2. Are flow sheets illustrating management of cholera and shigellosis cases prepared and available to medical staff? Are they posted in the DTC?

3. Does the flow sheet clearly provide type and volume of fluids, antibiotic selection, and doses?

4. Are appropriate I.V. polyelectrolyte solutions available at the DTC? What are the names (and formulas) of the solutions and do they conform to acceptable standards? Are they available in sufficient quantities?

5. Are appropriate ORT solutions available at the hospitals / treatment centers? Are they available in sufficient quantities?

6. Does the ORT solution conform to WHO formula?

7. Are cholera cots available at each of the DTCs?

8. Among the physicians who are to treat cholera and dysentery patients, how many have been trained in clinical management?

9. Is there a schedule for training these physicians in case management of diarrhea so that > 90% will be trained within 6 months?

10. Repeat #1.8 and 1.9 for nurses.

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CASE MANAGEMENT II. Items which indicate that medical care is accessible.

Most diarrhea deaths occur among persons who do not have ready access to care. Though poor access is usually thought related to geographic distance from health facilities, it may also be related to social distance due to language, ethnic, or socioeconomic separation. Subgroups within urban or rural areas may be at high risk, but have poor access, and these groups need to be especially targeted for treatment. For epidemic diarrhea, treatment delayed is treatment denied.

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>What proportion of households are within 2 hours of a diarrhea treatment center / hospital? How does this differ between urban and rural areas?</td>
</tr>
<tr>
<td>2</td>
<td>Have certain groups been identified who have poor access because of geographical remoteness, language or cultural barriers, or economic barriers?</td>
</tr>
<tr>
<td>3</td>
<td>Have plans been formulated to provide medical care to these groups?</td>
</tr>
<tr>
<td>4</td>
<td>Has a rapid-response team been formed to serve remote areas affected by epidemics?</td>
</tr>
<tr>
<td>5</td>
<td>Has a supply kit been formulated for rapid-response teams?</td>
</tr>
<tr>
<td>6</td>
<td>Are the cholera/dysentery supply kits ready and available?</td>
</tr>
</tbody>
</table>
Epidemic Dysentery in Africa

III. Epidemiology / Surveillance

Cholera and epidemic dysentery tend to be multi-focal in their appearance, striking certain areas and certain populations. Early detection of epidemics can assist with appropriate allocation of resources. National surveillance and reporting of cases to the World Health Organization allows for the problem to be monitored on an international scale. Reporting of cases depends on case definitions, and generally a case needs to be confirmed bacteriologically only if the case is among the first in a new area. After cholera or *Sh. dysenteriae* 1 is known to be occurring in the area, a clinical case definition can be used which does not depend on the laboratory and a sample system can monitor the course of the epidemic.

Tracking cases can also assist in developing strategies for epidemic diarrhea interventions. For example, health education messages can be targeted toward warning the population against foods or water known to be at high risk and medical care can be directed toward assisting high risk groups.

In addition to monitoring cases, identifying and reporting complications and deaths can provide an index of the quality of care. If cholera is being well managed, rates of renal failure or death should be extremely rare (< 0.5%) while higher rates should signal the need for additional interventions or improved treatment strategies. Though not all dysentery is preventable, the case fatality rate should still be less than 3%. 
1. Is there a monitoring system for counting and mapping cholera and dysentery cases?

2. Is there a system for reporting the surveillance information to the medical community?

3. Is there a standard "case definition" for the following terms -- "cholera case", "cholera complication", "cholera death", "dysentery case", "dysentery complication", and "dysentery death"? Does a cholera case depend on laboratory confirmation?

4. Based on knowledge of cholera and dysentery epidemiology, have high-risk vehicles of transmission (e.g., certain foods or certain water types) been identified for the country?

5. Has a rapid-response team been formed to investigate and treat outbreaks of cholera and/or dysentery?

6. Is there a clear definition of what type of outbreak will stimulate an investigation by the rapid-response team?

7. Has an instrument been developed for data collection by the rapid-response team?

8. Has a case-control-study protocol been developed to identify high-risk activities or vehicles of transmission?

9. Is there a system for sampling a proportion of cholera and dysentery cases for bacteriologic confirmation?

10. Are travelers from cholera and dysentery endemic areas being advised (via a handout) to report to a treatment facility if they develop diarrhea?
IV. Training of Health Professionals

Quality of care provided by the DTC will depend on the knowledge and abilities of the medical staff as well as on the availability of the resources. Improving knowledge and skills of the medical staff will likely start with workshops, courses, and conferences, but must continue to be reinforced with continuing education and messages. An especially important component of the training is the hands-on treatment of cholera patients under supervision, followed by the availability of reference materials when treating patients independently (to clarify certain details which may not have been remembered accurately).

1. Have brochures stating national policies on epidemic diarrhea treatment been published?

2. Have WHO manuals of treatment of cholera and dysentery (or comparable manuals) been distributed to appropriate physicians and DTCs?

3. Have national / district / municipal workshops on epidemic diarrhea treatment been held for physicians?

4. Have national / district / municipal workshops on epidemic diarrhea been held for nurses?

5. Have the workshops stressed practical "hands-on" treatment of patients?

6. Have national medical journals included review articles on treatment of cholera and dysentery?

7. Have regular reports been provided to health personnel on the dysentery and cholera situation in country?

8. Has there been a program of medical conferences in teaching and municipal hospitals on epidemic diarrhea?

9. Has a national or regional cholera/dysentery training center been established?
V. Water and Sanitation

Improved water and sanitation can cut the numbers of cholera and dysentery cases dramatically by decreasing the rates of secondary spread. Secondary spread can be divided into inter-community spread, inter-family spread, and intra-family spread. The many vehicles which can transmit cholera and dysentery make it difficult to stop all spread; however, fairly simple measures can decrease rates. Water and sanitation is often thought to depend on huge capital investment (e.g., municipal water and sewage systems) and, in the long run, this is true. However, in the short run, many improvements can be made which will decrease risk and which depend on the family and individuals.

WATER AND SANITATION I. Personal and Family Hygiene

1. Are current behaviors known? Including:
   - personal hygiene practices, especially handwashing and bathing
   - water handling practices
   - defecation and excreta disposal practices, including the disposal of infant and children's stools
   - water source selection
   - household disinfection of water, if any
   - solid waste disposal practices

2. If information on current behaviors is not available, is there a plan to collect such information? Are technical resources available to implement the plan?

3. Is hand soap available? How widely? Is it affordable?

4. How do families without in house piped water supplies store water inside and outside of the home? Are there small scale improvements, such as spigots on tanks, or small neck containers, that may decrease contamination of stored water supplies?
WATER AND SANITATION II. Municipal Water

1. What proportion of households are served by municipal water? Of these, what proportion have water piped into the home and what proportion are served by public standpipes?

2. What proportion of municipal water supplies have facilities for chlorination?

3. Is chlorination equipment in working order?

4. Is chlorine available? What are the current stocks of chlorine country-wide?

5. What are the obstacles to increasing chlorine supplies? Including:
   - foreign exchange
   - water authority budget
   - tariffs
   - storage facilities

6. Is there a system for monitoring chlorine levels? At the treatment plant? In the distribution system? At the tap?

7. If collected, are records on chlorine levels available? How are monitoring data used to adjust chlorine levels?

8. What is the percent water loss in municipal systems?

9. Is there a plan for identifying leaks and repairing them?

10. Is there constant positive water pressure in municipal systems? If not, how often is pressure down or negative in municipal systems?

11. Are there provisions for water conservation?
   - public education
   - rationing system
WATER AND SANITATION III. Non-Municipal Water

1. What is the proportion of households with other water sources? Determine this by type, including:
   - protected wells, pumps
   - unprotected wells
   - surface water sources
   - tanker trucks
   - others, as appropriate

2. What is the proportion of households with their primary water source greater than 150 meters from the house?

3. What proportion of these water sources are chlorinated (including tanker trucks)?

4. Have standard messages been developed for the method of chlorination? Is there a dissemination plan for the messages?

5. Are materials available for chlorination of water sources? Are they affordable?

6. Can access to potable water be increased in the short run?
   - by improving the tanker truck distribution system?
   - digging new wells
   - protecting existing wells
   - tapping springs

   If so, does such a plan exist?
WATER AND SANITATION IV. Long Term Issues in Water and Sanitation

1. Is there a plan to extend coverage of potable water systems? Sanitation?

2. Does the institutional capacity exist to increase water and sanitation coverage if capital is available? What is the absorptive capacity in 5 years? 10 years?

3. Is there a plan to increase the institutional capacity in the water and sanitation sector?

4. Are water tariff systems effective? Are they enforced? Is there a plan to improve tariff systems?

5. Does legislation exist that regulates water quality, solid waste disposal, waste-water disposal and waste-water reuse? Are the standards appropriate? Are they enforced?

6. Are there opportunities/needs to introduce or develop new technologies? Including:
   - latrines that consume less water
   - alternative methods of water collection and distribution, e.g., rainwater harvesting
   - exploiting new water sources
   - low cost sanitation and sewage systems
   - alternative waste-water treatment technologies
WATER AND SANITATION V. Solid waste

1. What are the practices of the community? Is solid waste a major concern with regard to cholera and SDI transmission? e.g. Do they dispose of fecal matter e.g. disposable diapers, toilet paper) in solid waste?

2. Is there a public education campaign regarding solid waste?
   If so:
   - Is it appropriate and based on knowledge of the solid waste practices of the community?
   - Is it targeted to appropriate populations? e.g. children who scavenge in dumps, mothers who throw diapers in the trash?
   - Does it provide a realistic, practical alternative to current practices?

3. Are disposal sites appropriate, located at safe distance from population centers?

4. What are the alternatives to solid waste disposal? e.g. burning waste, burying waste on a community level.
WATER AND SANITATION VI. Excreta collection

1. What is the percentage of sanitation coverage:
   - What percentage of the population is served by on-site excreta disposal? e.g. latrines?
   - What percentage off-site? e.g. septic tanks, sewers
   - What populations are most at risk? e.g. urban slums

2. In areas where latrines are used:
   - Are they being used correctly
   - Are they placed to avoid contamination of water supply?
   - What are the behaviors of adults vs children vis a vis the latrine

3. In case of off-site sanitation, where does it go? Is it treated? (see section on disposal waste-water)

4. Are education and public awareness campaigns disseminating appropriate messages (culturally and technically correct) regarding latrine usage etc.?

5. Are there opportunities to use new technologies in sanitation collection?
WATER AND SANITATION VII. Excreta disposal and waste-water treatment

1. Are there guidelines for disposal of feces for people who are known to be infected? (e.g. hospitals) Are necessary materials available?

2. Is waste water treatment practiced in any municipalities?

3. What government body is responsible for waste-water treatment?

4. Is there a waste water re-use program?

5. Are there regulations for waste-water use on crops? Are they enforced?

6. Are short term priorities established relating to waste-water treatment?

7. What is the state of the sewer systems? Is there cross contamination of water supplies? Is there a system for detecting problems and repairing the sewer system?

WATER AND SANITATION VIII. Hospital Sanitation.

1. Are the sewage systems adequate at hospitals where dysentery / cholera patients will be treated?

2. Do these hospitals have a plan for disinfecting soiled linens?

3. Do these hospitals have a plan for disposal of dysentery / cholera contaminated solid wastes?
VI. Laboratory Services

Each country should have laboratory capability to confirm cholera and dysentery cases and to monitor the course of the epidemic. Accurate detection of *V. cholerae* and *S. dysenteriae* in fecal specimens is not difficult nor expensive for adequately equipped and staffed laboratories, but careful plans must be formulated to test only appropriate specimens and to use optimal media and methods. Testing of specimens from every patient with suspected cholera or dysentery is clearly not indicated, but confirmation of initial cases in an area, and confirming a sample of cases as the epidemic continues is wise. Periodic antibiotic sensitivity testing is also appropriate from a sample of *V. cholerae* and *S. dysenteriae* isolates. Countries with more highly specialized laboratories will want to carry out additional research investigations on the isolates, but this is not necessary for routine surveillance.

1. Does the nation have a plan for collecting fecal specimens from (a sample of) suspected cases?

2. Is Cary Blair medium being used as transport medium for suspected cholera cases and buffered glycerol saline for suspected dysentery cases and does the specimen get plated within twenty four hours of collection?

3. Does the plan include confirmation of a proportion of cases at a reference laboratory?

4. Does the laboratory use TCBS agar and alkaline peptone enrichment for suspected cholera cases and at least two standard enteric media for suspected dysentery cases?

5. Are antibiotic sensitivity tests being carried out on a sample of cholera and shigella isolates?

6. Have the central laboratories participated in a workshop on cholera and shigella identification within 2 years?

7. Does the laboratory report cholera and shigella results to the national authorities at least monthly?
VII. Logistics and Supply

Effective care of patients depends on adequate availability of supplies and equipment, and an optimal system in which to work. Treatment is therefore not only the job of the clinician, but involves a team of persons working within a system to ensure that the rehydration fluids, antibiotics and other supplies, as well as the expertise, are all provided to patients in need. To develop a logistics system which is not only effective, but also cost-effective, careful planning is needed to be sure that supplies are available, but that overstocking of supplies is avoided, and that supplies are purchased at reasonable prices. In the midst of epidemics, panic frequently results in unwise purchases and inefficient planning.

1. Has an estimate of the numbers of cases of cholera and dysentery expected been prepared with an estimate of the timing of the cases?

2. Has a plan been prepared for procuring supplies for the estimated number of cases? What was the method used to estimate the supply lists?

3. Is there a list of vendors, prices, and plans for procuring cholera and dysentery supplies?

4. Is there a plan for distributing the supplies to centers around the country?

5. What supplies will be procured locally and what supplies will be procured from international sources?

6. Are supplies, antibiotics, fluids, etc. being obtained from reputable suppliers at optimal prices? Is there evidence of "comparison shopping?"

7. How will logistics for epidemic diarrhea control be coordinated with logistics for other activities of the CDD or other programs?

8. Are the logistic requirements sustainable within CDD or Essential Drugs Programs?
VIII. Information, Education, Communication

Education of the public is an important component of a control strategy so that families can avoid high risk behavior and can seek medical care when appropriate. Common mistakes have been to a) raise the awareness of the dangers without sufficient information to let families know what cholera and dysentery are, how they can be prevented and where to seek treatment, b) delivering mixed or conflicting messages leaving the population confused, and c) providing excessive numbers of messages so that none has the required impact. Because of the panic which often accompanies an epidemic, information needs to be presented effectively, but in a manner which does not unnecessarily raise additional panic. Epidemic diarrhea often stimulates rumors, and educational messages are needed to correct misunderstandings about its transmission, its symptoms and treatment.

Communication messages which are directed towards policy makers can also be significant stimuli toward correcting long neglected problems of poor sanitation and contaminated water. An educated public is more likely to demand needed services and can help direct public policy.

1. What are the major agencies, both implementing agencies and donors, involved in IEC work on epidemic diarrhea? What are the resources available to them?

2. Has a communication coordinating committee been established (e.g., including representatives from the MOH, the media, PVOs, churches, food industry, tourist industry)?

3. Have necessary linkages been established between the communication component and other related components, e.g., policy and training, to ensure consistent and technically correct messages?

4. Has a formal communication plan been written? Have specific objectives been articulated in the context of the country’s overall policy (i.e., increase awareness of the threat; teach effective management of cases; teach essential prevention behaviors)?

5. Have target audience segments been clearly identified and prioritized (e.g., special risk groups)?
6. Have specific behaviors to be changed been identified for each target group?

7. Has any research been carried out among target groups to investigate knowledge, attitudes, and practices relevant to cholera and dysentery?

8. Has the information from 4-7 above been summarized in a creative brief to guide the development of communication materials?

9. What communication materials have already been produced -- brochures, radio and TV spots, documentaries, instructional videos, posters? How were they developed? Who are the target audiences? Were the materials pre-tested within the target audience?

10. What are the resources available for future communication work on epidemic diarrhea in the areas of consumer research? Communication strategy and planning? Media production?

11. Have the costs IEC messages been checked for consistency?

12. Has the timing of the IEC activities been considered in relation to the epidemic and to other important events (e.g. festivals or holidays)?

13. Has the cost of the IEC activities been determined?
IX. Avoidance of Unnecessary and Controversial Measures

In the past many measures have been taken which have been either counterproductive, ineffective, or distracting. For example, the injectable cholera vaccine is no longer recommended, yet a few border stations still require it for travelers crossing borders. Prophylactic antibiotics have, in the past, been used indiscriminately, leading to the emergence of antibiotic resistant strains. Nonspecific and ineffective antidiarrheal drugs have been used and unnecessary isolation of patients has been carried out. Each of these detracts from the central mission of caring for ill patients effectively and minimizing the rate of disease transmission.

1. Cholera vaccine is not being given?

2. Prophylactic antibiotics are not being given (except perhaps single dose doxycycline to immediate family members of cholera patients)?

3. Vaccination is not required for travelers at the airport or other border crossings?

4. Antidiarrheal drugs (e.g., Lomotil, steroids, etc.) are not being given?

5. Cholera attendants are not carrying out unnecessary precautions (e.g., routine gowns and gloves and masks)?

6. Avoid duplication and/or contradiction with CDD activities.
APPENDIX 2

For this report, shigellosis data were available for the following African countries: Algeria, Djibouti, Ethiopia, Egypt, The Gambia, Kenya, Libya, Malawi, Mozambique, Nigeria, Rwanda, Somalia, South Africa, Sudan, Uganda, Zaire, Zambia, Zimbabwe. Countries with recent (last 10 years) or current SDI epidemics will be discussed first. Amount and quality of information varied greatly. Information was obtained through the following sources: A.I.D. Development Information System, MEDLINE databases, donor reports and publications and personal communications.

I. Epidemic Countries

Burundi

SDI shigellosis has been endemic with seasonal variations for the last 10 years. The dysentery situation in Burundi has considerably worsened in 1991-92, when dysentery visits to health centers practically doubled compared to previous years (110,361 in 1991 and 75,532 in 1992). This makes dysentery the fifth most common cause of health center visits in Burundi. The 1993 epidemic has already affected more than 50,000 individuals.

In November 1990, clinical and epidemiological information was obtained from an age-matched case-control study of patients seeking care for bloody diarrhea. Overall isolation rate of enteric pathogens was 66 per cent. *Shigellae dysenteriae* type I and *E. histolytica* comprised 67 and 5 per cent of isolations, respectively. SDI isolates were resistant to chloramphenicol, tetracycline, sulfisoxazole, nalidixic acid, ampicillin, streptomycin, and trimethoprim-sulfamethoxazole. Comparative analysis among shigella species infections revealed that SDI patients were significantly more likely to have had contact with a person with dysentery and to have recently taken antibiotic. Shiga patients also were twice more likely to complain to abdominal pain and to have blood detected in the stool specimen. SDI patients did not differ significantly from non-dysenteric controls, except for recent contact with a person with dysentery and history of diarrhea in the previous months. Approximately 25 per cent of individuals in households were at least one dysentery case occurred within the last three months also reported dysentery.

Kibuye -

February 1992 - CDC Epidemiologic Services Branch evaluated the 1991 epidemic by conducting a community survey with a nested case-control study to look at risk factors for history of recent episode of dysentery (previous 6 months). Survey results indicated the incidence in the study community during 1991 was 21/1000 persons. 76% of all patients visited a health center, and 65% went twice. Based on this results, it was estimated that one in 60 persons had dysentery in Burundi during 1991 (national annual incidence rate for dysentery of 16.3/1000). In the studied population, the case-fatality ratio was of 3.2 per cent, dysentery
mortality rate was of 0.7/1000, the crude mortality rate was of 7.2/1000 and the proportional mortality rate was of 9.1 per cent (one out of 11 deaths were associated with dysentery). However, mortality data was not reliable because of strong cultural reluctance in reporting deaths in Burundi. It was estimated that the all mortality data were underestimated by a factor of three.

The mean age of persons reporting dysentery was 35 years, which was significantly higher than non-dysenteric individuals. The age distribution was bi-modal with incidence of approximately 15/1000 population at 1-4 years and reaching close to 20/1000 and 78/1000 at 30-59 and 60-92 years of age, respectively. There was a significantly higher incidence among females than males (16 vs 11 per 1000 persons). Significant household risk factors were - carrying water in an open bucket, not having soap in the house, and not washing hands before preparing food. Individual risk factors were - being of older age, being female, using a cloth rag for anal cleansing after defecation, weight loss before disease onset, and no education. No association was observed for activities such as going to the market, travelling, receiving visitors, or taking care of the a sick person. Household transmission was estimated to account for only 7 per cent of the cases.

Health education of those affected confounded the evaluation of behavioral risk factors, but also showed a positive effect of health education during the presence of the epidemic: households which had experienced dysentery were less likely to engage in certain risky behaviors. For example, hand-washing after defecation was more common among affected households than among non-dysenteric households. When the analysis controlled for the effect of health education, no difference in reported household hygiene habits was seen. This suggests a positive impact of the education received at the health facility when the first case was brought in for care. Even though the facility-based health education did not avoid the initial case, it may have been responsible for the low secondary transmission rate observed in this community. To avoid this confounding effect, retrospective study of risk factors and secondary spread need to be done at the time of first contact with the patient at the clinic or in a prospective follow-up of susceptible individuals.

Muramvya

February 1993 - Study was carried out to evaluate 1992 epidemic, which started in October 1992, reached a peak in November and declined at the beginning of 1993. Patients were selected from all dysentery registers from 9 peripheral health centers and interviewed at their homes within a mean of 100 days from disease onset. The study evaluated case fatality ratio, the proportion of all deaths due to dysentery, and risk factors for death from dysentery. Seventy per cent of the cases were over 10 years of age. There was no difference between males and females. 83 per cent of patients received co-trimoxazole. Case-fatality ratio among was very high (7.2% overall, 7.3% for those bellow 5 years, 5.8% for 5-49 year-olds, and 18.5% for those over 50 years). One out of 14 patients died, with most dying in the first 10-14 days. In 1991, it was estimated that 9 per cent of the deaths were due to dysentery. The majority of the deaths (61.5%) occurred at home, and within 10 days from disease onset (80%). The largest proportion of deaths occurred in the youngest age-group. The probability of survival by 100 days after the onset of dysentery is significantly lower for the oldest age group than for
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the other two age groups. Individuals who waited longer than one day before going to the clinic were less likely to survive than individuals who went to the clinic earlier. That may reflect the effect of delayed treatment or other underlying condition associated with increase risk of death, such as low socio-economic status. There was no association between completeness of treatment and survival. Using a case-fatality ratio of 7.2 per cent and an annual incidence rate of 21/1000/yr (from 1992 survey), the dysentery mortality rate for 1991 was recalculated to be 1.53/1000/yr. Based on 1988 census, the crude mortality rate was estimated to be 17/1000/yr in 1992. The proportional mortality rate due to dysentery is 9.0 per cent in 1992.

Incomplete therapy was a major problem and was related to have been given all 5 days therapy at the first visit. 77 per cent of patients bellow 5 years of age did no take a complete course of treatment. 27 per cent of patients reported waiting longer than 1 day before going to a health center.

Sixty per cent of specimens from new cases occurring between March and April of 1993 were shigella species. Of the total positive specimens, 76.7 per cent were SD1. In contrast to 1991, 1993 isolates were susceptible to nalidixic acid.

Zambia

The first evidence of epidemic bloody diarrhea in Zambia was noted in June 1990 in a person in a Kaoma prison in Western province, which led to over eleven thousand cases and 148 deaths in the district over the course of following year (June 1990-November 1991). Disease spread to other prisons in Mongu (July-August 1990) and Kamala (March 1991) through transfer of infected prisoners, and apparently from prisons to the community through a mechanism that was not elucidated. Following the prison outbreak, Kaoma district reported a large number of cases from July 1991 to early 1992.

From June 1990 to November 1991, there were 28,984 dysentery cases reported and 532 deaths nationwide (CFR of 1.8%). The western province was responsible for about 78 per cent of the cases reported. Case-fatality ratio ranged from 0.4 in the eastern province to 10.8 per cent in Lusaka. All age groups were affected. Reporting of dysentery is known to be incomplete and according to APO/CDR WHO not all deaths have been recorded, therefore estimates of attack, incidence and mortality rates are underestimated. The incidence seemed to peak in November-December 1991 and declined gradually, ceasing (returning to endemic levels in April 1992).

The number of adult dysentery cases admitted at the University Teaching Hospital increased steadily, representing 0.7 per cent and 59 per cent of adult admissions for diarrheal illness in November 1990 and 1991, respectively. The 20-29 year age group had the most patients (46%); the proportions in the different age groups was similar for men and women. Sixty three per cent of adult cases were men. During November 1991, at least 4 per cent of adult dysentery admissions died. The rate among men was higher than the rate among women. For 1991 overall (up to November 30), the annualized rate of death with dysentery as a cause was 1.97 per 100,000 persons per year in urban Lusaka. The overall rates were higher for men than from women (2.36 vs 1.59). The highest adult rates were for men 20-29 and 40-49 years
old (3.34 and 3.64, respectively) and for 50-59 years old women (4.78). The annualized rate per 100,000 population for hospitalization with dysentery at UTH was 2.8 times higher for adults than for children. (Freund et al., 1993) But the highest death rate was among children age 0 to 4 years (5.5 per 100,000 per year).

An interesting difference in epidemic behavior was noted in Siavonga Hospital in the Southern Province. In November 1990, 34 per cent of the dysentery cases were in children (0-4 years), while the same period in 1991 registered no cases in this age group. The proportion of males increased from 47 per cent to 69 per cent in this same time interval. 85 per cent (11 of 13) deaths occurred in adults. Reportedly, many of the deaths occurred in fishermen who lived on nearby islands.

Between July 1990 and November 1991, pediatric admissions for dysentery increased markedly in number and as a proportion of all admissions at the pediatric Diarrhea Training Unit, Lusaka (DTU). Between August and November 1991, dysentery cases increased from 11 per cent to 50 per cent of diarrheal admissions. Attendant case-fatality ratio increased reaching 16 per cent in November 1991. The majority of the pediatric patients were below three years of age, and mortality was higher in this age group. Compared to children with nonbloody diarrhea, children with dysentery were significantly older (23 vs 10 months) and less likely to die (15% vs 39%). The annualized attack rate for dysentery pediatric cases admitted to the DTU during October and November 1991 was 38 per 100,000 urban Lusaka children under 15 years old and 65 per 100,000 children under 5 years old. These rates are probably underestimated because children seen at health centers were not counted. The majority of cases (53%) were treated with co-trimoxazole. Other drugs used included: chloramphenicol, ampicillin, erythromycin, gentamicin, benzil penicillin, and metronidazole. In 1991, the highest annualized death rate was among children 0-4 years.

In November 1990, no deaths in Lusaka had dysentery as underlying cause. During the same period in 1991, 4 per cent of all death were from dysentery. Sixty-three per cent of the deaths were in males and 58 per cent in children under 5 years of age.

Twenty-two per cent of deaths with dysentery occurred outside the hospital; 45 per cent in children age 0 to 4 years. The proportion of deaths that occurred outside the hospital was higher for older patients (27% vs 17%).

In July 1991, the Western province had Red Cross assistance to obtain erythromycin which was distributed and used without any prior susceptibility tests, the same happening again towards the end of 1991, when UNICEF agreed to import an emergency supply of Gentamicin without prior understanding of resistance patterns of the epidemic strain. Gentamicin use started in February 1992.

Drug sensitivity patterns were not evaluated until more than a year into the Zambian epidemic. SDI isolates were sensitive to nalidixic acid, ciprofloxacin, amdinocillin, gentamicin, and ceftriaxome. Partially sensitive to chloramphenicol and resistant to ampicillin, septrin (co-trimoxazole, streptomycin, sulfa, and tetracycline.

In December 1991, CDC provided technical assistance to the UTH in training laboratory
staff in culturing techniques and susceptibility testing. Ability to isolate shigella improved dramatically. SD1 was isolated from 48 per cent of specimens cultured and comprised 96 per cent of shigella strains isolated. Based on CDC results, nalidixic acid became the treatment of choice. UNICEF made arrangements to provide nalidixic acid sufficient to treat 50,000 cases. Nalidixic acid use started in August 1992.

Between February and March 1992, CDC conducted a facility-based case-control study in urban Lusaka, which identified the following individual risk factors: eating food (meat and vegetable dish) purchased from the market or street vendor, having a family member with dysentery and having contact with a person with dysentery, living in a home where drinking water was obtained from hand-dipping a cup in container rather than by pouring it out. There were no differences in regards to frequency of market visits, number of meals eaten outside of the home, or number of meals form a common plate. Households risk factors included: having food preparer with dysentery, having open latrines, and sharing latrines among households. There were no significant differences among households regarding incidence of preceding diarrheal illness in the family, family size, number of children, number of visitors to the home, or visits to other homes, source of drinking water, frequency of boiling water before consumption, or chlorine levels of stored drinking water, which was consistently below the recommended levels of 0.5 ppm (median 0.2 ppm; range 0-0.5 ppm). Households were also comparable in regards to other aspects of hygiene such as availability of soap and toilet paper, wash water volume, distance to wash water source, or cleanliness of latrine.

Dysentery mortality decline after the introduction of appropriate therapy. During 1993 dysentery deaths has declined to approximately 30 to 40 per month (around 10% of all diarrheal deaths) and shigella isolation rates of 17-18 per cent among patients with bloody diarrhea.

Zaire

Northeast Zaire: 1979 - 1982

In Zaire, Shiga epidemic was reported in 1928-1932 with a case fatality rate as high as 50 per cent. Another dysenteric epidemic occurred concomitant to the 1943-45 famine, with forty-one per cent of shigella isolates being SD1, and with a case-fatality rate of 25 per cent. Form 1949 to 1978 shigellosis remained endemic in Zaire, but with a predominance of Sh. flexneri, which occasionally caused small institution-related outbreaks. SD1 was not reportedly isolated again for about 30 years.

Epidemic shigellosis caused by an apparently different strain of Shiga's bacillus reappeared in late 1979. It was not until more than a year later (December 1980) that the causative organism, Sh. dysenteriae type 1, could be first isolated and tested for antibiotic susceptibility. This was the first epidemic caused by a strain resistant to ampicillin. The exact incidence rate is unknown, but it has been estimated that over 100,000 persons suffered from SD1 dysentery and that five to ten thousand deaths occurred during this epidemic in Zaire.

Nyankunde - 1979 - 1981
A review of hospitalized cases in Nyankunde during the epidemic peak (June 1980 to February 1981) revealed that 88 per cent of the cases were adults and that the overall attendant mortality was 5.6 per cent. Case-fatality rate reached close to 50 per cent in some areas (Kisenyi). *Shigella dysenteriae* type I was isolated from 58 per cent of patients investigated between February and March 1981. Beginning in early 1981 the choice of treatment was switched to predominately trimethoprim-sulfamethoxazole given that all isolates were resistant to ampicillin, tetracycline, chloramphenicol, sulfathiazole, and streptomycin. No single source of the epidemic was identified, but, as expected, poor public health standards of the village was considered to have played a major role in the rapid spread of disease in this area. The outbreak in Nyankunde ended by April 1981.

**Katana** - April 1981 - August 1982

The outbreak in Katana started in April of 1981 and worsened considerably at the beginning of the rainy season in September, reaching its peak in November and December 1981. Overall attack rate were 6.4 per cent (10.9, 8.8 and 3.0 per cent for women, men and children, respectively). Overall case-fatality rate varied according to drug resistance patterns: 2.4 per cent with effective co-trimoxazole (April to July 1981), 4.6 per cent when resistance to co-trimoxazole developed (August to October 1981) and 2.0 per cent when nalidixic acid was introduced (November 1981 to March 1982). CFR dropped to 1.1 per cent during April-July 1982. Case-fatality rates were higher in adult males and females (2.8 and 2.5 per cent) and lower for children (1.9 per cent). Overall mortality rate was 0.16 per cent (around 0.25 per cent for adults and 0.05 per cent for children). In this outbreak, direct transmission within families and in the hospital was said to play a more important role than waterborne transmission. Isolates went form fully sensitive to co-trimoxazole in the beginning of 1981 to 75 per cent resistant in October 1981. In July 1982, after a period of replacement with nalidixic acid, 75 per cent of SD1 isolates were again sensitive to co-trimoxazole. In May 1982, the first strain resistant to nalidixic acid was isolated.

Between 1981 and 1985 more than 10,000 patients in the Kivu province were treated with NA. As of July 1985, the majority of *Shigella dysenteriae* isolates in Kivu were nalidixic acid resistant.

**Rwanda**

Shiga epidemic was documented in Rwanda during the 1943-1944 famine. The last documented isolation of this organism was in 1951, and it was not until 29 years later (1980) that it reappeared at the borders with Zaire, causing a rapidly spreading epidemic. Isolation rates for SDI went from none in the beginning of 1980 to 59.1 per cent of the shigella isolates in 1983.

**Cyangugu** - September 1981 - October 1982

In September 1981, the Central Africa Shiga epidemic reached Rwanda through Cyangugu, located at the souther border with Zaire. The examination of epidemiological reports of Cyangugu regional health administration from September 1978 to December 1983 revealed
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an increase in the mean number of cases per month from 320 during the three years prior to the epidemic (September 1978 and August 1981) to 833 during the epidemic (September 1981 to October 1982). A total of 11,801 cases were reported, but estimates were that at least 6,000 more individuals in Cyangugu (5 per cent of the population) were affected. The epidemic onset coincided with the beginning of the rainy season. Study of hospitalized cases showed that females prevailed in the age groups over 5 years, while the reverse was observed for children under 5 years of age (61% males). Seventy-two per cent of cases were over 18 years of age. Malnutrition was detected in 21 per cent of the children under 4 years (controls were not available). Attendant case-fatality rates were of 12, 9, and 4 per cent for children, adult male and adult female, respectively. Mortality was higher among the very young and the elder. Sulfaguanidine, co-trimoxazole, chloramphenicol and tetracyclines were used in 72, 64, 43 and 7 per cent of hospitalized patients, respectively. Use of metronidazol and dehydroemetin was also reported. The transmission of disease seemed to be mostly by direct contact (family outbreaks).

Gisenyi - February - May 1985

SDI epidemic reached the Gisenyi area in February 1985. The outbreak had a shorter duration than in other areas (4 months in comparison to 12 months in Cyangugu). By May 1985 SDI isolations had returned to endemic levels. During the peak of the outbreak, SDI was isolated from 93 per cent of hospitalized patients. All age groups were affected, with the highest number of cases among the 20 to 24 year old. Males accounted for 55 per cent of dysenteric patients. Attendant case-fatality rate was of 2 per cent.


Isolation of SDI from patients in Butare (southern Rwanda) between February 1985 and February 1987 showed an endemic profile with seasonal variation, with peak incidence from September to March (a period of moderate rain fall). Of the 471 shigella strains isolated during this period, 55.6 per cent were SDI.

Nalidixic acid has been systematically used in Rwanda since 1984, when resistance to trimethoprim-sulphamethoxazole forced its replacement. In Butare, by 1985, 98 per cent of SDI isolates were resistant to trimethoprim and 31 per cent had already developed NA resistance. All the NA resistant strains remained sensitive to Gentamicin, Nitrofurantoin and Norfloxacin.

Tanzania

In Tanzania, bacillary dysentery is endemic with occasional outbreaks. The endemic predominant specie is Sh. flexneri (64% of shigella isolates). SDI comprised only 1.9 per cent of isolates in 1979-1980.

In November 1981, the country experienced the first reported nationwide epidemic of bacillary dysentery, with SDI isolation rates of 54 per cent in 1982-1983. Study of hospitalized dysenteric patients revealed that 63 per cent of the cases were over the age of 10 and that the most affected group was the 20-29 year olds (53%). Males accounted for 61.4 per cent of all cases and prevailed in all age groups. Thirty-five per cent of patients had prior contact with
dysenteric case at home, and close to 11 per cent appeared to have become infected in institutions such as schools, prisons and work places. One per cent were nosocomial infections. About 80 per cent of patients reported water storage at home in covered buckets, practice of eating with their hands, rather than utensils, and hand-washing using communal water basin and no soap before eating. These findings were not compared against non-dysenteric controls, therefore the magnitude of the risk involved in these practices cannot be evaluated.

Low isolation rate (20%) of shigella species was believed to reflect improper techniques of specimen collection and laboratory processing. SD1 isolates were resistant to ampicillin, chloramphenicol, streptomycin, sulfonamide, and tetracycline, but were sensitive to neomycin, kanamycin, trimethoprim, nalidixic acid, polymyxin and nitrofurantoin.

**Ethiopia**

Shigellosis is endemic in Ethiopia, with clinical isolation rates ranging from 3.5 to 9 per cent. *Sh. flexneri* is the predominant endemic species, followed by *Sh. dysenteriae*.\(^\text{49,50,51,52}\) Between 1978 and 1982, seasonal outbreaks of Shiga bacillus were reported almost yearly in rural Ethiopia. During 1983, a massive outbreak of dysentery occurred in Gimira, south-west Ethiopia.\(^\text{11}\) Cultural and serological tests identified two strains of Shiga’s bacillus, both of which were resistant to tetracycline, chloramphenicol, ampicillin, carbenicillin, streptomycin and sulfadiazine. In addition, one strain was resistant to co-trimoxazole. This was the first time that a co-trimoxazole resistant Shiga’s bacillus was implicated in an epidemic in Ethiopia. During May 1983, a total of approximately 5,000 cases were reported, with an overall attack rate of 7.3 per cent (22.6 per cent and 30.6 per cent among 1-4 and 25-44 year olds, respectively). Mortality data was not available.\(^\text{21}\)

The plasmid responsible for SD1 trimethoprim resistant in the Ethiopian strains was different from the one encountered in strains in Central Africa, and it is possible that the epidemic in Ethiopia was not epidemiologically related to the Central Africa epidemic.

**Somalia**

The epidemic in Somalia in 1963-64 was caused by a SD1 strain resistant to streptomycin, sulphonamides and erythromycin and many non-epidemic strains isolated in 1976 were multiresistant.\(^\text{3}\) The plasmid profiles of isolates associated with epidemic Shiga in Zaire and Rwanda showed great similarity with strains isolated in Somalia in 1976.\(^\text{14,15}\) All these African strains showed significant similarities in their plasmid content and can be considered in relation to each other.\(^\text{15}\)

Since 1976, shigellosis seems to have returned to endemic levels. A hospital-based prevalence study of children with severe diarrhea during 1983-1984 in Mogadishu, Somalia, showed 61 per cent overall isolation rate, with 9 per cent of isolates being shigella species. Of the shigella species, the majority were *Sh. flexneri* (71%). Four per cent (3/145) were *Sh. dysenteriae* (50% type 1). This study also revealed a much lower infection rate with shigella species in predominantly breast-fed infants (bellow 8 months of age) than in mixed and non-
breast-fed infants. Shigella infections displayed a seasonal pattern with high rates in the rainy season. (Casalino M et al., 1988)

Malawi and Mozambique

In 1993, CDC Branch of Foodborne and Diarrheal Diseases investigated large dysentery epidemic in Mozambique and Malawi. SD1 etiology has been confirmed in both countries. Isolated strain seems to be the same as the one in other currently affected Central Africa countries and has the same patterns of multi-drug resistance. Naladixic acid has been recommended as the primary dysentery therapy in these two countries.

Uganda

A review of laboratory records between 1967 and 1982 showed that Sh. dysenteriae type I corresponded to only 0.5 per cent of shigella isolates, and was not isolated between 1977 and 1982. It seems likely that recent Shiga epidemic has also spread from neighboring Central Africa countries into Uganda, but this spread needs to be further confirmed.

II. Shigellosis in Non-Epidemic Countries

Algeria - Incidence rate (IR) of dysentery during the period 1981 to 1990 showed that the highest rate was in 1982 with approximately 65 cases per 100,000 population. IR decreased steadily over the next years, reaching less than 10 cases per 100,000 population in 1990. Breakdown according to etiology was not presented.

Djibuti - Shigella accounts for 7.7 per cent of the diarrhea isolates. In a study of clinical strains isolated between September of 1991 and April of 1992, Sh. dysenteriae corresponded to 10 per cent of shigella isolates, but serotypes were not reported. Shigella strains were resistant to ampicillin, tetracycline, trimethoprim-sulfamethoxazol and sensitive to cefotaxine, naladixic acid and ciprofloxacine.

Kenya - Prevalence study in diarrheal children revealed shigella was the enteropathogen most frequently isolated (27 per cent of isolates). Serotypes were not provided.

A study of admission prevalence and nosocomial infection with shigella and salmonella conduct in 1988, revealed that 2.5 per cent of all screened hospital admissions in the Public Teaching Hospital, Nairobi had positive culture for shigella. Shigella prevalence was similar for children and adults (2.5 % vs 3.6 %, respectively). Factors that were associated with positive admission swab were evidence of malnutrition, diarrhea at admission, temperature greater than 37.5C, concurrent diagnosis of Kwashiorkor or malaria, and a diet that contained solid food for children under 6 years of age. Twenty-seven per cent of the HIV positive individuals (free of shigella or salmonella on admission) who developed nosocomial diarrhea had Salmonella or shigella species isolated, compared to 15 per cent of HIV negative individuals who developed...
nosocomial diarrhea. Twenty-eight percent of adult nosocomial infections had a diagnosis of HIV infection. HIV status was significantly associated with positive cultures. Forty-one per cent of subjects with positive cultures were HIV infected, compared to 25 per cent of those with negative cultures.35,36

In one study in 1990-1991, shigella strains isolated from females with bacillary dysentery and AIDS were evaluated for antibiotic resistance. Sixty-one percent of isolates were Sh. flexneri, 19.5 per cent were Sh. sonnei, 12.2 per cent were Sh. boydii, and 7.3 per cent were Sh. dysenteriae type 2. Isolates were over 90 per cent resistant to tetracycline, erythromycin, trimethoprim/sulfonamide and streptomycin and partially resistant to ampicillin and chloramphenicol. Close to 100 per cent were susceptible to naladixic acid, gentamicin and ciprofloxacin. Authors speculated whether the antimicrobial multiresistance among shigella species isolated from Kenyan AIDS patients were due to increased drug use among patients as a result of the AIDS epidemic, or if it is a result of general use in treatment of infectious diseases.58

Libya - Evaluation of fecal specimens from diarrheal patients submitted to laboratory examination in Tripoli during 1975-1980 identified endemic non-SDI shigellosis with seasonal variations. Overall isolation of enteropathogenic bacteria was of 29.7 per cent. Of this, 917 (10.22%) were shigella species (or 3.0% of investigated diarrhea cases. Sh. flexneri was the most frequently isolated species (66.4%) and Sh. dysenteriae was the lowest (7%). Type 1 comprised only 5 per cent (3/64) of the Sh. dysenteriae serotypes isolated. Fifth-two per cent of shigellosis cases were under the age of 11 years. The seasonality of shigella isolates in Tripoli was characterized by a summer peak, autumn high continuation and the pronounced February trough.59

At the end of 1977 a program of water treatment in the Brak area of Libya was expected to help reduce the incidence of water-related diseases.45 The total incidence of the diseases studied dropped from 12 per cent in 1977, to 8.5 per cent in 1978 and 6.0 per cent in 1979. The overall decrease was mainly attributed to the drop of the commonest reported disease - bacillary dysentery. Incidence of shigellosis gradually dropped during the years immediately following the introduction of the treatment plant. The highest bacillary dysentery rate in 1977 occurred in August (1.6 per cent of total population). The highest monthly rate in 1978 and 1979 occurred in May and were reduced to 0.7% and 0.46%, respectively. Incidence of bacillary dysentery showed a seasonal variation, with a peak between April and September every year. A direct relationship between the mean noon-time temperature and the incidence of the disease was also observed, and explained by the fact that the shigella seems to be more likely to survive and multiply in water during the warmer months.60

Nigeria - Bacteriologic investigation of childhood gastroenteritis in Ife, Nigeria had an overall isolation rate of 15 per cent. No shigella species were isolated.61 In Zaria, Northern Nigeria, shigella constituted 24.3 per cent of enteric pathogens isolated from out-patient dysenteric cases during January 1980 and December 1984. The most common species isolated was Sh. flexneri (60%). Sh. dysenteriae was the third most commonly isolated species (14%), but only 3 out of 45 Sh. dysenteriae type (7%) were type 1. The maximum incidence was during the rainy season.62
In 1984, the spacial distribution of mortality from leading 36 notifiable diseases in Nigeria revealed that dysentery was the **fifth leading cause of death** in the country, behind only to Measles, Malaria, Pneumonia and Tetanus. In 1984 there were 202 deaths due to dysentery, which corresponded to approximately 5.2 per cent of all deaths.63

A case-control study of children with acute diarrhea attending health centers in Sokoto (north-west Nigeria) revealed that shigella was the most frequently isolated enteropathogen from both the diarrheal (12.7%) and control children (6.6%). *Sh. flexneri* was the predominant species (60%) followed by *Sh. dysenteriae* (30%). Serotypes were not reported. Females were more often infected than males. shigella was more frequently isolated form bottle-fed diarrhoeal infants and from children who depended on surface water for drinking. After 6 months of age, the difference between breast- and bottle-fed children was not significant anymore. Most shigella were obtained from children aged 0 to 5 months and 3 to 5 years. The peak of isolation rates for shigella was during the rainy season (March-September).64

Bacteriological survey in Ile-Ife during 1986, revealed 3.2 per cent prevalence of shigella in apparently healthy school children. The prevalence decreased as age increased, and was higher among girls (4.6% vs 1.6%). Only *Sh. sonnei* and *Sh. boydii* were identified.65

A three-year (1986 to 1988) study of diarrheal patients in Calabar, revealed 4.9 per cent positive cultures for shigella. *Shigella flexneri* was the commonest species isolated (55%) followed by *Sh. dysenteriae* (24.1%). Fifty per cent of *Sh. dysenteriae* isolates were type 1. Isolates showed high and sometimes rising resistance of ampicillin, chloramphenicol, streptomycin and co-trimoxazole and complete resistance to tetracyclines and sulphonamides. The seasonal distribution of shigella isolated indicated higher isolation rate (74.1%) during the dry season than in the rainy season (25.9%). Patients under 15 years accounted for 51.4 per cent of cases.27

A prevalence study of acute diarrheal episodes in out-patients in Calabar during 1988, showed an overall isolation rate of 12.3 per cent, and 3.3 per cent prevalence of shigella species (17.6% of isolates). No serotyping was presented. (Utsalo SJ, 1991).66

Observation on distribution of shigella species in Lagos during 1985 and 1988 revealed that 2.4 per cent of isolates were shigella species, and that *Sh. dysenteriae* accounted for 28 per cent of Shigella isolates, while *Sh. flexneri*, *Sh. sonnei* and *Sh. boydii* accounted for 32, 27 and 20 per cent, respectively. No serotyping was presented.67

**Senegal** - An etiological study of infantile diarrhea in Sine-saloum, Senegal during February 1991 and January 1993, reported that shigella species comprised 18 per cent of isolates, with the predominance of *Sh. flexneri*. Isolates were susceptible to sulfamethoxazole-trimethoprim.68

**South Africa** - Prevalence study in south-western Cape of Good Hope during 1968-1985 revealed that shigellosis is endemic in the area with predominance of cases during late summer and autumn months. The most frequently isolated species was *Sh. flexneri* (72%). The second commonest isolate was *Sh. sonnei*. *Sh. dysenteriae* has not been isolated since 1979.69
Sudan - During 1984, a morbidity study of pediatric children revealed that dysentery (amoebic and bacillary) was the fourth most frequent disease among outpatient children (5.6 percent). Pneumonia, Malaria and Marasmus made the top three.
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