

COMMUNICABLE DISEASE TOOLKIT

SIERRA LEONE

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PREFACE

The purpose of the *Communicable Disease Toolkit* is to provide health professionals in United Nations agencies, nongovernmental organizations, donor agencies and local authorities working in Sierra Leone with up-to-date guidelines and standards for controlling communicable diseases.

The *Communicable Disease Profile Sierra Leone* aims to provide up-to-date information on the major communicable disease threats faced by the population. The list of endemic and epidemic diseases has been selected on the basis of the burden of morbidity and mortality and includes acute lower respiratory tract infections, African trypanosomiasis, cholera, bacillary dysentery, HIV/AIDS, Lassa fever, malaria, measles, tuberculosis and yellow fever. Diseases for which there are global eradication or elimination goals are also included. The document outlines the burden of communicable diseases in Sierra Leone for which data are available, provides data on recent outbreaks in the country, and presents disease-specific guidelines on the prevention and control of these diseases.

The *Surveillance Forms* and *Case Definitions* have been developed to provide early warning of epidemics but will also monitor acute lower respiratory tract infections, tuberculosis, sexually transmitted infections and malnutrition.

The *Guidelines for Outbreak Control, Case Management of Epidemic-Prone Diseases, Collection of Specimens for Laboratory Testing, and Outbreak Investigation Kit* are aimed at facilitating outbreak preparedness and response.

The control of communicable diseases represents a major challenge to those providing health care services in Sierra Leone and neighbouring countries. It is hoped that the *Communicable Disease Toolkit for Sierra Leone* will facilitate the coordination of communicable disease control activities between all agencies working in this region.

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Edited by Dr Michelle Gayer, Dr Máire Connolly, Dr Pamela Mbabazi, and Dr Albis Gabrielli of the Programme on Communicable Diseases in Complex Emergencies, WHO/CDS.

The *Communicable Disease Toolkit for Sierra Leone* is a collaboration between the Communicable Disease Working Group on Emergencies (CD-WGE) at WHO/HQ, the Division of Communicable Disease Prevention and Control (DCD) at WHO/AFRO and the Office of the WHO Representative for Sierra Leone. The CD-WGE provides technical and operational support on communicable disease issues to WHO Regional and Country Offices, MoHs, other UN agencies, NGOs and international organizations. This Working Group includes the Departments of Control, Prevention and Eradication (CPE), Surveillance and Response (CSR) in Communicable diseases (CDS), Roll Back Malaria (RBM), Stop TB (STB) and HIV/AIDS (HIV) in HTM; and the Departments of Child and Adolescent Health and Development (CAH), Immunizations, Vaccines and Biologicals (IVB) and Health and Action in Crisis (HAC).

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COMMUNICABLE DISEASE TOOLKIT

SIERRA LEONE

COMMUNICABLE DISEASE PROFILE



World Health Organization
2004

*Communicable Disease Working Group on Emergencies, WHO/HQ
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Introduction

The purpose of this document is to provide public health professionals working in Sierra Leone with up-to-date information on the major communicable disease threats faced by the population. The list of endemic and epidemic diseases has been selected on the basis of the burden of morbidity and mortality. Diseases for which there are global eradication or elimination goals are also included. The document outlines the burden of communicable diseases in Sierra Leone for which data are available, provides data on recent outbreaks in the country, and presents disease-specific guidelines on the prevention and control of these diseases.

The control of communicable represents a major challenge to those providing health care services in Sierra Leone. It is hoped that this document will facilitate the coordination of communicable disease control activities between all agencies working in the country.

1. ACUTE LOWER RESPIRATORY INFECTIONS (ALRI)

CHILDREN UNDER FIVE YEARS OF AGE

DESCRIPTION

Infectious agent	Bacteria: the most common are likely to be <i>Streptococcus pneumoniae</i> and <i>Haemophilus influenzae</i> (and <i>Staphylococcus aureus</i> to a less extent). Several respiratory viruses
Case definition	Clinical description ALRI are bronchitis, bronchiolitis, and pneumonia. Pneumonia is the most severe and it is fatal in 10–20% of cases if inappropriately treated Pneumonia Cough or difficult breathing and Breathing 50 or more times per minute for infants aged 2 months to 1 year. Breathing 40 or more times per minute for children aged 1–5 years; and No chest indrawing, stridor or general danger signs. Severe pneumonia Cough or difficult breathing and any general danger sign or Chest indrawing or stridor in a calm child. In infants under 2 months of age the presence of any of the following indicates severe pneumonia: cough or difficult breathing and breathing 60 or more times per minute or grunting or nasal flaring or fever or low body temperature or any general danger sign. General danger signs For children aged 2 months to 5 years: unable to drink or breast feed; vomiting; convulsions; lethargic or unconscious.
Mode of transmission	Airborne, droplets.
Incubation	Depends on the infective agent. Usually 2–5 days.
Period of communicability	Depends on the infective agent. Usually during the symptomatic phase.

EPIDEMIOLOGY

Burden	According to the Multiple indicator cluster survey (MICS-2) Survey Report (2000), 9% of children under 5 years of age had an acute respiratory infection in the 2 weeks before the survey (data from April–May).
Geographical distribution	Throughout the country. MICS-2 found the highest prevalence of ALRI in the north (11%). Other areas of the country had a prevalence of 4–9%.
Seasonality	The ALRI season peaks in July and August.
Alert threshold	An increase in the number of cases above the expected level for a specified period.
Recent epidemics	No data available.

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Influx of non-immune population/infected individuals into areas of new pathogens.
Overcrowding	Yes	Overcrowding increases the risk of developing ALRI.
Poor access to health services	Yes	Prompt identification and treatment of the cases are the most important control measures.
Food shortages	No	However, malnutrition can play a major role in susceptibility to infection and development of disease.
Lack of safe water and poor sanitation	No	
Others	Yes	Indoor air pollution. Low temperatures may increase the relative risk of children acquiring pneumonia.
Risk assessment conclusions		<p>In the first 4 months of 2000, the Ministry of Health and Sanitation surveillance system reported acute respiratory infections as the second leading cause of morbidity in the country after malaria, accounting for 31% of the total disease burden.</p> <p>A household level health survey conducted in 1993 in rural locations in western Sierra Leone revealed that the most frequently mentioned disease symptoms at the time of death of an infant or a child were, in order, fever, cough, and troubled breathing.</p> <p>The MICS-2 Survey Report showed that children in the northern regions of Sierra Leone endured the highest prevalence of ARI in the country (11% in the north versus 4–9% in other regions) and were the least likely to be treated by an appropriate health provider (40% in the north versus 53–68% in other regions).</p> <p>MICS-2 showed that 17% of children with ALRI were taken to a hospital for treatment and 22% were taken to a health centre. Overall, 50% of children with ALRI were taken to an appropriate health provider (i.e. hospital, health centre, dispensary, village health worker, MCH or mobile/outreach clinic, or private physician).</p> <p>MICS-2 showed that recognition of the symptoms for ALRI was generally difficult for non-health workers.</p>

PREVENTION AND CONTROL MEASURES

<p>Case management</p>	<p>The priority is early recognition and adequate treatment of cases</p> <p>First-line antibiotic for ARI in under-fives classified as pneumonia is co-trimoxazole; second-line antibiotic is amoxicillin.</p> <p>Pre-referral antibiotics for severe cases that cannot tolerate oral antibiotics or treatment for severe cases that cannot be referred are:</p> <ul style="list-style-type: none"> – for children 2 months up to 5 years old, intramuscular chloramphenicol, – for infants under 2 months of age, intramuscular benzylpenicillin <i>and</i> gentamicin. <p>Children with fever in addition to cough or difficult breathing may be also treated for malaria, according to their exposure to malaria risk (high vs low malaria risk areas) and laboratory results (blood film) if these services are available.</p> <p>Supportive measures, such as continued feeding to avoid malnutrition, vitamin A if indicated, antipyretics to reduce high fever and protection from cold (especially keeping young infants warm) are part of integrated case management. Prevention of low blood glucose may be necessary for severe cases.</p> <p>Integrated management of illness should be practised in any sick child seen by a provider trained in IMCI.</p> <p>Proper advice should be given to those caring for non-severe cases at home, including compliance with antibiotic treatment instructions.</p> <p>Signs of malnutrition should be assessed. Malnutrition increases the risk of death from pneumonia. Severely malnourished children (weight-for-height index <70%) should be referred to hospital.</p>
<p>Prevention</p>	<p>Health education on early danger signs for prompt care-seeking.</p> <p>Adequate feeding, including exclusive breastfeeding, to avoid malnutrition.</p> <p>Improved immunization coverage.</p>
<p>Immunization</p>	<p>Measles, diphtheria and pertussis (whooping cough) immunization is effective in reducing the impact of ALRI. Immunization coverage rates for these antigens are currently suboptimal in Sierra Leone.</p>

2. AFRICAN TRYPANOSOMIASIS (African sleeping sickness)

DESCRIPTION

Infectious agent	Protozoa: <i>Trypanosoma brucei gambiense</i> . Only the gambiense form of the disease is present in Sierra Leone.
Case definition	<p>Clinical description</p> <p>1st stage (haemolymphatic involvement):</p> <ul style="list-style-type: none"> – possibly painful chancre (papular or nodular) at the primary site of Tsetse fly bite (rare in <i>T. b. gambiense</i> infection) – possibly fever, intense headache, insomnia, painless cervical lymphadenopathy, anaemia, local oedema and rash. <p>2nd stage (neurological involvement):</p> <ul style="list-style-type: none"> – parasites cross the blood–brain barrier and attack the central nervous system – cachexia, somnolence and signs of central nervous system involvement. <p>The disease may last for several months or even years. The natural progression of the disease (when not treated) leads to body wasting, somnolence, coma and death. The disease is always fatal without treatment.</p> <p>Laboratory criteria</p> <p>Serological:</p> <ul style="list-style-type: none"> – Card Agglutination Trypanosomiasis Test (CATT): for <i>T. b. gambiense</i> only. A negative CATT results indicates that there is no disease; a positive result must be confirmed by microscopy. – Immunofluorescent assay: for <i>T. b. rhodesiense</i> mainly and possibly for <i>T. b. gambiense</i>. <p>Parasitological: Detection (microscopy) of trypanosomes in blood, lymph node aspirates or cerebrospinal fluid (CSF).</p> <p>Case classification</p> <p>Suspected: Any case without direct demonstration of the parasite that is compatible with the clinical description and/or with a positive serology.</p> <p>Confirmed: A case with direct demonstration of the parasite, whether or not compatible with the clinical description.</p> <p>1st stage: parasite seen in blood and/or lymph nodes, with CSF containing no detectable trypanosomes and a leukocyte count $\leq 5/\mu\text{l}$.</p> <p>2nd stage: CSF containing trypanosomes and/or a leukocyte count $>5/\mu\text{l}$.</p> <p>NOTE: <i>in the 1st stage or early in the 2nd stage of the disease there are often no clinical signs or symptoms classically associated with the disease. Suspicion is then based on local risk of contracting the disease and local disease history.</i></p>
Mode of transmission	The disease is transmitted primarily by the bites from infected tsetse flies (<i>Glossina</i> spp.). Transmission is also possible through contamination with infected blood or through the placenta (congenital).
Incubation	In <i>T.b. gambiense</i> infection there is a long incubation period of several months or even years.
Period of communicability	The disease is communicable to the tsetse fly as long as the parasite is present in the blood of the infected person or animal (from 5–21 days after infective bite). Parasitaemia occurs in waves of varying intensity in untreated cases during all stages of the disease. Once infected, the tsetse fly remains infective for life (1–6 months).

EPIDEMIOLOGY

Burden	<p>The available information on African trypanosomiasis in Sierra Leone revealed that very few cases were diagnosed until 1964. The disease was thought to affect mainly animals, and a report written in 1964 mentions that human cases were sporadic and that there was no proof of any active focus.</p> <p>Nevertheless, between February and May 1973, 7 patients, all in the neurological stage, were admitted to the Bonthe Hospital (Sherbro Island). At the same time a patient was seen in the adjacent Moyamba District Hospital.</p> <p>This disease is not a priority of the Ministry of Health and Sanitation and not under routine surveillance.</p>
Geographical distribution	No data available, but foci in Guinea are close to the Sierra Leonean border.
Seasonality	The disease has no clearly evident seasonal pattern.
Recent epidemics in the country	Not epidemic prone in Sierra Leone.

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	<p>Risk of settlement in a high-transmission area.</p> <p>Good organization and efficient health structures are essential to diagnose and treat the disease.</p>
Overcrowding	No	Tsetse density is not related to the density of the human population.
Poor access to health services	Yes	The complex nature of the disease requires efficient health structures and trained personnel for diagnosis and treatment.
Food shortages	No	
Lack of safe water and poor sanitation	No	The tsetse fly is not attracted by dirty water.
Others	Yes	<p>Sleeping sickness is a neglected disease with 100% mortality when not treated; 90% of the population at risk have little or no access to proper diagnosis and treatment.</p> <p>Medications most commonly used for the second stage of the disease has to be administered in hospital settings under conditions that are not always available in the most affected areas.</p> <p>There is little research on alternative medications that are less toxic or that could be more accessible.</p> <p>Studies indicated that a small number of infected tsetse flies can maintain endemic transmission cycles.</p>
Risk assessment conclusions		<p>Because of political instability, there has been no investigation of potential foci of African trypanosomiasis foci in recent years. Nevertheless, the close contact between humans, animals and the vector, as well as the proximity of the Guinean and Liberian foci, make it likely that such foci exist in the country. Moreover, movements of populations to and from neighbouring countries may increase the risk of re-emergence of the disease.</p>

PREVENTION AND CONTROL MEASURES

<p>Case management</p>	<p>Early screening and diagnosis are essential, as treatment is easier in the 1st stage of the disease: the patient does not present with psychiatric symptoms, fewer injections are required, and treatment poses less risk to the patient and can be given on an outpatient basis.</p> <p>Diagnosis and treatment require trained personnel and self-treatment is not possible. All confirmed cases must be treated as soon as possible. Most available drugs are old, difficult to administer in poor conditions and not always successful.</p> <p><u>T. b. gambiense infection</u></p> <p>1st stage: Pentamidine. 4 mg/kg per day IM for 7 consecutive days on an outpatient basis.</p> <p>2nd stage: Melarsoprol. Hospitalization with three series of injections administered with a rest period of 8–10 days between each series. A series consists of one daily IV injection of 3.6 mg/kg melarsoprol for 3 consecutive days.</p> <p>In case of melarsoprol treatment failure, eflornithine, 400 mg/kg per day, is given four daily slow IV infusions (lasting approximately 2 hours). Infusions are given every 6 hours, which represents a dose of 100 mg/kg per infusion.</p> <p>Note: Melarsoprol causes reactive <i>encephalopathy</i> in 5–10% of patients, with fatal outcome in about half the cases. The treatment has a 10–30% treatment failure rate, probably due to pharmacological resistance. Increasing rates of resistance to melarsoprol (as high as 25%) have been reported from various countries.</p> <p>A Human African Trypanosomiasis Treatment and Drug Resistance Network has been established by WHO. Four working groups are dealing with: (a) drug availability and accessibility; (b) coordination of drug development and clinical trials; (c) research on resistance and treatment schedules; (d) surveillance of resistance.</p> <p><u>Drug procurement</u></p> <p>Since 2001, a public–private partnership signed by WHO has made all drugs widely available. The drugs are donated to WHO. Requests for supplies are made to WHO by governments of disease-endemic countries and organizations working in association with these governments. Stock control and delivery of the drugs are undertaken by <i>Médecins Sans Frontières</i> in accordance with WHO guidelines. All the drugs are provided free of charge: recipient countries pay only transport costs and customs charges.</p>
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<p>Prevention</p>	<p><u>Routine preventive measures</u></p> <p>Public education on the following measures should be encouraged:</p> <ul style="list-style-type: none"> – avoidance of known foci of sleeping sickness and/or tsetse infestation – wearing suitable clothing (including long sleeves and long trousers) in endemic areas – prohibition of blood donation from those who live (or have stayed) in endemic areas. <p><u>Detection</u></p> <ul style="list-style-type: none"> – Containment of the human reservoirs through periodical population screening and chemotherapy of cases remains the cornerstone of disease control for gambiense sleeping sickness. – Active periodic screening (active case-finding) of the population of endemic foci by mobile screening teams is the best option, since infected subjects can remain asymptomatic and contagious for months or years before developing overt symptoms. – Screening usually comprises CATT-testing of the entire population visited by teams. <p><u>Vector control</u></p> <p>Through tsetse fly control programmes:</p> <ul style="list-style-type: none"> – application of residual insecticides or aerosol insecticides – use of insecticide-impregnated traps and screens (expensive) – destruction of Tsetse habitats by selective clearing of the vegetation: clearing bushes and tall grasses around villages is useful when peri-domestic transmission occurs, but indiscriminate destruction of vegetation is NOT recommended.
<p>Epidemic control</p>	<p>Trypanosomiasis is not epidemic-prone in Sierra Leone. Control measures normally recommended involve:</p> <ul style="list-style-type: none"> – mass surveys to identify affected areas. – early identification of infection in the community, followed by treatment. – urgent implementation of tsetse fly control measures (e.g. aerosol insecticides sprayed by helicopter and fixed-wing aircraft).

3. BACILLARY DYSENTERY (shigellosis)

DESCRIPTION

Infectious agent	Bacterium: Genus <i>Shigella</i> , of which <i>Shigella dysenteriae</i> type 1 causes the most severe disease and is the only strain responsible for epidemics
Case definition	Case classification Suspected: Diarrhea with visible blood in the stools Confirmed: A case corresponding to the clinical case definition with isolation of <i>Shigella</i> from stools
Mode of transmission	Faecal–oral route, particularly through contaminated water and food
Incubation	Incubation period is usually 1–3 days but may be up to a week for <i>S. dysenteriae</i> type 1
Period of communicability	During acute infection and until 4 weeks after illness (without treatment). With appropriate treatment, 2–3 days. Asymptomatic carriers exist.

EPIDEMIOLOGY

Burden	A total of 30 592 cases were reported from 9 districts in 4 regions of the country from June 1999–February 2000.
Geographical distribution	Countrywide
Seasonality	Cases occur all year round, and seasonal incidence patterns are not consistent between years.
Alert threshold	In the absence of a clear epidemic threshold, an epidemic should be suspected if: <ul style="list-style-type: none"> – there is an unusual and sudden rise of new cases or deaths due to bloody diarrhoea in weekly reports; – there is an increase in the proportion of bloody diarrhoea within diarrhoeal cases; – there are five or more linked cases of bloody diarrhoea. Any of these scenarios should lead to investigation of the disease agent by laboratory testing.
Recent epidemics	1999–2000: An outbreak due to <i>Shigella dysenteriae</i> type 1 (Sd1) was confirmed in November 1999 in western Sierra Leone. <i>Shigella flexneri</i> was also isolated in stool samples. Both strains were sensitive to nalidixic acid and ciprofloxacin. Further cases of Sd1 were later confirmed in Moyamba District (in the south) and Koinadugu district (in the north). Between 6 December 1999 and 16 January 2000, a total of 3094 cases of shigellosis with 132 deaths (CFR = 4.27) were reported by the Ministry of Health and Sanitation. Sporadic outbreaks continue to occur in different parts of the country particularly in areas with large concentration of returnees and other populations undergoing resettlement.

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Spreads the infectious agent.
Overcrowding	Yes	Very important

Poor access to health services	Yes	<p>Early detection and containment of cases are paramount in reducing transmission.</p> <p>In the absence of proper treatment, the case fatality rate of <i>S. dysenteriae</i> type 1 can be as high as 10% in children under 10 years old.</p>
Food shortages	No	<p>However, malnutrition increases gastrointestinal tract susceptibility to invasiveness of the organism and severity of disease.</p>
Lack of safe water and poor sanitation	Yes	<p>The most important risk factor.</p>
Others	Yes	<p>Contaminated food, lack of soap, and poor hygiene are also very important risk factors.</p>
Risk assessment conclusions		<p>Risk of epidemics of <i>S. dysenteriae</i> type 1 is high in the refugee camps (up to one-third of the population at risk may be affected).</p> <p>In the general population the risk is strictly related to the availability of safe water.</p>

PREVENTION AND CONTROL MEASURES

Case management	<p>Early and appropriate therapy is very important: treatment with an effective antimicrobial can reduce the severity and duration of shigellosis. Selection of the appropriate antibiotic antimicrobial depends on resistance patterns of the bacteria and drug availability.</p> <p>The problem of rapid acquisition of antibiotic resistance is a cause for concern in the treatment of <i>Shigella</i> dysentery in Africa. It is therefore important to confirm the sensitivity of <i>S. dysenteriae</i> to nalidixic acid in the early stages of an outbreak of shigellosis, in order to avoid the use of ciprofloxacin and decrease the risk of an early development of resistance to this antibiotic.</p> <p>Supportive treatment using ORS, continued feeding (frequent, small meals) and antipyretics to reduce high fever is also essential.</p> <p><i>S. dysenteriae</i> type 1 is often more severe or fatal in young children, the elderly, and the malnourished, and prompt treatment with antibiotics is essential. If antibiotics are in short supply, they should be reserved for such high-risk groups.</p>
Epidemic control	<p>Inform the health authorities if one or more suspected cases are identified.</p> <p>Early detection and notification of epidemic dysentery, especially among adults, allows for timely mobilization of resources needed for appropriate case management and control.</p> <p>Confirm the outbreak, following WHO guidelines. See <i>Guidelines for outbreak control</i> in this Toolkit.</p> <p>Rectal swabs from suspected cases should be collected and shipped, refrigerated (2–8°C), in an appropriate medium (e.g. Cary-Blair medium) to laboratories for culture to confirm the diagnosis of Sd1. (The viability of bacteria in Cary-Blair medium when refrigerated is generally 1–3 days but this is variable.) Fresh stool samples can be sent if Cary-Blair medium is unavailable but the sample must reach the laboratory and be processed within 6 hours.</p>

	<p>It is recommended that at least 10 cases be used to confirm the cause, identify antibiotic sensitivity and verify the outbreak. Once confirmation has been obtained, it is not necessary to obtain laboratory confirmation for every patient.</p> <p>Testing of Sd1 isolates for antimicrobial sensitivity (which may change within an outbreak) should be done at regular intervals to determine whether treatment guidelines remain appropriate. International referral laboratories are available to assist in identification of the organism and confirmation of the antimicrobial resistance pattern.</p> <p>Do not wait for laboratory results before starting treatment/control activities.</p> <p>See:</p> <ul style="list-style-type: none"> – <i>Guidelines for the control of epidemics due to Shigella dysenteriae type 1.</i> Geneva, World Health Organization, 1995 (WHO/CDR/95.4, available at: http://www.who.int/emc-documents/cholera/whocdr954c.html)
Prevention	<p>See:</p> <ul style="list-style-type: none"> – “Prevention” in “Section 5, “Diarrhoeal diseases (others)”, and Appendix 3, “Safe water and sanitation”

4. CHOLERA

DESCRIPTION

Infectious agent	Bacterium: <i>Vibrio cholerae</i>
Case definition	<p>A cholera outbreak should be suspected if:</p> <p>A person older than 5 years develops severe dehydration or dies from acute watery diarrhoea (clinical case definition)</p> <p>or</p> <p>There is a sudden increase in the daily number of patients with acute watery diarrhoea, especially patients who pass the “rice water” stools typical of cholera.</p> <p>Confirmed case: Isolation of <i>Vibrio cholerae</i> O1 or O139 from stools in any patient with diarrhoea.</p>
Mode of transmission	<p>Faecal–oral route:</p> <ol style="list-style-type: none"> 1. Person to person <ul style="list-style-type: none"> – when taking care of cholera patients. – through direct contact with bodies of deceased cholera patients (e.g. washing the body for funeral ceremonies). 2. Drinking contaminated water 3. Eating food (fruits and vegetables) contaminated through <ul style="list-style-type: none"> – water – soil – contamination <i>during</i> preparation (rice, millet, food from street vendors) – contaminated seafood. 4. Indirect contamination (hands)
Incubation	Incubation period is usually a few hours to 5 days.
Period of communicability	During the symptomatic phase until 2–3 days after recovery. Very rarely for months. Asymptomatic carriers are common.

EPIDEMIOLOGY

Burden	<p>Number of cases and deaths notified to WHO:</p> <p>2001: 0 cases 2000: 0 cases 1999: 834 cases, 5 deaths 1998: 2096 cases, 57 deaths 1997: 0 cases 1996: 0 cases 1995: 10 285 cases, 447 deaths</p>
Geographical distribution	The entire country is at risk. The source of most epidemics has been through Yeliboya, an overpopulated fishing island with very poor sanitation, in Kambia district. The risk of outbreaks appears to be higher in the south in Moyamba District.
Seasonality	Peak between March and June.
Alert threshold	Any suspected case must be investigated.

Recent epidemics in the country	<p>1999: Between 1 and 6 September, 134 cases and 1 death were reported by the Ministry of Health and Sanitation (location not specified). Of 8 stool specimens sent for laboratory tests, 6 were positive for <i>Vibrio cholerae</i> O1 subtype Ogawa, with antibiotic sensitivity to tetracycline, doxycycline and co-trimoxazole.</p> <p>1998: As at 16 September, 1770 cases and 55 deaths were reported by the Ministry of Health and Sanitation (start date and location not specified). <i>Vibrio cholerae</i> O1 El Tor was found to be responsible for the outbreak. Some cases of <i>V. cholerae</i> non-O1 were also identified, but tests for isolation of <i>V. cholerae</i> O139 were negative.</p>
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RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Spread of the infectious agent to others and to different sites.
Overcrowding	Yes	Very important. Close living increases risk of contact with vomitus, excreta and contaminated water or food.
Poor access to health services	Yes	Early detection and containment of the cases (isolation facilities) are paramount in reducing transmission.
Food shortages	No	
Lack of safe water and poor sanitation	Yes	The most important risk factor.
Others	No	Poor hygiene and lack of soap. Funerals and cultural practices that involve contact with the dead body (cholera case) could become important sources for spread of the disease.
Risk assessment conclusions		Given the existence of overcrowding, ongoing population movements (returnees, resettlement), poor access to health services in many areas, and lack of adequate safe water and sanitation, there is a high risk of cholera epidemics in Sierra Leone.

PREVENTION AND CONTROL MEASURES

Case management	<p>The mainstay of the case management of cholera is treatment of dehydration using ORS or IV fluids (Ringer's lactate).</p> <p>Use of antibiotics (doxycycline/tetracycline) is not essential for disease treatment but, in severe cases, may be used to reduce the volume of diarrhoea (and of the rehydration solutions required), its duration and the period of vibrio excretion.</p> <p>The antimicrobial sensitivity pattern should be assessed in order to select the appropriate antibiotic.</p> <p>The case fatality rate (CFR) can be extremely high (5–40%) in the absence of proper treatment. With appropriate case management, the rate may be <1%.</p>
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<p>Epidemic control</p>	<p>Inform the health authorities immediately if one or more suspected cases are identified</p> <p>Set up ORS corners to increase the coverage of the population.</p> <p>Confirm the outbreak, following WHO guidelines. Stool samples must be taken with a rectal swab and transported in Cary-Blair medium. If a transport medium is not available, a cotton-tipped rectal swab can be soaked in the liquid stool, placed in a sterile plastic bag, tightly sealed, and sent to the laboratory.</p> <p>It is recommended that at least 10 cases be used to confirm the cause, identify antibiotic sensitivity and verify the outbreak. Once confirmation has been obtained, laboratory confirmation for every patient is unnecessary.</p> <p>Do not wait for laboratory results before starting treatment/control activities</p> <ul style="list-style-type: none"> – Ensure prompt treatment and confirm the diagnosis. – Isolate cases in cholera treatment centres. – Provide adequate health education to patients, families and communities. – Ensure access to safe water and proper sanitation. <p>See:</p> <ul style="list-style-type: none"> – Leaflet, <i>First steps for managing an outbreak of acute diarrhoea</i>, Geneva, WHO, 2003 (WHO/CDS/CSR/NCS/2003.7). – www.who.int/csr/diseases/cholera
<p>Prevention</p>	<p>See:</p> <ul style="list-style-type: none"> – “Prevention” in Section 5, “Diarrhoeal diseases (others)”, and Appendix 3, “Safe water and sanitation”. – <i>Guidelines for cholera control</i>, Geneva, WHO, 1993. – www.who.int/csr/diseases/cholera
<p>Immunization</p>	<p>Cholera vaccines can complement, but cannot replace, conventional control measures. Their use as an additional public health tool is under consideration.</p> <p>Two oral vaccines are currently available – killed cholera vaccine (WC/rBS, two doses) and live attenuated vaccine (CVD103-HgR, single dose) – and have been licensed in some countries. Both could be used in chronic emergency situations, such as refugee camps or slum residents, under carefully evaluated conditions.</p> <p>See:</p> <ul style="list-style-type: none"> – Potential use of cholera vaccines in emergency situations. Geneva, WHO, 1999 (WHO/CDS/EDC/99.4) – WHO report, Oral Cholera Vaccine (OCVs) meeting of experts, Geneva, December 2002. State of the art new vaccines research and development (http://www.who.int/vaccine_research/documents/en/stateofart_excler.pdf) – <i>Cholera vaccines: a new public health tool? Report, WHO meeting, 10–11 December 2002</i>, Geneva, Switzerland. Geneva, WHO, 2004 (WHO/CDS/CPE/ZFK/2004.5). <p>For more specific information on cholera vaccines and their use, contact the Global Task Force on Cholera Control at WHO-Geneva: cholera@who.int.</p>

5. DIARRHOEAL DISEASES (others)

DESCRIPTION

Infectious agent	Bacteria: such as <i>Salmonellae</i> (commonly <i>S. enteritidis</i> , <i>S. typhimurium</i>) and <i>Escherichia coli</i> . The most severe outbreaks are caused by <i>Shigella dysenteriae</i> type 1 and <i>Vibrio cholerae</i> (see “Bacillary dysentery” and “Cholera”) Protozoa: such as <i>Entamoeba histolytica</i> , <i>Giardia lamblia</i> and <i>Cryptosporidium parvum</i> Viruses: such as Rotavirus and Norwalk virus
Case definition	Clinical case definition for acute diarrhoea Three or more abnormally loose or fluid stools over 24 hours.
Mode of transmission	Faecal–oral route, particularly through contaminated water and food.
Incubation	The incubation period for <i>Salmonella</i> is generally 8–48 hours, whereas that for <i>E. coli</i> is typically longer at 2–8 days (median 3–4 days). The duration of the disease in both cases is usually 2–5 days. The average incubation period for <i>E. histolytica</i> is 2–4 weeks, for <i>G. lamblia</i> 7–10 days and for <i>C. parvum</i> 7 days. The incubation period for <i>Rotavirus</i> is about 48 hours, and symptoms may last for up to 1 week.
Period of communicability	During the acute stage of the disease and for the duration of faecal excretion. People can continue to be temporary <i>Salmonella</i> carriers for several months.

EPIDEMIOLOGY

Burden	The MICS-2 Survey Report in 2000 showed that 25% of children under 5 years had diarrhoea in the 2 weeks preceding the survey.
Geographical distribution	The prevalence of diarrhoea was lower in the south (15%) and west (22%) of the country than in the east and north (approximately 28% in each region).
Seasonality	The “diarrhoea season” in Sierra Leone is at the beginning of the rainy season in May and June, at which time prevalence rates in excess of 30% can be expected.
Alert threshold	An increase in the number of cases above the expected level compared with the same period in previous years.
Recent epidemics	No data available.

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Can import cases.
Overcrowding	Yes	Very important.
Poor access to health services	Yes	Early detection and management of the cases are paramount in reducing transmission.
Food shortages	No	However, malnutrition increases both gastrointestinal tract susceptibility to invasiveness of the organism and severity of disease.

Lack of safe water and poor sanitation	Yes	<p>The most important risk factor. Prevention of diarrhoeal diseases depends on the provision and use of safe water, adequate sanitation and health education. The supply of adequate quantities of water should be one of the highest priorities in an emergency situation: the minimum daily requirement is 20 litres/person.</p> <p>Common sources of infection in emergency situations are:</p> <ul style="list-style-type: none"> – contaminated water sources (e.g. faecally contaminated surface water entering an incompletely sealed well) or water contaminated during storage (e.g. by contact with hands soiled by faeces) – shared water containers and cooking pots.
Others	Yes	<p>Poor hygiene; lack of soap; contaminated food items.</p>
Risk assessment conclusions		<p>Dehydration caused by diarrhoea is a major cause of mortality among children in Sierra Leone. Among different age groups, MICS-2 showed that diarrhoea prevalence peaked among children being weaned and/or being introduced to solid foods (i.e. children aged 6–23 months).</p> <p>A household-level health survey conducted in 1993 in rural locations in western Sierra Leone revealed that the most frequently mentioned disease symptoms at the time of a death of an infant or a child were, in order, fever, cough, troubled breathing and diarrhoea.</p> <p>In Sierra Leone's hot and humid climate, the high prevalence of diarrhoea coupled with inadequate fluid intake put the children in Sierra Leone at substantial risk of potentially fatal dehydration due to diarrhoea. MICS-2 revealed that only 28% of caregivers knew how to correctly manage diarrhoea at home.</p> <p>In camp situations, diarrhoeal diseases can account for 25–40% of deaths in the acute phase of an emergency. More than 80% of deaths usually occur among children under 2 years old.</p>

PREVENTION AND CONTROL MEASURES

Case management	<p>Reduction of mortality due to diarrhoeal disease is primarily related to effective management of dehydration, particularly in children.</p> <p>Prevention</p> <p>Give recommended home fluid (RHF) and oral rehydration salts (ORS).</p> <p>Treatment of dehydration</p> <p>ORS for mild to moderate dehydration, or IV fluids (Ringer's lactate) for severe dehydration is the mainstay of the management of diarrhoeal illness.</p> <p>Continue breastfeeding in infants and young children.</p> <p>Resume feeding with a normal diet when vomiting has stopped. It is important to separate those who are eating from those who are not. Food should be cooked on site.</p> <p>Use of antibiotics depends on the infectious agent.</p> <p>See:</p> <ul style="list-style-type: none"> – <i>The management and prevention of diarrhoea: practical guidelines</i>. Geneva, WHO, 1993. – <i>The treatment of diarrhoea: a manual for physicians and other senior health workers</i>. Geneva, WHO, 1995 (WHO/CDR/95.3).
Epidemic control	<p>Inform the health authorities immediately if an increase in the number of cases above the expected level is identified.</p> <p>Confirm the diagnosis and ensure prompt treatment.</p> <p>Confirm the outbreak following WHO guidelines</p>

<p>Prevention</p>	<p>The prevention of diarrhoeal diseases depends on the provision and use of safe water, adequate sanitation and health education.</p> <p><u>Safe drinking-water</u> Provision of an adequate and safe supply, collection and storage system. Provision of information on the importance of clean water, also covering system maintenance and household storage (see Appendix 3, "Safe water and sanitation").</p> <p><u>Safe disposal of human excreta</u> Provision of adequate facilities for the disposal of human waste. Provision of information on the importance of human waste disposal, also covering use and maintenance of the facilities.</p> <p><u>Food safety</u> Provision of adequate storage facilities for food (both uncooked and cooked), cooking utensils, adequate quantities of water and fuel to allow for cooking and re-heating of food. Health education on the importance of food safety and safe food handling</p> <p><u>Hand-washing with soap</u> Provision of soap in sufficient quantities for hand washing, bathing and laundry needs. Health education on the relationship between disease spread and lack of or poor hand-washing before eating, after toileting, before food preparation and after cleaning/changing children.</p> <p><u>Breastfeeding</u> Provision of information on the protective qualities of breastfeeding, and the importance of breastfeeding ill children. Practical support for breastfeeding ill children.</p>
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6. DIPHTHERIA

DESCRIPTION

Infectious agent	Bacterium: <i>Corynebacterium diphtheriae</i>
Case definition	<p><u>Clinical description</u></p> <p>Upper respiratory tract illness with laryngitis or pharyngitis or tonsillitis plus Adherent membranes of tonsils or nasopharynx.</p> <p><u>Laboratory confirmation</u></p> <p>Isolation of <i>C. diphtheriae</i> from a clinical specimen (throat swab), or a fourfold or greater rise in serum antibody (but only if serum samples are obtained before the administration of diphtheria toxoid or antitoxin).</p> <p><u>Case classification</u></p> <p>Suspected case: not applicable Probable case: a case that meets the clinical description. Confirmed case: probable case confirmed by laboratory or epidemiologically linked to a laboratory-confirmed case. Carrier: presence of <i>C. diphtheriae</i> in nasopharynx, no symptoms. Note: persons with positive <i>C. diphtheriae</i> identification but who do not meet the clinical description (i.e. asymptomatic carriers) must not be reported as probable or confirmed cases.</p>
Mode of transmission	<p>Contact (usually direct, rarely indirect) with the respiratory droplets of a case or carrier.</p> <p>In rare cases, the disease may be transmitted through foodstuffs (raw milk has served as a vehicle).</p>
Incubation	Usually 2–5 days, occasionally longer.
Period of communicability	Until virulent bacilli have disappeared from discharges and lesions; usually 2 weeks or less and seldom more than 4 weeks. The rare chronic carrier can shed bacilli for 6 months or more. The disease is usually not contagious 48 hours after antibiotics are instituted.

EPIDEMIOLOGY

Burden	<p>Number of cases reported:</p> <table> <tr> <td>2001: NA</td> <td>1997: 0 cases</td> </tr> <tr> <td>2000: 0 cases</td> <td>1990: NA</td> </tr> <tr> <td>1999: NA</td> <td>1980: 0 cases</td> </tr> <tr> <td>1998: NA</td> <td></td> </tr> </table>	2001: NA	1997: 0 cases	2000: 0 cases	1990: NA	1999: NA	1980: 0 cases	1998: NA	
2001: NA	1997: 0 cases								
2000: 0 cases	1990: NA								
1999: NA	1980: 0 cases								
1998: NA									
Geographical distribution	Throughout the country.								
Seasonality	No data available.								
Alert threshold	One suspected, probable or confirmed case must be investigated.								
Recent epidemics	No data available.								

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Importation
Overcrowding	Yes	Crowded conditions facilitate transmission.
Poor access to health services	Yes	No access to routine immunization services. Early detection and containment of cases are paramount in reducing transmission.
Food shortages	No	
Lack of safe water and poor sanitation	No	
Others	No	
Risk assessment conclusions		<p>Outbreaks can occur when social or natural conditions lead to overcrowding of susceptible groups, especially infants and children. This frequently occurs when there are large-scale movements of non-immunized populations.</p> <p><u>DTP3 coverage in Sierra Leone</u></p> <p>2001: 44% (National Coverage Survey 2001) 2000: 24% (official country estimates); 44% (WHO–UNICEF survey database) 1999: Data not available 1998: 56% (official country estimates) 1997: 26% (official country estimates) 1990: 83% (official country estimates) 1980: 13% (official country estimates)</p>

PREVENTION AND CONTROL MEASURES

Introduction	<p>The control of diphtheria is based on 3 measures:</p> <ul style="list-style-type: none"> – ensuring high population immunity through vaccination (primary prevention) – rapid investigation and treatment of contacts (secondary prevention of spread) – early diagnosis and proper case management (tertiary prevention of complications and deaths).
Immunization	<p>Immunize the population at risk as soon as possible. In an epidemic involving adults, immunize groups that are most affected and at highest risk. Repeat immunization procedures 1 month later to provide at least 2 doses to recipients.</p> <p>Diphtheria-toxoid-containing vaccine (preferably a vaccine with reduced diphtheria content (Td) should be given.</p> <p>To ensure injection safety during immunization, auto-destructible syringes and safety boxes are recommended. Safe disposal of used sharps should be ensured</p>

<p>Case management</p>	<p>Diphtheria antitoxin and antibiotic therapy are the cornerstone of therapy for diphtheria. The antibodies neutralize toxin only <i>before</i> its entry into cells. It is therefore critical that diphtheria antitoxin be administered as soon as a presumptive diagnosis has been made.</p> <p>Antibiotic therapy, by killing the organism, has three benefits:</p> <ul style="list-style-type: none"> – termination of toxin production – improvement of local infection – prevention of spread of the organism to uninfected persons. <p><i>Do not wait for laboratory results before starting treatment/control activities</i></p> <p><u>Patients</u></p> <p>Diphtheria antitoxin IM (20 000 to 100 000 units) in a single dose, immediately after throat swabs have been taken</p> <p>plus</p> <p>Procaine penicillin IM (25 000 to 50 000 units/kg per day for children; 1.2 million units/day for adults in 2 divided doses), or parenteral erythromycin (40–50 mg/kg per day with a maximum of 2 g/day) until the patient can swallow</p> <p>then</p> <p>Oral phenoxymethylpenicillin (125–250 mg) in 4 doses a day, or oral erythromycin (40–50 mg/kg per day with a maximum of 2 g/day) in 4 divided doses.</p> <p><i>Antibiotic treatment should be continued for a total period of 14 days.</i></p> <p>Isolation: strict for pharyngeal diphtheria or contact isolation only for cutaneous diphtheria for 14 days.</p> <p>Note: Clinical diphtheria does not necessarily confer natural immunity, and patients should therefore be vaccinated before discharge from a health facility.</p> <p><u>Close contacts</u>¹</p> <p>Surveillance for 7 days for all close contacts, regardless of vaccination status, and throat culture.</p> <p>All must receive a single dose of benzathine benzylpenicillin G IM (600 000 units for children under 6 years; 1.2 million units for those aged 6 or older) Erythromycin can be used also as second choice. If culture is positive, give antibiotics as for patients above.</p> <p><u>Carriers</u></p> <p>All must receive a single dose of benzathine benzylpenicillin IM (600 000 units for children under 6 years; 1.2 million units for those aged 6 and older).</p> <p>Note: Clinical diphtheria does not necessarily confer natural immunity, and patients should therefore be vaccinated before discharge from a health facility.</p>
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¹ Close contacts include household members and other persons with a history of direct contact with a case, as well as health care staff exposed to oral or respiratory secretions of a case.

<p>Epidemic control</p>	<p>Inform the health authorities if one or more suspected cases are identified.</p> <p>Confirm the suspected outbreak, following WHO guidelines.</p> <p>Investigate any probable case: check if it fulfils the case definition, record date of onset, age and vaccination status.</p> <p>Confirm the diagnosis: collect both nasal and pharyngeal swabs for culture and swabs from any wounds or skin lesions. If appropriate facilities are available, determine the biotype and toxigenicity of <i>C. diphtheriae</i>.</p> <p>Identify close contacts and define population groups at high risk. Adult contacts must avoid contact with children and must not be allowed to undertake food handling until proven not to be carriers.</p> <p>Implement outbreak response measures: give priority to case management and immunization of population in areas not yet affected where the outbreak is likely to spread.</p> <p>Immunize the population at risk as soon as possible, especially children. In an epidemic involving adults, immunize groups that are most affected and at highest risk. Repeat immunization procedures 1 month later to provide at least 2 doses to recipients.</p> <p>In endemic situations, Td vaccine (a combination of diphtheria and tetanus toxoids with reduced diphtheria content) should preferably be given.</p> <p>To ensure injection safety during immunization, auto-disable syringes and safety boxes are recommended. Safe disposal of used sharps should be ensured.</p>
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7. DRACUNCULIASIS (Guinea worm)

DESCRIPTION

Infectious agent	Nematode: <i>Dracunculus medinensis</i>
Case definition	<p>Clinical description: Diagnosis is usually easy and unambiguous: the gravid female worm (up to 1 meter long) emerges through the skin of any part of the body about one year after infection. When the anterior part of the worm reaches the surface of the skin an intensely painful oedema and a papule are formed. The papule is succeeded (within 1 to 3 days) by a blister which ruptures (after 3 to 5 days) leaving a small superficial ulcer. Systemic symptoms include fever, nausea and vomiting. Functional lesions of the affected limb are frequent. Lower extremities are involved in 90% of the cases with resulting crippling. Secondary bacterial infections are also of major concern. No immunity to infection develops, and people in endemic areas suffer from infection year after year. Each infection lasts about one year.</p> <p>Case definition: Anyone exhibiting or having a history of a skin lesion with the emergence of a Guinea worm within the current year.</p> <p>Diagnosis</p> <p>Confirmation by visual recognition of the adult worm protruding from a skin lesion or by microscopic identification of larvae.</p>
Mode of transmission	<p>Rupture of the blister or vesicle of an infected person</p> <p>Gravid female worm discharges larvae whenever the infected part is immersed in water.</p> <p>The discharged larvae are ingested by minute crustacean copepods – <i>Cyclops</i> – found in freshwater. The larvae develop into infective forms in 2 weeks.</p> <p>Ingestion by person</p> <p><i>Cyclops</i> may be swallowed through drinking infested water (step-in wells, ponds). Larvae are liberated in the stomach or duodenum and migrate through the viscera, becoming adults.</p> <p>After mating, the female migrates to subcutaneous tissues (legs) where it grows and develops to full maturity.</p> <p>The gravid female, 60–10 cm in length, migrates to the subcutaneous tissues (90% to the legs) to discharge larvae.</p>
Incubation	About 12 months
Period of communicability	<p>The time from rupture of the vesicle to complete evacuation of larvae from uterus of the gravid worm is 2–3 weeks.</p> <p>Larvae swimming in freshwater are infective for <i>Cyclops</i> for 5 days.</p> <p>Ingested larvae in <i>Cyclops</i> become infective to humans in 12–14 days and remain so for 1 month.</p> <p>There is no person-to-person transmission.</p>

EPIDEMIOLOGY

Burden	No case has been recorded since the 1980s. However, in May 2002, a field assessment found 32 cases of documented drancuculosis in communities visited.
Geographical distribution	Disease found in five districts of the Northern, Southern and Eastern provinces (Koinadugu; Kono, Kenema, and Kailahun; and Bo).
Seasonality	Insignificant
Alert threshold	A single suspected case
Recent epidemics	Not applicable

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Possible importation of cases by movement of population from endemic foci.
Overcrowding	No	
Poor access to health services	Yes	Untimely detection of cases and lack of health education.
Lack of safe water and poor sanitation	Yes	Unprotected and polluted water sources such as step-in wells, stagnant water, ponds etc. Unprotected and contaminated water sources especially wells, stagnant ponds and slow flowing streams
Others	Yes	Susceptibility is universal
Risk assessment conclusions		WHO adopted a resolution (WHA 44.5, May 1991) to eradicate dracunculiasis by 1995. Eradication has been successful in many parts of the world. Sierra Leone is currently implementing pre-certification surveillance. Districts still considered to be at highest risk are in Northern, Southern and Eastern provinces (Koinadugu; Kono, Kenema, and Kailahun; and Bo).

PREVENTION AND CONTROL MEASURES

Case management	<p>Prognosis is good unless bacterial infection of the lesions occurs. Specific treatment includes local treatment with topical antibiotics and occlusive bandages. Carefully controlled aseptic surgical extraction just before worm emergence is effective. Serious secondary infection results from accidental rupture of the worm, and can give rise to arthritis, synovitis, ankylosis, and contractures of the affected limb. Some drugs, including thiabendazole, albendazole, ivermectin and metronidazole, along with corticosteroids, may be of value.</p> <p>The disease is rarely fatal.</p>
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Prevention	<p>Provide health education programmes in endemic communities to convey key messages:</p> <ul style="list-style-type: none"> – guinea worm infection comes from drinking unsafe or unprotected water; – persons with blisters or ulcers should not enter any source of drinking-water; – drinking-water should be filtered through a fine-mesh cloth (such as nylon gauze of 100 µm) to remove copepods. <p>Provide safe water sources. Construction of protected springs and rainwater catchments can provide uninfected water.</p> <p>Control copepod populations in ponds, tanks, reservoirs and step-in wells by use of safe insecticide.</p> <p>Report all cases to local health authorities, as part of the WHO eradication programme. Strengthen community-based case containment and surveillance.</p> <p>Investigate contacts and sources of infection. Obtain information as to source of drinking-water at probable time of infection (up to 12 months previously). Search for other cases in community.</p>
Immunization	<p>Immunize high-risk populations against tetanus.</p>

8. HIV/AIDS

DESCRIPTION

<p>Infectious agent</p>	<p>Human immunodeficiency virus (HIV). Two types, HIV-1 and HIV-2, have been identified. They have similar epidemiological characteristics, but HIV-2 is less pathogenic than HIV-1.</p>
<p>Case definition</p>	<p><u>AIDS case definition</u> Acquired immunodeficiency syndrome (AIDS) is the late clinical stage of HIV infection, defined as an illness characterized by one or more indicator diseases.</p> <p><u>WHO Staging System for HIV Infection and Disease in Adults and Adolescents</u></p> <p>Stage 1 1. Asymptomatic 2. Persistent generalized lymphadenopathy (PGL) Performance Scale 1: <i>asymptomatic, normal activity</i></p> <p>Stage 2 3. Weight loss <10% of body weight 4. Minor mucocutaneous manifestations (seborrheic dermatitis, prurigo, fungal nail infections, recurrent oral ulcerations, angular cheilitis) 5. Herpes zoster within the past 5 years 6. Recurrent upper respiratory tract infections (e.g. bacterial sinusitis) And/or Performance Scale 2: <i>symptomatic, normal activity</i></p> <p>Stage 3 7. Weight loss >10% of body weight 8. Unexplained chronic diarrhoea, >1 month 9. Unexplained prolonged fever (intermittent or constant), >1 month 10. Oral candidiasis (thrush) 11. Oral hairy leukoplakia 12. Pulmonary tuberculosis within the past year 13. Severe bacterial infections (i.e. pneumonia, pyomyositis) And/or Performance Scale 3: <i>bedridden <50% of the day during the last month</i></p> <p>Stage 4 14. HIV wasting syndrome, as defined by the Centers for Disease Control and Prevention (CDC)^a 15. <i>Pneumocystis carinii</i> pneumonia 16. Toxoplasmosis of the brain 17. Cryptosporidiosis with diarrhoea >1 month 18. Cryptococcosis, extrapulmonary 19. Cytomegalovirus (CMV) disease of an organ other than liver, spleen or lymph nodes 20. Herpes simplex virus (HSV) infection, mucocutaneous >1 month, or visceral of any duration 21. Progressive multifocal leukoencephalopathy (PML) 22. Any disseminated endemic mycosis (e.g. histoplasmosis, coccidiomycosis) 23. Candidiasis of the oesophagus, trachea, bronchi or lungs 24. Atypical mycobacteriosis, disseminated 25. Non-typhoid <i>Salmonella</i> septicaemia 26. Extrapulmonary tuberculosis 27. Lymphoma 28. Kaposi sarcoma 29. HIV encephalopathy, as defined by CDC^b</p>

NOTE: Both definitive and presumptive diagnoses are acceptable

(a) HIV wasting syndrome: weight loss of >10% of body weight, plus *either* unexplained chronic diarrhoea (>1 month) *or* chronic weakness and unexplained prolonged fever (>1 month).

(b) HIV encephalopathy: clinical finding of disabling cognitive and/or motor dysfunction interfering with activities of daily living, progressing over weeks to month, in the absence of a concurrent illness or condition other than HIV infection that could explain the findings.

Expanded WHO case definition for AIDS surveillance^c

An adult or adolescent (>12 years of age) is considered to have AIDS if a test for HIV antibody gives a positive result, and one or more of the following conditions are present:

1. >10% body weight loss or cachexia, with diarrhoea or fever, or both, intermittent or constant, for at least 1 month, not known to be due to a condition unrelated to HIV
2. Cryptococcal meningitis
3. Pulmonary or extrapulmonary tuberculosis
4. Kaposi sarcoma
5. Neurological impairment that is sufficient to prevent independent daily activities, not known to be due to a condition unrelated to HIV infection (e.g. trauma or cerebrovascular accident)
6. Candidiasis of the oesophagus (which may be presumptively diagnosed based on the presence of oral candidiasis accompanied by dysphagia)
7. Clinically diagnosed life-threatening or recurrent episodes of pneumonia, with or without etiological confirmation
8. Invasive cervical cancer

^c *Weekly Epidemiological Record*. 1994, 69:273–275.

Laboratory evidence of HIV

This is most commonly based on detection of HIV antibody in serum samples using enzyme-linked immunoassay (ELISA or EIA). When this test is positive, it must be confirmed with another test of higher specificity such as the Western blot, the indirect fluorescent antibody (IFA) test or a second ELISA test that is methodologically and/or antigenically independent.

The rapid tests that are recommended by WHO have been evaluated at WHO collaborating centres and have levels of sensitivity and specificity comparable to WHO-recommended ELISA tests. The use of rapid HIV tests may afford several advantages in emergency and disaster settings: rapid tests that do not require refrigeration will be more suitable for remote and rural areas and sites without a guaranteed electricity supply. Long shelf-life is also important, especially for remote areas and sites performing smaller numbers of tests.

Many rapid tests require no laboratory equipment and can be performed in settings where electrical and water supplies need not be guaranteed.

Rapid tests can detect HIV antibodies in whole blood (finger prick samples) as well as serum/plasma, and testing may therefore be performed by non-laboratory personnel with adequate training and supervision.

Mode of transmission	<p>Sexual intercourse (vaginal or anal) with an infected partner, especially in presence of a concurrent ulcerative or non-ulcerative sexually transmitted infection (STI). The vast majority of transmission in Sierra Leone (over 90%) is sexual.</p> <p>Infected mother to her child during pregnancy, labour and delivery or through breastfeeding (perinatal transmission is the second main cause of transmission in Sierra Leone).</p> <p>Transfusion of infected blood or blood products.</p> <p>Contaminated needles, syringes, other injecting equipment and injecting solutions (contamination often occurs when drug solutions are mixed or when multiple users draw up solutions from a single container).</p>
Incubation	<p>Variable. On average, time from HIV infection to clinical AIDS is 8 to 10 years, though AIDS may be manifested in less than 2 years or be delayed in onset beyond 10 years. Incubation times are shortened in resource-poor settings and in older patients. They can be prolonged by provision of primary prophylaxis for opportunistic infections or antiretroviral treatment.</p>
Period of communicability	<p>Any person who is infected with HIV may pass the infection to another through the routes of transmission described above.</p> <p>Infectiousness is observed to be high during the initial period after infection. Studies suggest it increases further with increasing immune deficiency, clinical symptoms and presence of other STIs.</p>

EPIDEMIOLOGY

Burden	<p>Since the first case was diagnosed in 1987, 34 735 people tested positive for HIV up to 2002 according to the National Aids Secretariat (NAS). A cumulative total of 794 AIDS cases and 438 deaths were recorded officially.</p> <p>A national survey in 2002 (CDC, Atlanta) showed a seroprevalence of 4.9%; however, this was subsequently revised to 0.9% (subject to further verification by the NAS).</p> <p>At the health facility level, of 12 645 people tested in various facilities throughout the country in 2002, 787 (6.2%) tested positive for HIV.</p> <p>Among commercial sex workers (CSW), the estimated prevalence was 26.7% in 1995 and 70.7% in 1997.</p> <p>Prevalence among pregnant women attending antenatal clinics was estimated at 4.0% in 1995, 5.5% in 1996, 7.0% in 1997 and 11.5% in 2002.</p> <p>Estimated number of people actually living with HIV/AIDS at the end of 2001 (all people with HIV infection, whether or not developed symptoms of AIDS):</p> <table border="0"> <tr> <td>Adults (15–49):</td> <td>150 000 (7% of all adults)</td> </tr> <tr> <td>Women (15–49):</td> <td>90 000</td> </tr> <tr> <td>Children (0–15):</td> <td>16 000</td> </tr> </table> <p>Estimated number of deaths due to AIDS (2001): 11 000 Estimated number of living orphans (2001): 42 000</p>	Adults (15–49):	150 000 (7% of all adults)	Women (15–49):	90 000	Children (0–15):	16 000
Adults (15–49):	150 000 (7% of all adults)						
Women (15–49):	90 000						
Children (0–15):	16 000						
Geographical distribution	Countrywide, with more cases reported in urban than in rural settings						
Seasonality	Not applicable						
Alert threshold	One suspected case must be investigated						
Recent epidemics in the country	NA						

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	<p>Population movement may result in:</p> <ul style="list-style-type: none"> – breakdown of family and social ties – erosion of traditional values and coping strategies, which can result in higher-risk sexual behaviour and increased risk of HIV spread. <p>influence of illicit drug-trafficking and drug use, increasing risk of HIV transmission through injecting drug use.</p>
Overcrowding	Yes	<p>Groups with differing levels of HIV awareness and differing rates of infection are often placed together in crowded temporary locations, such as refugee camps or other temporary shelters, where there is greater potential for sexual contact. Overcrowding can also influence injecting drug use patterns and result in increased risk of sharing contaminated injecting equipment (which has been noted in refugee camps).</p>
Poor access to health services	Yes	<p>Without adequate medical services, STIs, if left untreated in the patient or partners, greatly increase the risk of acquiring HIV.</p> <p>Important materials for HIV prevention, particularly condoms, are likely to be lacking in an emergency situation.</p> <p>Similarly, services for drug dependence treatment usually do not exist. It is more likely to be difficult to access sterile injecting equipment.</p>
Food shortages	Yes	<p>The need for food is paramount in emergency situations, and exchanging sex for money to buy food and other essentials can occur (see “Sex work”, below).</p>
Lack of safe water and poor sanitation	No	
Others	Yes	<p><u>Sexual violence</u></p> <p>Refugees and IDPs are often physically and socially powerless, with women and children at particular risk of sexual coercion, abuse or rape. Sexual violence carries a higher risk of infection because the person violated cannot protect herself or himself from unsafe sex, and because the virus can be transmitted more easily if bodily tissues are torn during violent sex.</p> <p><u>Sex work</u></p> <p>Exchange of sexual favours for basic needs, such as money, shelter, security, etc, is common in or around refugee camps, and inevitably involves both the refugee and host communities. Both sex workers and clients are at risk of HIV infection if unprotected sex is practised.</p> <p><u>Injecting drug use</u></p> <p>In Sierra Leone, none of the AIDS cases officially reported from the beginning of the epidemic in 1987 to the end of 2001 had contracted the disease by injecting drugs. In the typical conditions of an emergency, it is highly likely that the drug injectors will be sharing needles, a practice that carries a very high risk of HIV transmission if one of the people sharing is infected.</p> <p><u>Unsafe blood transfusions</u></p> <p>Transfusion with HIV-infected blood is a highly efficient means of transmitting the virus. It is particularly difficult to ensure blood safety in emergency situations when transfusion services have broken down.</p> <p><u>Adolescent health</u></p> <p>Children in complex emergency settings may have little to occupy themselves which may lead them to experiment with sex earlier than children in other situations.</p>

<p>Risk assessment conclusions</p>	<p>As a result of years of population displacement, overcrowded living conditions, lack of access to health services and lack of knowledge of HIV/AIDS, transmission of HIV has gone unchecked, leading to rapidly increasing prevalence among high-risk groups such as pregnant women and CSWs.</p> <p>MICS-2 (2000) showed that only 54% of women aged 15–49 in Sierra Leone had heard of AIDS. The majority of women of childbearing age have either never heard of HIV/AIDS or lack basic knowledge regarding the disease.</p> <p>Sexual violence against women and girls (including rape), unwanted teenage pregnancies and unsafe abortions, low condom use and risky sexual behaviour have no doubt increased the risk for transmission of HIV and STIs in Sierra Leone.</p> <p>All stakeholders involved in humanitarian activities must be sensitized to the importance of addressing HIV/AIDS. Activities should include HIV prevention (promotion of safer sexual behaviours, treatment of STIs, blood safety) and care and support for people living with HIV/AIDS. They must reach vulnerable populations and address the needs of women and children.</p> <p>All stakeholders must also be sensitized to the HIV risks associated with injecting drug users and the need for drug dependence treatment and risk reduction education and counselling.</p>
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PREVENTION AND CONTROL MEASURES

<p>Case management</p>	<p>Provide high-quality care and support to all people living with HIV/AIDS (PLHA); this should include counselling, psychosocial support, treatment for opportunistic infections (e.g. TB), palliative care and access to antiretroviral therapy where feasible.</p> <p>Support PLHA to live normal and productive lives that are free of stigmatization and discrimination.</p>
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<p>Prevention</p>	<p><u>Reduce sexual transmission</u></p> <ul style="list-style-type: none"> – <i>Awareness and life skills education</i>, ensuring that all people, especially youth, are well informed of what does, and does not, constitute a mode of transmission; are told how and where to acquire free condoms and medical attention if necessary; and are given information on basic hygiene. MICS-2 (2000) showed that 29% of surveyed women correctly believed that having only one uninfected sex partner can prevent HIV transmission; only 27% correctly believed that using a condom every time they have sex can prevent HIV transmission; 27% correctly believed that abstaining from sex can prevent HIV transmission. Knowledge of the means of HIV/AIDS transmission varied dramatically across regions. All three ways of preventing HIV transmission were known by 2% of women in the north of Sierra Leone, 43% in the east, 20% in the south, and 33% in the west. Knowledge of HIV/AIDS transmission also varies greatly by level of education: women with secondary or higher education were more likely to know the modes of HIV transmission and how to prevent it than women with no education. – <i>Condom promotion</i>, including ensuring that good-quality condoms are freely available to those who need them, using culturally sensitive instructions and distribution. – <i>STI management</i>, including for sex workers, using the syndromic STI management approach, with partner notification and promotion of safer sex. <p><u>Reduce mother-to-child transmission of HIV</u></p> <ul style="list-style-type: none"> – Primary prevention of HIV among women, especially young women. – Avoidance of unintended pregnancies among HIV-infected women by promotion of family planning methods. – Preventing the transmission of HIV from infected pregnant women to their infants through: <ul style="list-style-type: none"> – antiretroviral prophylaxis regimen; – avoiding unnecessary obstetrical invasive procedures, such as artificial rupture of membranes or episiotomy; and – modifying infant feeding practices (replacement feeding given with a cup when acceptable, feasible, affordable, sustainable and safe; otherwise exclusive breastfeeding for the first 6 months of life is recommended. See: http://www.who.int/child-adolescent-health/New_Publications/NUTRITION/ISBN_92_4_159136_6.pdf) <p>MICS-2 (2000) showed that 34% of women in Sierra Leone correctly believed that AIDS can be transmitted from mother to child.</p> <p><u>Blood safety</u></p> <ul style="list-style-type: none"> – HIV testing of all transfused blood. – Avoidance of non-essential blood transfusion. – Recruitment of safe blood donor pool. <p><u>Prevention among injecting drug users</u></p> <ul style="list-style-type: none"> – Ready access to sterile needles, syringes and other injecting equipment (and disposal of used equipment). – HIV risk-reduction education and counselling for injecting drug users (including peer outreach when possible). – Service for treatment of drug dependence, including substitution treatment (e.g. methadone) where possible. – Access to STI and HIV/AIDS management for injecting drug users.
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	<p><u>Universal precautions</u></p> <ul style="list-style-type: none"> – Thorough hand-washing hands with soap and water, especially after contact with body fluids or wounds. – Use of protective gloves and clothing when there is risk of contact with blood or other potentially infected body fluids. – Safe handling and disposal of waste material, needles, and other sharp instruments. – Proper cleaning and disinfection of medical instruments between patients. <p><u>Physical protection</u></p> <p>The protection of the most vulnerable, especially women and children, from violence and abuse is not only an important principle of human rights but is also essential for reducing the risk of HIV infection.</p>
<p>Protecting health care workers</p>	<p>In order to reduce nosocomial transmission, health workers should strictly adhere to universal precautions with all patients and laboratory samples – whether or not known to be infected with HIV.</p> <p>Health care workers should have access to voluntary counselling, testing and care. Health workers deployed in complex emergencies often experience significant occupational stress and those tested as part of the management of occupational exposures will require additional support.</p>
<p>Voluntary counselling and testing programmes</p>	<p>The establishment of voluntary counselling and testing services to help individuals make informed decisions on HIV testing should be considered when relative stability is restored. Displaced populations are often coerced into testing, or are required to make decisions with regard to testing, when they are suffering acute or post-traumatic stress disorders.</p> <p>As refugees are often tested prior to resettlement in other countries, it is critical that they receive counselling on the legal and social implications of the test. Often, migration or temporary residency status is contingent on the applicant's having HIV antibody seronegative status.</p> <p>Post-test counselling is essential for both seronegative and seropositive results. Refugees and conflict survivors who are already traumatized will require additional psychosocial support if they test seropositive. Typically the support networks of displaced persons are disrupted and suicide risk assessment forms an important part of post-test counselling in a refugee or conflict context.</p> <p>Orphaned minors should be tested only where there is an immediate health concern or benefit to the child and with the consent of the child's official guardian. There should be no mandatory screening prior to admittance to substitute foster care.</p>
<p>Immunization</p>	<p>Asymptomatic HIV-infected children should be immunized with the EPI vaccines. Symptomatic HIV-infected children should NOT receive either BCG or yellow fever vaccine.</p>

9. LASSA FEVER

DESCRIPTION

Infectious agent	Lassa virus (genus <i>Arenavirus</i> , family <i>Arenaviridae</i>)
Case definition	<p>While Lassa fever is mild or causes no observable symptoms in about 80% of people infected with the virus, the remaining 20% have a severe multi-system disease (15–20% mortality in hospitalized patients). Lassa fever is also associated with occasional epidemics, during which the case fatality rate can reach 50%.</p> <p><u>Clinical case definition</u></p> <p>An illness of gradual onset with one or more of the following:</p> <ul style="list-style-type: none"> – malaise, fever, headache, sore throat, cough, nausea, vomiting, diarrhoea, myalgia, chest pain, hearing loss, and – a history of contact with excreta of rodents or with a probable or confirmed case of Lassa fever. <p><u>Laboratory criteria</u></p> <p>Isolation of Lassa virus from a clinical specimen (e.g. blood, throat swabs, urine and other tissues) by immunohistochemistry (post-mortem diagnosis) or RT-PCR (reverse transcriptase–polymerase chain reaction), or Serological diagnosis.</p> <p>The most common diagnostic test is the enzyme-linked immunosorbent assay (ELISA), which can detect IgM antibody (acute infection) and IgG antibody (recent infection) as well as Lassa virus antigen.</p> <p><u>Case classification</u></p> <p>Suspected: A case compatible with the clinical description. Probable: A suspected case that is epidemiologically linked to a confirmed case. Confirmed: A suspected case that is laboratory confirmed.</p> <p>The most important differential diagnoses include falciparum malaria, typhoid, other viral haemorrhagic fevers, meningococcaemia and septicaemia. In an endemic area of Sierra Leone, the combination of fever, exudative pharyngitis, retrosternal pain and proteinuria was able to distinguish Lassa fever from other febrile illness with a positive predictive value of 80%.</p> <p>Sensorineural deafness occurs as a late complications in 30% of patients, and is often permanent. It is thought to be immune-mediated.</p>
Mode of transmission	<p><u>Rodent to human</u></p> <p>The only known reservoir is wild rodents – in west Africa, the multimammate rat of the <i>Mastomys</i> genus. It is not certain which species of <i>Mastomys</i> are associated with Lassa, but the species <i>M. huberti</i> and <i>M. erythroleucus</i>, and <i>M. natalensis</i> are known to carry the virus in Sierra Leone.</p> <p>Infected rats continually shed virus in their excreta; transmission occurs primarily through virus-containing aerosol (inhalation of tiny airborne particles contaminated with rodent excretions), by direct contact of abraded skin and mucous membranes with urine or droppings deposited on surfaces such as floors or beds, or by ingestion of food and water contaminated with rodent excreta.</p> <p><i>Mastomys</i> are common domestic rodents in West Africa, and highly commensal with humans, scavenging on food remains or poorly stored food. Their movement within a village is limited, usually near the house they occupy, and most virus transmission to humans takes place in and around homes. Rodent to human transmission is also associated with practices such as catching, cooking, and eating rodents.</p>

	<p><u>Person to person</u></p> <p>Person-to-person transmission occurs when a person is exposed to blood, tissue, secretions, or excreta of an individual infected with the Lassa virus. Person-to-person spread in households is common although less frequent than rodent-to-human spread. Risk of infection is usually associated with direct contact or sexual contact with, or nursing care of, someone infected (see “Period of communicability” below).</p> <p><u>Nosocomial and laboratory-associated</u></p> <p>Spread in hospitals can occur through pharyngeal secretions or urine of a patient, through exposure to blood during surgery, or through contaminated medical equipment. Prominent early in epidemics, these modes of transmission can be effectively prevented with simple isolation and barrier nursing techniques.</p> <p>Laboratory spread can occur by direct contact with blood, secretions or contaminated equipment such as needles and other sharp instruments.</p>
Incubation	Incubation period is usually 6 to 21 days.
Period of communicability	Person-to-person spread may occur during the acute febrile phase when virus is present in the throat, or during the convalescent phase , when virus can be excreted in urine and semen of patients (3–12 weeks from onset of illness).

EPIDEMIOLOGY

Burden	<p>Lassa fever is endemic in Sierra Leone and, before the declaration of the ongoing outbreak in 1996, only a few cases were recorded in the country annually.</p> <p>The number of Lassa virus infections per year in west Africa is estimated at approximately 300, with approximately 1.5% mortality (2001 data). Most of these cases occur in Sierra Leone. Unfortunately, such estimates are crude because surveillance is not uniformly performed.</p> <p>In Guinea, between October 1998 and March 2002 (42 months), 7 out of 24 detected cases were positive; no cases were diagnosed in Côte d’Ivoire in the same period, (Survey/Research Project on VHF in West Africa, report by the International Co-operation with Developing Countries (INCO-DC/Epicentre)).</p>
Geographical distribution	<p>Lassa fever has been endemic in eastern Sierra Leone since first diagnosed in 1971 as part of an epidemic originating in Panguma. The area of endemicity is a triangle defined by Kailahun, Tongo and Kenema, also known as the “Lassa belt”. Most cases have been reported in this region, although in 2002 and 2003, some cases were reported from Bo district, west of this traditional endemic zone.</p>
Seasonality	Disease rates are expected to peak in the dry season (November to April).
Alert threshold	One probable case must lead to an alert.
Recent epidemics in the country	<p>Epidemics have been reported in recent years. Three outbreaks occurred between 1996 and 2000 in Kenema district.</p> <p>In 1996, 470 cases with 110 deaths were reported (CFR = 23.4%). From January to April 1997, 353 cases with 43 deaths were reported (CFR = 12.2%).</p> <p>Between January and April 2003, a total of 133 cases of suspected Lassa fever with 11 deaths were recorded – more than 80% of these were in refugees in camps in south-eastern Sierra Leone. Diagnosis of all cases was on the basis of clinical suspicion. Two of the camps, Jimmi Bagbo and Gerihun, are located in Bo district, a region previously thought to be outside the Lassa fever endemic area.</p> <p>Reliable data on the number of cases are not available as laboratory confirmation of cases is difficult (requiring P4 level laboratory) and not done systematically.</p>

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Massive population movement with subsequent overcrowding is closely related to increased transmission of Lassa fever. Rates of seroconversion range from 5% to 20% in village populations in Sierra Leone . The highest rates are in overcrowded, highly mobile populations.
Overcrowding	Yes	See above.
Poor access to health services	Yes	<p>Person-to-person transmission can easily be prevented by managing patients in isolation wards and applying appropriate infection control measures with barrier nursing. In conflict situations, isolation and protective measures are often compromised and can result in transmission to staff and other patients.</p> <p>Poor access to health services also leads to an increased exposure in the community as the disease is unrecognized and untreated.</p>
Food shortages	No	
Lack of safe water and poor sanitation	Yes	Lack of hygiene increases the chances of contact with objects and/or food contaminated with rodent excreta.
Others	Yes	<p>Increase in the reservoir population. <i>Mastomys</i> rodents breed very frequently, and produce large numbers of offspring. Lassa virus can be transmitted horizontally between rodents, as well as vertically to their offspring.</p> <p>Poor environmental sanitation (attracts rodents).</p> <p>Unsafe food handling and storage practices (storing food, water in non-sealable containers where rats can access).</p> <p>Practices such as catching, cooking, and eating rodents.</p>
Risk assessment conclusions		<p>Lassa Fever is a serious public health problem in Sierra Leone and outbreaks with high mortality have occurred regularly since 1996 in the traditional Lassa belt in eastern Sierra Leone.</p> <p>Furthermore, there may be spread beyond the traditional endemic zone possibly due to:</p> <ul style="list-style-type: none"> – environmental changes during the past 10 years that have changed the distribution of Lassa virus-infected <i>M.natensis</i>, the natural host of Lassa virus in Sierra Leone; – migration and displacement of populations of infected individuals from the previously endemic region, which has resulted in cases of Lassa fever in new areas. <p>Hospitals in Sierra Leone do not have access to advanced diagnostic technologies and, as clinical symptoms of Lassa fever are nonspecific, physicians rely on differential diagnosis to identify the disease. This, combined with poor access to health services and a poorly developed referral system, makes early diagnosis and treatment very difficult.</p> <p>The risk for further outbreaks is high given continuing population movements (returnees, resettlement), overcrowding in camps and communities, poor hygiene and sanitary conditions, poor environmental management, and low access to health care in some areas.</p>

PREVENTION AND CONTROL MEASURES

<p>Case management</p>	<p>Ribavirin, an antiviral drug, has been used with success in Lassa fever patients. It has been shown to be most effective in decreasing viraemia and reducing the mortality rate when given intravenously early in the course of the illness, especially within the first 6 days of fever.</p> <p>Medication should be given orally or intravenously. Intramuscular and subcutaneous injections are contraindicated because of the risk of haematomas.</p> <p>Evidence about the effectiveness of oral ribavirin in Lassa fever treatment is not available, however oral ribavirin therapy <i>may</i> be attempted where IV therapy is not feasible. General supportive treatment, as well as treatment of any other complicating infection are also very important in the management of Lassa patients.</p> <p>Side-effects of ribavirin are largely restricted to reversible haemolysis. Plasma transfusion has not shown to be beneficial during Lassa fever convalescence and is not recommended, especially due to the potential for transmitting other viruses such as HIV and HBV.</p> <p><u>Intravenous ribavirin treatment</u></p> <p>The threshold number of cases at which intravenous therapy becomes impossible depends on a variety of factors, including number of patients and local health care resources.</p> <p>Adults</p> <ol style="list-style-type: none"> 1. Loading dose* of 17mg/kg IV (max. 1 g per dose) 2. Followed by 17 mg/kg IV (max. 1 g per dose) every 6 hours for 4 days 3. Followed by 8 mg/kg IV (max. 500 mg per dose) every 8 hours for 6 days. <p>*If there is some delay in beginning the treatment a loading dose of 30 mg/kg IV (max. 2 g) might be necessary.</p> <p>Pregnant women</p> <p>Same as for adults. Ribavirin is contraindicated in pregnancy; in the context of VHF, however, the benefit appears likely to outweigh any risk to the foetus of ribavirin therapy (the associated mortality of VHF tends to be higher in pregnancy), and ribavirin is therefore recommended.</p> <p>Children</p> <p>Same as for adults, dosed according to weight.</p> <p><u>Oral ribavirin treatment</u></p> <p>Adults</p> <ol style="list-style-type: none"> 1. Loading dose of 2000 mg orally once. 2. Followed by 1000 mg orally every 6 hours for 4 days. 3. Followed by 500 mg orally every 6 hours for 6 days. <p>Pregnant women</p> <p>Same as for adults.</p> <p>Children</p> <ol style="list-style-type: none"> 1. Loading dose of 30 mg/kg orally once. 2. Followed by 15 mg/kg every 6 hours for 4 days. 3. Followed by 7 mg/kg every 6 hours for 6 days.
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	<p><u>Supportive treatment</u></p> <p>All Lassa fever patients should receive supportive treatment, with careful maintenance of fluid and electrolyte balance, circulatory volume, blood pressure and oxygenation, as well as treatment of any other complicating infection. Mechanical ventilation, renal dialysis, and anti-seizure therapy may be required.</p> <p>Medication should be given orally or intravenously. Intramuscular and subcutaneous injections are contraindicated because of the risk of haematomas.</p> <p>Approximately 15–20% of patients hospitalized for Lassa fever die from the illness. Overall, however, only about 1% of infections with Lassa fever result in death. The death rates are particularly high for women in the third trimester of pregnancy, and for the fetuses of infected pregnant, about 95% of which die in utero.</p> <p><u>Protective measures</u></p> <p>Patients with probable or confirmed Lassa fever should be isolated and cared for using barrier nursing techniques. Isolation precautions, to reduce the risk of transmission of Lassa fever in the health care setting, should follow the guidelines developed by WHO/CDC.</p> <p>See:</p> <ul style="list-style-type: none"> – “VHF outbreak control” in <i>Guidelines for Outbreak Control</i>, in this Toolkit. – <i>Infection control for viral haemorrhagic fevers in the African health care setting</i>. Geneva, WHO, 1998 (WHO/EMC/ESR/98.2, available at: http://www.who.int/emc-documents/haem_fevers/whoemcesr982c.html) <p>Universal precautions must be observed when handling specimens of blood or tissues, and when disposing of waste material, needles, or other sharp instruments.</p> <p>See:</p> <ul style="list-style-type: none"> – “Prevention” in Section 8, “HIV/AIDS”. – Annex 8 in <i>Guidelines for Collection of Specimens for Laboratory Testing</i>, in this Toolkit.
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<p>Prevention</p>	<p><u>Rodent control</u></p> <p>The key to prevention and control would be to eliminate contact with rodents. Rodent control should include adequate site planning, sanitation facilities, safe refuse disposal, environmental sanitation, development of local traps and use of cats to catch rats. Although studies that have involved trapping and destruction of rodents (trap-out studies) have been effective in Sierra Leone, controlling the rodent population as the only means to prevent Lassa fever is unrealistic and not sustainable.</p> <p><u>Safe food storage, personal hygiene and environmental sanitation</u></p> <ul style="list-style-type: none"> – Safe water and food storage in solid, sealed containers so those rats cannot contaminate them. – Personal hygiene, environmental sanitation and hand-washing. – Elimination of rat habitats and minimizing of activities that produce aerosols containing rodent excreta. <p>Educational programmes on the above measures and transmission modes are essential in Lassa fever control.</p> <p><u>Prevention of nosocomial spread</u></p> <p>Basic barrier nursing methods (gloves, gowns and masks) are highly effective in preventing secondary spread of the infection.</p> <p>Strict isolation with rigorous barrier nursing should be combined with full medical care, including surgery if indicated, to ensure the safety of the staff and survival of the patient.</p> <p>Extensive nosocomial epidemics may result from reuse of inadequately sterilized equipment (needles, syringes, gloves) during surgery or midwifery.</p>
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10. LYMPHATIC FILARIASIS

DESCRIPTION

Infectious agent	Helminth: <i>Wuchereria bancrofti</i> , a filarial worm belonging to the class Nematoda.
Case definition	<p><u>Clinical case definition</u></p> <p>Hydrocele or lymphoedema (in a resident of an endemic area) for which other causes have been excluded.</p> <p><u>Laboratory criteria</u></p> <p>Positive parasite identification by:</p> <ul style="list-style-type: none"> – direct blood examination, or – ultrasound (adult worms moving), or – positive antigen-detection test. <p><u>Case classification</u></p> <p>Suspected: Not applicable. Probable: A case that meets the clinical case definition. Confirmed: A person with positive laboratory criteria even if he/she does not meet the clinical case definition.</p> <p>The burden of lymphatic filariasis, as measured in disability-adjusted life years (DALYs), is the highest of all tropical diseases after malaria.</p>
Mode of transmission	Bite of infected blood-feeding female mosquitoes (mainly <i>Anopheles</i> spp, also <i>Culex</i> spp.) which transmit immature larval forms of the parasitic worms from human to human.
Incubation	<p><u>1 month to 1 year and more</u></p> <p>Recidivant attacks of “filarial fever” (pain and inflammation of lymph nodes and ducts, often accompanied by fever, nausea and vomiting).</p> <p><u>5 to 20 years</u></p> <p>Chronic illness manifestations may include elephantiasis (massive swelling of limbs), hydrocele (swelling of the scrotum in males), and enlarged breasts in females.</p>
Period of communicability	As long as microfilariae (pre-larval stage released by adult female parasite) are present in the peripheral blood (6–12 months to 5–10 years after the infective bite).

EPIDEMIOLOGY

Burden	Sierra Leone is in the African tropical endemic region. In a study conducted in 1999, 14.5% of the subjects examined (by antigen detection) were found to be positive for <i>W.bancrofti</i> . Those examined came from 55 towns (including Freetown); prevalence was similar in different areas of the country (eastern area 12.7%; northern area 19.6%; southern area 10.9%; western area 12.8%).
Geographical distribution	The disease is widespread in the country.
Seasonality	Not known
Recent epidemics	The disease is not outbreak prone.

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Disease-free population can be displaced into endemic areas. Furthermore, in endemic areas acute manifestations of filariasis tend to develop more often and earlier in refugees or newcomers than in local populations who are continuously exposed to infection.
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Overcrowding	Yes	The proximity of people in conditions of overcrowding increases the risk of transmission.
Poor access to health services	Yes	Lack of early diagnosis and treatment due to difficulties in accessing health services (geographical, financial, security) increases the risk of transmission.
Food shortages	No	
Lack of safe water and poor sanitation	Yes	<p>Providing safe water is a means of secondary prevention (prevention of disease progression, not of the infection) since it facilitates some of the hygienic measures recommended for the affected body parts.</p> <p>Poor sanitation may contribute to creation of breeding sites for mosquito vectors (especially <i>Culex</i> spp.).</p>
Others	Yes	There is an established link between the degree of poverty and the prevalence of lymphatic filariasis.
Risk assessment conclusions		<p>Lymphatic filariasis is highly endemic and widespread in Sierra Leone. The infections seen in young children indicate that transmission intensity may be very high in some areas.</p> <p>The national prevalence of the infection may well be higher than figures shown under "Burden", as many of the subjects involved in that study came from urban areas; in areas where <i>W. bancrofti</i> is transmitted by anopheline mosquitoes, the disease tends to be more endemic in rural communities.</p> <p>The complex emergency situation of Sierra Leone was one of the reasons why the country has not so far been included in the Global Programme to Eliminate Lymphatic Filariasis (GPELF). Efforts to control lymphatic filariasis in Sierra Leone may therefore be suboptimal, and the situation poses a risk for elimination of LF in neighbouring countries since Sierra Leone could represent a source of transmission.</p> <p>Completion of health mapping for lymphatic filariasis in coming years should allow localization of populations at risk. It will then be possible to implement control programmes, monitor drug coverage over time, and monitor the elimination of the disease in space and time.</p> <p>The introduction of GPELF in Sierra Leone would bring benefits "beyond filariasis"; for example, albendazole is also an effective and safe drug for treating soil-transmitted helminth infections, and ivermectin is also effective against many intestinal parasites, scabies and lice.</p>

PREVENTION AND CONTROL MEASURES

<p>Case management</p>	<p>Hygiene measures for the affected body parts (and, when necessary, antibiotics and antifungal agents) can reduce the risk of adenolymphangitis:</p> <ul style="list-style-type: none"> – Wash the affected parts twice daily with soap and clean water. – Raise the affected limb at night. – Exercise to promote lymph flow. – Keep nails short and clean. – Wear comfortable footwear. – Use antiseptic or antibiotic creams to treat small wounds or abrasions; in severe cases, systemic antibiotics may be necessary. <p>Drug regimen for individual microfilarial positive patients:</p> <ul style="list-style-type: none"> – Diethyl carbamazine (DEC), 6 mg/kg single dose for 12 days, repeated at intervals of 1–6 month if necessary. Since the use of DEC in patients with either onchocerciasis or loiasis can be unsafe, it is important that patients with bancroftian filariasis who live in areas endemic for these other infections be examined for coinfection with these parasites before being treated with DEC. – Alternatively, ivermectin and albendazole can be used. Ivermectin, though very effective in decreasing microfilaraemia, appears not to kill adult worms (i.e. it is not macrofilaricidal) and thus does not cure infection completely. – Albendazole can be macrofilaricidal for <i>W. bancrofti</i> if given daily for 2–3 weeks, but optimization of its usage has not been attempted.
<p>Prevention and control</p>	<p>Prevention of infection can be achieved only by reducing contact between humans and vectors or by treating the human host to reduce the amount of infection the vector can acquire.</p> <p>A. Population level</p> <p>Filariasis control through reducing the number of vectors has proved largely ineffective. Even when good mosquito control can be implemented, the long lifespan of the parasite (4–8 years) means that the infection remains in the community for a long period of time, generally longer than the period over which intensive vector control efforts can be sustained.</p> <p>The recent advent of the extremely effective single-dose, once-yearly drug regimen has permitted an alternative approach and the launch of GPELF in 1998. When Sierra Leone is included in GPELF, the following steps will need to be taken:</p> <ul style="list-style-type: none"> – The national territory is divided into areas called Implementation Units (IUs). – In IUs known to be endemic, where the prevalence is >1%, mass drug administration (MDA) will be implemented. – In each IU where lymphatic filariasis status is uncertain, a village will be selected that has the greatest risk of transmission (or will be randomly selected if there is no information at all). – In the selected villages, a sample of 250 persons aged 15 years and over should be examined using the immunochromatographic card test (ICT). If any person has a positive result, the IU should be classified as endemic. – For each village the number of persons examined and the number of persons positive is required for the calculation of prevalence. – MDA will be implemented if the prevalence in the IU is >1%. <p>GPELF has two main goals:</p> <ul style="list-style-type: none"> – to interrupt transmission of infection; and – to alleviate and prevent suffering and disability caused by the disease. <p><u>To interrupt transmission of infection</u></p> <p>The entire at-risk population must be treated for a long enough period to ensure that levels of microfilariae in the blood remain below those necessary to sustain transmission. Therefore, a <i>yearly, 1-dose</i> MDA of the following drugs must be</p>

	<p>given:</p> <p>Areas with concurrent onchocerciasis:</p> <ul style="list-style-type: none"> – Albendazole 400 mg + ivermectin 150 µg/kg of body weight once a year for 4–6 years. <p>Areas with no concurrent onchocerciasis:</p> <ul style="list-style-type: none"> – Albendazole 400 mg + DEC 6 mg/kg of body weight once a year for 4–6 years, or – DEC-fortified salt for daily use for at least 6 -12 months. <p>Areas with concurrent loiasis:</p> <ul style="list-style-type: none"> – Systematic mass interventions cannot be envisaged because of the risk of severe adverse reactions in patients with high-density <i>Loa loa</i> infections (about 1 in 10 000 treatments). <p><u>To alleviate and prevent suffering and disability</u></p> <ul style="list-style-type: none"> – <i>Increase lymph flow</i> through elevation and exercise of the swollen limb. – <i>Decrease secondary bacterial and fungal infections</i> of limbs or genitals where the lymphatic function has already been compromised by filarial infection. Secondary infection is the primary determinant of the worsening of lymphoedema and elephantiasis. <p>Scrupulous hygiene and local care are dramatically effective in preventing painful, debilitating and damaging episodes of lymphangitis. Such care consists of regular washing with soap and clean water, daily exercising of the limbs, wearing of comfortable footwear, and other simple, low-cost procedures that can be carried out at home (see “Case management” for details).</p> <p>Whereas MDA can be generally expected to reduce or interrupt transmission of LF, the goal of GPELF could be achieved more rapidly through additional vector control in some situations. Where MDA coverage rates or duration are limited, the added impact of effective vector control can most usefully augment the GPELF.</p> <p>B. Individual level</p> <p>Lymphatic filariasis vectors usually bite between the hours of dusk and dawn. Contacts with infected mosquitoes can be reduced through the use of repellents, bednets or insecticide-impregnated materials.</p>
Epidemic control	Because of relatively low infectivity and long incubation, outbreaks of lymphatic filariasis are unlikely.

11. MALARIA

DESCRIPTION

Infectious agent	In Sierra Leone, more than 85% of all malaria cases are caused by the protozoan parasite <i>Plasmodium falciparum</i> . This causes the most life-threatening form of the disease. <i>P. malariae</i> and <i>P. ovale</i> are responsible for the remaining malaria burden. <i>P. vivax</i> is not present in the country.
Case definition	<p><u>Uncomplicated malaria</u></p> <p>Patient with fever or history of fever within the past 48 hours (with or without other symptoms such as nausea, vomiting and diarrhoea, headache, back pain, chills, myalgia).</p> <p><u>Severe malaria</u></p> <p>Patient with the same symptoms as for uncomplicated malaria, plus drowsiness with extreme weakness and associated signs and symptoms related to organ failure (e.g. disorientation, loss of consciousness, convulsions, severe anaemia, jaundice, haemoglobinuria, spontaneous bleeding, pulmonary oedema and shock).</p> <p>Confirmed case (uncomplicated or severe)</p> <p>Patient with uncomplicated or severe malaria <i>with laboratory confirmation of diagnosis</i> by malaria blood film or other diagnostic test for malaria parasites.</p> <p>In Sierra Leone, children with fever and no general danger sign or stiff neck should be classified as having malaria. Although a number of children will be treated for malaria when they in fact have another febrile illness, treatment for malaria is justified given the high malaria risk and the possibility that another illness might cause the malaria infection to progress.</p>
Mode of transmission	<p>The malaria parasite is transmitted by various species of <i>Anopheles</i> mosquitoes, which bite mainly between sunset and sunrise.</p> <p>In Sierra Leone, the primary malaria vectors are: <i>An. melas</i> in the coastal area of Freetown, and <i>An. gambiae</i> and <i>An. funestus</i> in the rest of the country. In a study conducted in a high-rainfall, forested area in the Bo District (southern Sierra Leone), <i>An. gambiae</i> was the most abundant species anywhere, especially in low-altitude villages. <i>An. funestus</i>, on the other hand, was more common in high-altitude than low-altitude villages and was found to be mainly a dry-season vector. Malaria may also be transmitted by injection of infected blood. Rarely, infants may contract malaria in utero (through transplacental transfer of parasites) or during delivery.</p>
Incubation	The incubation period is approximately 7–14 days for <i>P. falciparum</i> , 8–14 days for <i>P. ovale</i> and 7–30 days for <i>P. malariae</i> . However, malaria should be considered in all cases of unexplained fever that starts at any time between 1 week after the first possible exposure to malaria risk and 2 months (or even more in rare cases) after the last possible exposure.
Period of communicability	Communicability is related to the presence of infective <i>Anopheles</i> mosquitoes and of infective gametocytes in the blood of patients. Untreated or insufficiently treated patients may be a source of mosquito infection for more than 3 years in <i>P. malariae</i> malaria, and usually not more than 1 year in <i>P. falciparum</i> malaria.

EPIDEMIOLOGY

Burden	<p>The prevalence of <i>P. falciparum</i> in a cohort of over 900 children aged 0–7 years living in a rural area of the country (Bo District, in the south) was found to be approximately 61%, both before and after the rainy season of 1992. <i>Plasmodium malariae</i> rates measured in the same children were approximately 12%, and <i>P. ovale</i> rates averaged about 1%.</p> <p>The MICS-2 Survey Report (2000) showed that almost half the children under 5 years in Sierra Leone had been ill with fever within the 2 weeks before the survey, and 6 out of 10 ill children had taken antimalarial medication. This suggests that 30% of children under 5 years were taking antimalarial medication every 2 weeks.</p>
Geographical distribution	<p>Risk of transmission is high in the whole country, including the cities.</p> <p>The prevalence of fever is approximately the same for urban and rural children, while differences are seen across regions. The northern regions have the highest morbidity and the fewest resources in terms of prevention and treatment. The percentage of children under 5 ill with fever in northern Sierra Leone is higher than in any other region, while the percentages of ill children receiving appropriate antimalarial drugs and of children sleeping under impregnated mosquito nets are lower in the north than in any other region.</p>
Seasonality	<p>Malaria risk exists throughout the year, especially during the rainy season from May to September. A study showed that the highest prevalence occurs in August, the wettest month of the year.</p>
Alert threshold	<p>Any increase in the number of cases above what is expected for that time of the year in a defined area.</p>
Recent epidemics	<p>No data available.</p>

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	The potential for epidemics can increase with the influx of non-immune populations moving from areas with no malaria, or with low transmission, to highly endemic areas.
Overcrowding	Yes	As a result of increased population density and increased exposure to mosquito bites in temporary shelters.
Poor access to health services	Yes	Delays in access to effective treatment increase the likelihood of severe disease and death. Delays in access to effective treatment also increase the pool of carriers of the malaria gametocyte (the mature sexual stage of the parasite in humans which, once picked up in the blood meal of a mosquito, develops into the infective stage for transmission to another human).
Food shortages	No	However, malnutrition increases vulnerability to severe malaria once infection has occurred. Case management also becomes more complicated, resulting in increased mortality.
Lack of safe water and poor sanitation	No	However, temporary stagnant surface water bodies may increase malaria vector breeding opportunities
Others	Yes	Breakdown of control measures, and lack of preventive interventions (insecticide-treated materials such as bednets, sheeting, etc. and residual insecticide spraying of shelters) contribute to the increase of the malaria burden.

<p>Risk assessment conclusions</p>	<p>Although Sierra Leone was the first country in Africa to institute anti-anopheline measures, malaria still remains one of the major health problems, causing great suffering and loss of life. Control is becoming increasingly difficult, and the situation has deteriorated in the last few years.</p> <p>In the first 4 months of 2000, the Ministry of Health and Sanitation (MoHS) surveillance system reported malaria as the leading cause of morbidity among the 19 diseases targeted for routine surveillance. It accounted for 49% of the total disease burden, followed by acute respiratory infections (31%), and diarrhoea and dysentery (1.1%).</p> <p>Malaria is hyperendemic in Sierra Leone and transmitted throughout the year (see above under "Seasonality").</p> <p>Studies by the MoHS in Moyamba District showed an increase in the prevalence of malaria from 20.6% in 1978 to 45.1% in 1990.</p> <p>Malaria infection is highly prevalent in children, indicating that the disease is a significant public health problem in the country.</p> <p>In a study conducted among indigenous, parturient women in Freetown, 18.5% of placentas were found to be infected with malaria, with <i>P. falciparum</i> being the dominant species. The proportion of low-birth-weight babies from infected placentas (22.5%) was significantly greater than the proportion from uninfected (9.6%).</p> <p>The current approach to malaria control in Sierra Leone is both curative and preventive. However, while the majority of ill children are taking some form of medication, very few are using bednets.</p> <p>MICS-2 showed that only 15% of children under 5 years slept under a bednet the night before the survey interview. This percentage was constant across all age groups. Overall, only about 10% of the bednets used are impregnated with insecticide, indicating that only about 1.5% of children under 5 years sleep under insecticide-treated bednets in Sierra Leone.</p> <p>Non-prescribed artemisinin-based compounds such as artesunate, available over the counter and employed in suboptimal doses, could pose a new threat to malaria control in the country, as their indiscriminate and injudicious use may lead to increased levels of resistance among the local strains of <i>P. falciparum</i>.</p>
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PREVENTION AND CONTROL MEASURES

<p>Case management</p>	<p><u>Current national treatment policy for <i>P. falciparum</i> malaria</u></p> <ul style="list-style-type: none"> - Uncomplicated malaria unconfirmed: chloroquine laboratory-confirmed: chloroquine - Treatment failure sulfadoxine–pyrimethamine (SP) - Severe malaria quinine (7 days) <p>Drugs should be provided free of charge at all health facilities.</p>
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	<p>Recent antimalarial drug efficacy studies indicate high resistance of <i>P. falciparum</i> to both chloroquine and sulfadoxine–pyrimethamine. An alternative first-line treatment would be artemisinin-based combination therapy (ACT). The MoHS is planning a consensus meeting to review national treatment policy.</p> <p>Malaria diagnosis is done by rapid diagnostic tests (RDT) in some clinics. Some RDTs (HRP-II) can stay positive for 7–14 days after successful treatment in a substantial proportion of individuals, and a positive RDT in a highly endemic area is not always a reliable indicator of the cause of disease. RDTs may lose their sensitivity when stored in hot and humid conditions. It is recommended that heat-stability data are requested from the manufacturer before purchase.</p>
<p>Prevention and control</p>	<p>Malaria control in Sierra Leone used to be based largely on mosquito eradication. However, experience has shown that mosquito control is often not cost-effective in areas where the interruption of transmission cannot be sustained. Emphasis should now be placed on early diagnosis, treatment with effective antimalarials, and the selective use of preventive measures, including vector control and insecticide-treated materials, where they can be sustained.</p> <p>Insecticide-treated bednets (ITNs) for individual and family protection have proved their efficacy in reducing annual morbidity and mortality. They also have the potential for reducing transmission when used on a large scale.</p> <p>Periodic spraying of shelters with residual insecticide reduces transmission and is recommended in refugee camps, particularly among populations with low immunity occupying mud huts or houses. When large numbers of dwellings are sprayed, a mass effect on the density of vector population can result.</p> <p>Environmental control may be difficult except on a local scale, and impact is often limited. To reduce the number of vector breeding sites:</p> <ul style="list-style-type: none"> – drain clean water around water tap stands and rainwater drains – use larvicides in vector breeding sites if these are limited in number (seek expert advice) – drain ponds (although this may not be acceptable if ponds are used for washing). <p>Chemoprophylaxis for malaria should be limited to pregnant women, expatriate staff, and special groups such as the army. The recommended prophylaxis for internationals is currently mefloquine. Chemoprophylaxis must be complemented by personal protection. Its mass use is not recommended because implementation and monitoring on a large scale are extremely difficult and because it can accelerate the development of drug resistance.</p> <p>Intermittent preventive treatment (IPT) at least twice during pregnancy (during 2nd and 3rd trimesters) is advisable for pregnant women living in areas where transmission is high. National policy has recently changed to IPT in pregnancy with sulfadoxine–pyrimethamine, once in the 2nd trimester and again in the 3rd, but this has yet to be introduced.</p> <p>Vigorous health education at community level is important to improve early treatment-seeking behaviour for fever cases during the transmission season.</p> <p>Cattle sponging with insecticides can be another approach, especially against vectors showing some degree of zoophily, such as <i>An. funestus</i>.</p> <p>Distribution of <i>Gambusia</i> fish as a means of vector control.</p>

Poor access to health services	Yes	Case fatality rates can be reduced by effective case management, including the administration of vitamin A supplements
Food shortages	No	However, disease is more severe among children with malnutrition and vitamin A deficiency.
Lack of safe water and poor sanitation	No	
Others	Yes	Low immunization coverage in the area of origin of the refugees or internally displaced people, and/or the host area.
Risk assessment conclusions		<p>In Sierra Leone, measles immunization activities have proved difficult: logistic and past security problems have made it difficult to establish and maintain an effective cold chain in many areas.</p> <p>Data have been often unavailable or incomplete because of the unstable situation in the country. Because of insufficient administrative and survey data during the 1980s and 1990s it is not possible to estimate a trend in coverage.</p> <p><u>MCV (measles-containing vaccine) coverage:</u></p> <p>2001: 53% (official country estimates); 37% (WHO/UNICEF estimates) 2000: 43% (official country estimates); 37% (WHO/UNICEF database) 1999: 40% (official country estimates) 1998: 68% (official country estimates) 1997: 28% (official country estimates) 1990: 75% (official country estimates) 1980: 36% (official country estimates)</p>

PREVENTION AND CONTROL MEASURES

Introduction	Sierra Leone has a routine immunization policy that requires a dose of single-antigen measles vaccine at 9 months of age (see Appendix 7). However, supplementary measles immunization campaigns may be required in order to reduce the risk of a measles outbreak.
Immunization in emergency and post-emergency phases	<p>Immunize the population at risk as soon as possible. The priority is to immunize children 6 months to 15 years old, regardless of vaccination status or history of disease. Expansion to older children is of lesser priority and should be based on evidence of high susceptibility among this age group.</p> <p>Children who are vaccinated against measles before 9 months of age must receive a second measles vaccination. This should be given as soon as possible after 9 months, with an interval of at least 1 month between doses.</p> <p>All children aged 6 months to 5 years should also receive prophylactic vitamin A supplementation. If evidence of clinical vitamin A deficiency in older age groups, treatment with vitamin A should be initiated as per WHO guidelines.</p> <p>To ensure safety of injection during immunization, auto-disable syringes and safety boxes are recommended. Safe disposal of used sharps should be ensured.</p>
Outbreak response	<p>Inform the health authorities if one or more suspected cases are identified</p> <p>Confirm the suspected outbreak, following WHO guidelines</p> <p>Investigate suspected case: check whether it fulfils the case definition, record date of onset, age and vaccination status</p> <p>Confirm the diagnosis: collect blood specimen from 3–5 initial reported cases</p> <p>Assess the extent of the outbreak and the population at risk.</p>

	<p>Implement outbreak response measures as follows:</p> <ul style="list-style-type: none"> – Give priority to proper case-management and immunization of groups at highest risk (e.g. children 6 months to 15 years¹) as soon as possible, even in areas not yet affected but where the outbreak is likely to spread. – Promote social mobilization of parents in order to ensure that previously unvaccinated children aged 6 months to 5 years are immunized. – The presence of several cases of measles in an emergency setting does not preclude a measles immunization campaign. Even among individuals who have already been exposed to, and are incubating, the natural virus, measles vaccine, if given <u>within 3 days</u> of exposure, may provide protection or modify the clinical severity of the illness. <p>Isolation is not indicated and children should not be withdrawn from feeding programmes.</p>
<p>Case management</p>	<p>For uncomplicated cases</p> <ul style="list-style-type: none"> – Give vitamin A immediately upon diagnosis and ensure the child receives a second dose the next day (can be given to parent to administer at home). – Advise the parent to treat the child at home (control fever and provide nutritional feeding). <p>For cases with non-severe eye, mouth or ear complications</p> <ul style="list-style-type: none"> – Children can be treated at home. – Give vitamin A immediately upon diagnosis and ensure that the child receives a second dose the next day (can be given to parent to administer at home). – If pus is draining from the eyes, clean eyes and treat with 1% tetracycline eye ointment. – If there are mouth ulcers, treat with gentian violet. – If pus is draining from the ear, clean ear discharge and treat with antibiotics for 5 days (amoxicillin 1st line, or co-trimoxazole 2nd line, as per national ARI policy and IMCI guidelines currently under development) – Treat malnutrition and diarrhoea, if present, with sufficient fluids and high-quality diet. <p>For cases with severe, complicated measles (any general danger signs² or clouding of cornea, deep or extensive mouth ulcers, pneumonia)</p> <ul style="list-style-type: none"> – Refer urgently to hospital. – Treat pneumonia with an appropriate antibiotic. – If there is clouding of the cornea or pus draining from the eye, clean eyes and apply 1% tetracycline eye ointment. – If the child has any eye signs indicating vitamin A deficiency (night blindness, Bitot spots, conjunctival and corneal dryness, corneal clouding or corneal ulceration), he or she should receive a third dose of vitamin A two weeks later.

¹ *This range can be reduced (e.g. 6 months to 12 years or 6 months to 5 years) if resources are limited.

² Inability to drink or breastfeed, vomiting everything, convulsions, lethargy or unconsciousness.

13. MENINGOCOCCAL DISEASE (meningitis and meningococcal septicaemia)

DESCRIPTION

Infectious agent	Bacterium: <i>Neisseria meningitidis</i> serogroups A, B, C, Y, W135
Case definition	<p><u>Clinical case definition</u></p> <p>An illness with sudden onset of fever (>38.5 °C rectal, >38.0 °C axillary) and one or more of the following:</p> <ul style="list-style-type: none"> – neck stiffness – altered consciousness – other meningeal sign or petechial or purpurial rash. <p>In patients under one year of age, suspect meningitis when fever is accompanied by bulging fontanelle.</p> <p><u>Laboratory criteria</u></p> <p>Positive CSF antigen detection, or Positive culture.</p> <p><u>Case classification</u></p> <p>Suspected: a case that meets the clinical case definition above. Probable: a suspected case as defined above and: turbid CSF (with or without positive Gram-stain), or ongoing epidemic and epidemiological link to a confirmed case. Confirmed: a suspected or probable case with laboratory confirmation.</p>
Mode of transmission	Direct contact with respiratory droplets.
Incubation	Incubation period varies between 2 and 10 days (most commonly 4 days).
Period of communicability	From the beginning of the symptoms till 24 hours after the institution of therapy, but the most important source of infection is asymptomatic carriers.

EPIDEMIOLOGY

Burden	No data available
Geographical distribution	Two districts (Kono in the east and Koinagugu in the north) are within the African meningitis belt.
Seasonality	Outbreaks tend to occur during the dry season (December to April)

Alert threshold¹	<p>Population >30 000: 5 cases per 100 000 inhabitants per week or a cluster of cases in an area.</p> <p>Population <30 000: 2 cases in 1 week or an increase in the number of cases compared with previous non-epidemic years.</p> <p>Intervention:</p> <ol style="list-style-type: none"> 1. Inform authorities 2. Investigate 3. Confirm 4. Treat cases 5. Strengthen surveillance 6. Prepare
Epidemic threshold	<p>Population > 30 000: 10 cases per 100 000 inhabitants per week if</p> <ul style="list-style-type: none"> – no epidemic for 3 years and vaccination coverage <80%; – alert threshold crossed early in the dry season. <p>15 cases per 100 000 inhabitants per week in other situations.</p> <p>Population < 30 000: The population should be vaccinated if:</p> <ul style="list-style-type: none"> – 5 cases occur in 1 week or – Doubling of the number of cases in a 3-week period or – for mass gatherings, refugees and displaced persons, 2 confirmed cases in 1 week. <p>Other situations should be studied on a case-by-case basis.</p> <p>Intervention:</p> <ol style="list-style-type: none"> 1. Mass vaccination 2. Distribute treatment to health centres; 3. Treat according to epidemic protocol; 4. Inform the public. <p>Caution: Current thresholds are established from data in meningitis belt countries and have not been validated in countries outside the belt.</p>
Recent epidemics in the country	<p>A major outbreak occurred between December 1994 and April 1995 in Kono district; 700 cases with 330 deaths were reported.</p> <p>Smaller-scale outbreaks have been reported in Kono and Koinagugu districts between 1997 and 2002.</p>

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Travel, migration and displacement facilitate the circulation of virulent strains within a country or from country to country.
Overcrowding	Yes	High density of susceptible people is an important risk factor for outbreaks. IDP/Refugee camps, crowding because of cattle or fishing-related activities, military camps and schools facilitate spread of the disease.

¹ Detecting meningococcal meningitis epidemics in highly-endemic African countries. *Weekly Epidemiological Record*, 2000, 38: 306–309.

	<ul style="list-style-type: none"> • Once diagnosis of meningococcal disease has been established: <ul style="list-style-type: none"> – Either <i>penicillin</i> or <i>ampicillin</i> is the drug of choice. – <i>Chloramphenicol</i> is a good and inexpensive alternative. – In Sierra Leone, the drugs of choice include <i>benzylpenicillin</i> or <i>chloramphenicol</i> injection or other antibiotics based on antimicrobial sensitivity tests. – The third-generation cephalosporins, <i>ceftriaxone</i> and <i>cefotaxime</i>, are excellent alternatives but are more expensive. – A 7-day course is still the general rule for the treatment of meningococcal disease beyond the neonatal period. – The single-dose long-acting (oily) form of chloramphenicol has also been shown to be effective and is preferred for mass interventions. <p><u>Epidemic conditions</u></p> <p>During epidemics of confirmed meningococcal disease, case management needs to be simplified to permit the health system to respond to rapidly increasing numbers of cases.</p> <ul style="list-style-type: none"> • Diagnosis: As the flood of patients could make the routine use of lumbar puncture to confirm meningitis impossible, every suspected case of meningitis should be considered and treated as one of meningococcal meningitis. • Treatment: Long-acting oily chloramphenicol (100 mg/kg up to 3 g in a single dose) IM is the drug of choice for all age groups, particularly in areas with limited health facilities. For those who do not improve rapidly, an additional dose of the same antimicrobial is recommended 48 hours later.
<p>Prevention</p>	<p><u>Non-epidemic conditions</u></p> <p>Vaccination: To prevent secondary cases around a sporadic case of meningococcal disease, vaccine can be used for close contacts of patients with meningococcal disease due to serogroup A, C, Y, or W135.</p> <p>Chemoprophylaxis: The aim of chemoprophylaxis is to prevent secondary cases by eliminating nasopharyngeal carriage. To be effective in preventing secondary cases, chemoprophylaxis must be initiated as soon as possible (i.e. not later than 48 hours after diagnosis of the case). Its use should be restricted to close contacts of a case, which are defined as:</p> <ul style="list-style-type: none"> – household members: persons sleeping in the same dwelling as the case; – institutional contacts: persons who share sleeping quarters (room-mates in boarding schools or orphanages; persons sharing barracks in military camps; – nursery school or child-care-centre contacts; children and teachers who share a classroom with the case; – others: persons who have had contact with the patient's oral secretions through kissing or sharing of food and beverages.

	<p><u>Epidemic conditions</u></p> <ul style="list-style-type: none"> • Vaccination: A mass vaccination campaign, if appropriately carried out, can halt an epidemic of meningococcal disease. For an accurate determination of causative serogroups, as many samples of CSF as possible (20–30) should be collected in the early stages of a meningococcal meningitis outbreak. <p>Laboratory diagnosis and confirmation of epidemic serogroups will guide the type of vaccine needed, either meningococcal polysaccharide bivalent A/C (if serogroup A or C is confirmed as the epidemic serogroup), or meningococcal polysaccharide tetravalent vaccine A/C/Y/W135 (if serogroup Y or W135 is confirmed). Vaccination should be targeted to areas crossing the epidemic threshold.</p> <ul style="list-style-type: none"> – Refugee camp population: Following confirmation (serogroup identified) of two cases, mass vaccination is recommended if the serogroup/s identified is/are included in either the bivalent (A/C) or tetravalent (A/C/Y/W135) vaccine. At risk populations (e.g. 2-30 years of age) should be given priority. – General population: If an outbreak is suspected, vaccination should be considered only after careful investigation (including confirmation and serogroup identification) and assessment of the population group at highest risk. <ul style="list-style-type: none"> • Chemoprophylaxis: Chemoprophylaxis of contacts of meningitis patients is NOT warranted during an epidemic for several reasons: vaccination is effective in rapidly controlling an outbreak, it is also cheaper and easier than to provide chemoprophylaxis to a large proportion of the population that can qualify as being in close contact with a meningococcal meningitis patient. In small clusters or outbreaks among closed populations (e.g. extended household, boarding schools), chemoprophylaxis may still be appropriate.
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14. ONCHOCERCIASIS (river blindness)

DESCRIPTION

Infectious agent	<i>Onchocerca volvulus</i> , a filarial worm belonging to the class Nematoda.
Case definition	<p><u>Clinical case definition</u></p> <p>In an endemic area, a person with fibrous nodules in subcutaneous tissues. These must be distinguished from lymph nodes or ganglia.</p> <p>Persons suffering from onchocerciasis may experience:</p> <ul style="list-style-type: none"> • Skin lesions: dermal changes are secondary to tissue reactions to the motile larvae as they migrate subcutaneously, or to their destruction in the skin. <ul style="list-style-type: none"> – Itching: the pruritus of onchocerciasis is the most severe and intractable that is known; in lightly infected persons, it may persist as the only symptom. – Rashes: the rash usually consists of many raised papules, are due to microabscess formation, and may disappear within a few days or may spread. Sowda, from the Arabic for black or dark, is an intensely pruritic eruption, usually limited to one limb and including oedema, hyperpigmented papules, and regional lymphadenopathy. It is common in Yemen and less frequent in Sudan. – Depigmentation of the skin: areas of depigmentation over the anterior shin, with islands of normally pigmented skin, commonly called “leopard skin”, are found in advanced dermatitis – Subcutaneous nodules: these are asymptomatic subcutaneous granulomas, 0.5–3.0 cm, resulting from a tissue reaction around adult worms. They occur most often over bony prominences: in Africa the nodules are often located over the hips and lower limbs. – Lymphadenopathy: frequently found in inguinal and femoral areas, lymphadenopathy can result in “hanging groin” (especially when associated with skin atrophy and loss of elasticity), and elephantiasis of the genitalia. • Eye lesions: ocular onchocerciasis is related to the presence of live or dead microfilariae in the eye. Involvement of all tissues of the eye has been described, and many changes in both anterior and posterior segments of the eye can occur. The more serious lesions lead to serious visual impairment including blindness. • General debilitation: onchocerciasis has also been associated with weight loss and musculoskeletal pain. <p><u>Laboratory criteria</u></p> <p>Presence of one or more of the following:</p> <ul style="list-style-type: none"> – microfilariae in skin snips taken from the iliac crest (Africa) or scapula (Americas) – adult worms in excised nodules – typical ocular manifestations, such as microfilariae in the cornea, the anterior chamber or the vitreous body, observed by slit-lamp – positive serology (especially for non-indigenous persons). <p><u>Case classification</u></p> <p>Suspected: A case that meets the clinical case definition. Probable: Not applicable Confirmed: A suspected case that is laboratory confirmed.</p>

<p>Mode of transmission</p>	<p>Vector-borne. Onchocercal microfilariae produced in one person are carried to another by the bite of infected female blackflies of the genus <i>Simulium</i> (<i>Simulium damnosum</i> species complex in west Africa). The blackfly lays its eggs in the water of fast-flowing rivers – thus the name “river blindness”. Adult blackflies emerge after 8–12 days and live for up to 4 weeks, during which they can cover hundreds of kilometres in flight.</p> <p>Microfilariae are ingested by a blackfly feeding on an infected person; these microfilariae then penetrate the thoracic muscles of the fly. Here a few of them develop into infective larvae and after several days migrate to the cephalic capsule, to be liberated into human skin during the bite wound of a subsequent blood meal. Infective larvae develop into adult parasites in the human body where adult forms of <i>O. volvulus</i> can live for 14–15 years and are often found encased in fibrous subcutaneous nodules. Each adult female produces millions of microfilariae that migrate under the skin and through the eyes, giving rise to a variety of dermal and ocular symptoms.</p> <p>Humans are the only reservoir. Other <i>Onchocerca</i> species found in animals cannot infect humans but may occur together with <i>O. volvulus</i> in the insect vector.</p>
<p>Incubation</p>	<p>Larvae take at least 6–12 months to become adult worms. Adult worms are usually innocuous, apart from the production of the subcutaneous nodules (these can develop as early as 1 year after infection).</p> <p>The main pathologic sequelae of <i>O. volvulus</i> infection are caused by the microfilariae in skin and ocular tissue, where they can be found after a period of 7–34 months. Usually, microfilariae are found in the skin only 1 year or more after the time of the infective bite.</p>
<p>Period of communicability</p>	<p>Human → blackfly</p> <p>Infected individuals can infect blackflies as long as living microfilariae occur in their skin. Microfilariae are continuously produced by adult female worms (about 700 per day), and can be found in the skin after a prepatent period of 7–34 months following introduction of infective larvae. They may persist for up to 2 years after the death of the adult worms.</p> <p>Blackfly → human</p> <p>Blackfly vectors become infective (i.e. able to transmit infective larvae) 7–9 days after the blood meal.</p>

EPIDEMIOLOGY

Burden	<p>Forest form (eastern area)</p> <p>A study conducted in 1988 in two forest districts of Sierra Leone found that onchocerciasis was hyperendemic, where 85% of persons were found to have at least one sign of onchocerciasis. The emergence of microfilariae from skin snips or the presence of nodules accounted for 96.5% of all persons positive for onchocerciasis. Clinical manifestations of onchocerciasis can differ from one region of the country to another region. In the forest area, low intensity of infection, mild skin disease and low blindness rate occur.</p> <p>Savanna form (northern area)</p> <p>A study conducted in 1988 in two savanna districts (Koinadogu and Kambia, Northern province) in northern Sierra Leone showed that the prevalence of onchocerciasis was 78% and 73% respectively). The savanna area (northern part of the country) is characterized by high intensity of infection, mild skin disease and high blindness rate.</p> <p>Mixed form (southern area)</p> <p>A mixture of the forest and the savanna form exists in the southern region of the country, with high intensity of infection, mild skin disease and high blindness rate, sometimes even higher than blindness rates from the savanna region.</p>
Geographical distribution	<p>Forest areas</p> <p>Sierra Leone is essentially a forested country, and woodland is widespread all over its territory, except the savanna belt in the north.</p> <p>Savanna areas</p> <p>Savanna areas consist of an indeterminate derived savanna-forest mosaic in the northeast (Koinadugu District), and woodland savanna in the northwest.</p>
Seasonality	Vector breeding and disease transmission are perennial in some location.
Recent epidemics	Not applicable.

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Migration can lead to the establishment of new foci.
Overcrowding	Yes	Increased risk of infectious bites.
Poor access to health services	Yes	Community-directed treatment with ivermectin (CDTI) is an effective tool for the control of transmission, although health infrastructure and access to health services are necessary.
Food shortages	No	
Lack of safe water and poor sanitation	No	
Others	Yes	Subsistence farming (rice, cassava and groundnuts), fishing, bathing and (in some areas) mining, were found to be the main activities associated with an increased risk of exposure of the population to blackfly bites in forest areas. Proximity to fast-flowing rivers.
Risk assessment conclusions	Sierra Leone was included in the OCP (Onchocerciasis Control Programme) in 1988. The disease is widely distributed. Given the security situation in Sierra Leone over the past decade, it is likely that the prevalence of onchocerciasis has as a result of difficulties in implementing and sustaining control programmes.	

PREVENTION AND CONTROL MEASURES

<p>Case management</p>	<p>Administration of ivermectin once a year over a period of at least 15–20 years reduces infection to insignificant levels and prevents the appearance of clinical manifestations. The recommended dosage is equivalent to 150 µg/kg body weight. In practice, dosage is according to height, using 1–4 tablets of a 3-mg formulation. Established clinical manifestations are also treated with ivermectin.</p> <p>Treatment with ivermectin is contraindicated in:</p> <ul style="list-style-type: none"> – children under 5 years of age – children under 15 kg in weight – children under 90 cm in height – pregnant women – lactating mothers of infants less than 1 week old – severely ill persons. <p>Note: Ivermectin should be used with extreme caution in areas co-endemic for <i>Loa loa</i>.</p>
<p>Epidemic control</p>	<p>Recrudescence of transmission may occur and can be managed by the mass administration of ivermectin where programmes can maintain good treatment coverage.</p>
<p>Prevention</p>	<p>The two main strategies for prevention and control of onchocerciasis in Africa are:</p> <p><u>Vector control</u></p> <p>Destruction of <i>Simulium</i> larvae by application of insecticides such as temephos (Abate®) through aerial spraying to breeding sites in fast-flowing rivers, in order to interrupt the cycle of disease transmission. Once the cycle has been interrupted for 14–15 years, the reservoir of adult worms dies out in the human population, thus eliminating the source of the disease. This was the basic strategy of the Onchocerciasis Control Programme (OCP) in west Africa, which concluded in 2002.</p> <p>In the west African savannah zone, onchocerciasis was a severely blinding disease. It was also responsible for the depopulation of fertile river valleys in OCP countries and was thus a major impediment to economic development. The large-scale vector control operations of the OCP, based on the aerial application of insecticides, were therefore considered economically justified.</p> <p>The African Programme for Onchocerciasis Control (APOC) was started in 1996, covering 19 African countries including Sierra Leone. APOC uses focal vector eradication as a control option. This implies that the whole focus is covered at once, resulting in the total eradication of the vector over a very short time scale. The programme aims to establish, within a period of 12 years, effective and self-sustainable community-based treatment with ivermectin throughout the endemic areas covered by the Programme, and to eliminate the disease by vector control in selected foci.</p>

	<p><u>Community-directed treatment with ivermectin (CDTI)</u></p> <p>CDTI involves the once-yearly administration of ivermectin (150 µg/kg of body weight). The introduction of ivermectin in 1987 provided a feasible chemotherapy regimen for large-scale treatment of onchocerciasis for the first time.</p> <p>Ivermectin is an effective microfilaricide that greatly reduces the numbers of skin microfilariae for up to a year.</p> <p>It alleviates symptoms (greatly reduces morbidity by preventing development of ocular lesions and blindness) and renders the infected person less infective for the vector by greatly reducing parasite transmission. This, however, does not kill the adult worm (which can survive for 14–15 years), and annual, long-term (15–20 years), large-scale treatment therefore needs to be continued.</p> <p>CDTI is the main strategy adopted by APOC, with distribution carried out on a house-to-house basis or at central meeting points in villages. Onchocerciasis remains a major cause of blindness in the 19 countries included this programme, but does not appear to be the cause of major depopulation of fertile lands. Partly for this reason, large-scale vector control operations, as carried out by the OCP, are not likely to be as cost-effective as they have been in the OCP area.</p> <p>APOC administers ivermectin to communities in high-risk areas as determined by rapid epidemiological mapping of onchocerciasis (REMO) and through use of geographical information systems (GIS). Continued annual distribution of ivermectin will control onchocerciasis to a point where it is no longer a public health problem or an impediment to economic development (Dadzie Y, Neira M, Hopkins D. Final report of the conference on the eradicability of onchocerciasis. <i>Filaria Journal</i>, 2003, 2(1):2).</p> <p>REMO – a tool developed by TDR (UNDP/World Bank/WHO Special Programme for Research and training in Tropical Diseases) in collaboration with the WHO Regional Office for Africa – makes it possible to assess quickly and cheaply which communities are at high risk of onchocerciasis and where they are located (TDR, 13th Programme Report).</p> <p>The main challenge facing ivermectin-based control is to develop and implement simple methods of ivermectin delivery that can be sustained by communities themselves.</p>
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15. PERTUSSIS (whooping cough)

DESCRIPTION

Infectious agent	<i>Bordetella pertussis</i> , the pertussis bacillus.
Case definition	<p><u>Clinical description</u></p> <p>The initial stage – the catarrhal stage – is characterized by the insidious onset of coryza (runny nose), sneezing, low-grade fever, and a mild, occasional cough, similar to the common cold. It has an insidious onset. An irritating cough that gradually becomes paroxysmal subsequently develops, usually within 1–2 weeks, and lasts for 1–2 months or more.</p> <p>The patient has bursts, or paroxysms, of numerous, rapid coughs, apparently due to difficulty in expelling thick mucus from the tracheobronchial tree. At the end of the paroxysm, a long inspiratory effort is usually accompanied by a characteristic whoop. In younger infants, periods of apnoea may follow the coughing spasms, and the patient may become cyanotic (turn blue).</p> <p>The disease generally lasts 4–8 weeks. In the convalescent stage, recovery is gradual. The cough becomes less paroxysmal. However, paroxysms often recur with subsequent respiratory infections for many months after the onset of pertussis. Fever is generally minimal throughout the course of pertussis.</p> <p>Complications: most commonly, pneumonia. Otitis, haemorrhages (subconjunctival petechiae and epistaxis), convulsions, encephalopathies and death occur more rarely.</p> <p>Complications are more frequent and severe in younger infants. In developing countries case fatality rates are estimated at 3.7% for children under 1 year and 1% for children aged 1–4 years. Older persons (adolescent and adults) and those partially protected by the vaccine may become infected with <i>B. pertussis</i>, but usually have milder disease.</p> <p><u>Clinical case definition</u></p> <p>A case diagnosed as pertussis by a physician, or A person with a cough lasting at least 2 weeks with at least one of the following symptoms:</p> <ul style="list-style-type: none"> – paroxysms (i.e. fits) of coughing – inspiratory “whooping” – post-tussive vomiting (i.e. vomiting immediately after coughing) without other apparent cause. <p><u>Laboratory criteria</u></p> <p>Isolation of <i>Bordetella pertussis</i>, or Detection of genomic sequences by polymerase chain reaction (PCR) Positive paired serology (acute and convalescent sera).</p> <p><u>Case classification</u></p> <p>Clinical case: A case that meets the clinical case definition. Confirmed case: A clinical case that is laboratory confirmed.</p>
Mode of transmission	<p>Primarily by direct contact with discharges from respiratory mucous membranes of infected persons via the airborne route (droplets). Humans are the only hosts.</p> <p>Infected older persons, even though they may have milder disease, may transmit the disease to other susceptible persons, including non-immunized or under-immunized infants. An adult is often the first case in a household with multiple pertussis cases.</p>

Incubation	Most commonly 7–10 days, with a range of 7–20 days.
Period of communicability	<p>Pertussis is highly communicable in the early catarrhal stage. Communicability gradually decreases after the onset of the paroxysmal cough stage (2 weeks). Thereafter, communicability decreases and becomes negligible in about 3 weeks, despite persisting spasmodic cough with whoop.</p> <p>Untreated patients may be contagious for up to 3 weeks after the onset of paroxysmal cough in the absence of treatment, or up to 5 days after onset of treatment.</p>

EPIDEMIOLOGY

Burden	<p>Number of cases reported:</p> <p>2001: data not available 1997: data not available 2000: no cases 1990: data not available 1999: data not available 1980: 1619 cases 1998: data not available</p>
Geographical distribution	Pertussis is an endemic disease common to children everywhere regardless of ethnicity, climate or geographical location.
Seasonality	Pertussis has no distinct seasonal pattern but activity may increase in the summer and fall.
Alert threshold	One case is sufficient to alert and must be investigated, especially if the case occurs in high-risk areas (low vaccination coverage).
Recent epidemics in the country	No data available

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Facilitates spread of <i>B. pertussis</i> .
Overcrowding	Yes	Crowded conditions facilitate transmission. The disease is usually introduced into a household by an older sibling or a parent.
Poor access to health services	Yes	No access to routine immunization services. Susceptibility of non-immunized individuals is universal, and vaccination is the mainstay of pertussis control. Low vaccination coverage (DTP3 coverage <80%) is a major risk factor for increased transmission.
Food shortages	No	
Lack of safe water and poor sanitation	No	
Others	Yes	Low DTP3 coverage (<80%).

<p>Risk assessment conclusions</p>	<p>Pertussis is a potential problem if introduced into overcrowded communities or IDP/refugee settings with many non-immunized infants and children; it is particularly lethal in those with underlying malnutrition and multiple enteric and respiratory infections.</p> <p>The exact burden of the disease is unknown, although it is likely that incidence rates have increased as in other countries where pertussis immunization rates have fallen.</p> <p>The disruption of vaccination activities in the country and the low DTP coverage rates (see below), lack of access to health services in many areas combined with overcrowded living conditions put the Sierra Leonean population at high risk for this disease. Yearly fluctuations in the number of reported cases reflect a weak surveillance system.</p> <p>DTP3 coverage:</p> <p>2001: 38% (official country estimates); 44% (WHO/UNICEF estimates) 2000: 24% (official country estimates); 44% (WHO/UNICEF survey database) 1999: NA 1998: 56% (official country estimates) 1997: 26% (official country estimates) 1990: 83% (official country estimates) 1980: 13% (official country estimates)</p> <p>To reduce disease risk, it is essential to improve access to health services and increasing vaccination coverage among the population. Surveillance activities must be strengthened in order to monitor vaccination coverage and pertussis incidence, identify high-risk areas and detect possible outbreaks as early as possible.</p>
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PREVENTION AND CONTROL MEASURES

<p>Case management</p>	<p>The drug of choice for the treatment of pertussis is erythromycin or erythromycin estolate, which should be administered for 7 days to all cases and close contacts of persons with pertussis, regardless of age and vaccination status, and for households where there is an infant under 1 year of age. Clarithromycin and azithromycin are also effective.</p> <p>Drug administration both modifies the course of illness (if initiated early), and eradicates the organism from secretions, thereby decreasing communicability, but does not reduce symptoms except when given during the catarrhal stage or early in the paroxysmal stage.</p> <p>Symptomatic treatment and supportive case-management are important.</p>
<p>Immunization</p>	<p>Vaccination is the most effective way to control pertussis. Active primary immunization against <i>B. pertussis</i> infection with the <i>whole-cell vaccine</i> (wP) is recommended in association with diphtheria and tetanus toxoids (DTP). No single-antigen pertussis vaccine is available.</p> <p>Although the use of <i>acellular vaccines</i> (aP) is less commonly associated with adverse reactions, their use is limited by considerations of price, and wP vaccines are the vaccines of choice for most countries, including Sierra Leone.</p> <p>In general, pertussis vaccine (wP) is not given to persons aged 7 years or older, since local reactions may be increased in older children and adults and the disease is less severe in older children.</p> <p>The efficacy of the vaccine in children who have received at least 3 doses is estimated to be over 80%. Protection is greater against severe disease and begins to wane after about 3 years.</p>

<p>Epidemic control</p>	<p>The highly contagious nature of the disease leads to large numbers of secondary cases among non-immune contacts. Prophylactic antibiotic treatment (with erythromycin) in the early incubation period may prevent disease, but is limited to selected individual cases because of the difficulties of early diagnosis, the costs involved and concerns related to the occurrence of drug resistance.</p> <p>Priority must be given to:</p> <ul style="list-style-type: none"> – protecting children under 1 year old and pregnant females in the last 3 weeks of pregnancy because of the risk of transmission to the newborn, and – stopping infection among household members, particularly if the household includes children aged under 1 year and pregnant women in the last 3 weeks of pregnancy. <p>The strategy relies on chemoprophylaxis of contacts within a maximum delay of 14 days following the first contact with the index case.</p> <p>Index cases must avoid contact with day-care centres, schools and other places where susceptible individuals are grouped, for up to 5 days after starting treatment, or for up to 3 weeks after onset of paroxysmal cough, or until the end of cough, whichever comes first.</p> <p>All contact cases must have their immunization status verified and brought up to date.</p>
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16. POLIOMYELITIS

DESCRIPTION

Infectious agent	Poliovirus (Enterovirus group): types 1, 2, 3.
Case definition	<p><u>Clinical description</u></p> <p>All 3 types of wild poliovirus may cause paralysis, although most infections (at least 95%) remain asymptomatic. Most symptomatic cases report a non-specific febrile illness lasting a few days, corresponding to the viraemic phase of the disease. In a few cases, fever can be followed by the abrupt onset of meningitic and neuromuscular symptoms, such as stiffness in the neck and pain in the limbs. Initial symptoms may also include fatigue, headaches, vomiting, constipation (or, less commonly, diarrhoea).</p> <p>In a very small percentage of cases ($\leq 1\%$ of infected susceptible persons), this is followed by gradual onset (2–4 days) of flaccid paralysis. Paralytic disease usually affects the lower limbs, and is typically asymmetric and more severe proximally. Bulbar (brainstem) paralysis may also occasionally occur, leading to respiratory muscle involvement and death unless artificial respiration can be applied. The mortality from paralytic poliomyelitis is 2–10%, mainly as a result of bulbar involvement and/or respiratory failure.</p> <p>Risk factors for paralytic disease are a large inoculum of virus, increasing age, pregnancy, recent tonsillectomy, strenuous exercise and intramuscular injections during the incubation period.</p> <p>After the acute illness there is often a degree of recovery of muscle function: 80% of eventual recovery occurs within 6 months, although recovery of muscle function may continue for up to 2 years.</p> <p>After many years of stable neurological impairment, new neuromuscular symptoms develop (weakness, pain and fatigue, post-polio syndrome) in 25–40% of patients.</p> <p><u>Clinical case definition</u></p> <p>Acute flaccid paralysis (AFP) in a child aged <15 years, including Guillain–Barré syndrome,* or Any paralytic illness in a person of any age when poliomyelitis is suspected.</p> <p>* For practical reasons, Guillain-Barré syndrome is considered as poliomyelitis until proven otherwise.</p> <p><u>Laboratory criteria</u></p> <p>Isolation of wild poliovirus in stool sample.</p> <p><u>Case classification</u></p> <p>Suspected: A case that meets the clinical case definition. Confirmed: AFP with laboratory-confirmed wild poliovirus in stool sample. Polio-compatible: AFP clinically compatible with poliomyelitis, but without adequate virological investigation.</p>
Mode of transmission	Highly communicable, primarily person-to-person via the faecal–oral route.
Incubation	The time between infection and onset of paralysis is 4–30 days.
Period of communicability	From 36 hours after infection, for 4–6 weeks.

EPIDEMIOLOGY

Burden	Number of cases reported: 2002: 0 wild-virus confirmed cases 1998: 0 wild-virus confirmed cases 2001: 0 wild-virus confirmed cases 1997: 0 wild-virus confirmed cases 2000: 0 wild-virus confirmed cases 1996: 0 wild-virus confirmed cases 1999: 2 wild-virus confirmed cases
Geographical distribution	Not known
Seasonality	Increased transmission during rainy season.
Alert threshold	Any AFP case must be notified and investigated.
Recent epidemics	None reported.

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Facilitates spread of virus.
Overcrowding	Yes	Very important in promoting transmission.
Poor access to health services	Yes	No access to routine immunization services. Risk of undetected poliovirus circulation.
Food shortages	No	
Lack of safe water and poor sanitation	Yes	As the virus is spread by faecal–oral route, lack of water and poor sanitation promote transmission.
Others	Yes	Unhygienic practices (e.g. not hand-washing after using toilet)
Risk assessment conclusions		<p>Polio tends to thrive in complex emergency settings where the above risk factors facilitate the spread of enteroviruses. No polio cases have been reported in Sierra Leone since 1999; however, surveillance and polio vaccination programmes have been irregular and disrupted, particularly in the north and east of the country because of many years of insecurity. There is a risk that polio cases may arise because of the low vaccination coverage rates shown below.</p> <p><u>OPV3 vaccination coverage:</u></p> <p>2001: 38% (official country estimates); 46% (WHO/UNICEF estimates) 2000: 26% (official country estimates); 46% (WHO/UNICEF survey database) 1999: 43% (official country estimates) 1998: 56% (official country estimates) 1997: 26% (official country estimates) 1990: 83% (official country estimates) 1980: 7% (official country estimates)</p>

PREVENTION AND CONTROL MEASURES

<p>Case management</p>	<p>Management of the acute phase of paralytic poliomyelitis is supportive and symptomatic:</p> <ul style="list-style-type: none"> – bed rest – close monitoring of respiration: respiratory support in case of respiratory failure or pooling of pharyngeal secretions – moist hot-packs for muscle pain and spasms – passive physical therapy to stimulate muscles and prevent contractures – anti-spasmodic drugs – frequent turning to prevent bedsores. <p>If hospitalization is required, the patient should be isolated.</p> <p>Disinfection of any discharge, faeces and soiled articles, and immediate reporting of further cases are essential.</p>
<p>Immunization</p>	<p>Two types of poliovirus vaccine are available:</p> <p><u>Oral poliovirus vaccine (OPV)</u></p> <p>OPV is an orally administered vaccine that includes live attenuated strains of all three virus types. It is easily administered by health workers or volunteers, induces a good humoral (antibody) and mucosal (intestinal) immune response and is four times cheaper than inactivated poliovirus vaccine (IPV). OPV is the only vaccine of choice for poliomyelitis eradication because it achieves much better mucosal immunity than IPV while limiting the dissemination of wild poliovirus.</p> <p><u>Inactivated poliovirus vaccine (IPV)</u></p> <p>IPV can be given by only intramuscular injection and requires trained health workers. It elicits an excellent antibody response, but only minimal intestinal mucosal response and is much more expensive than OPV.</p> <p><u>Immunization policy in Sierra Leone</u></p> <p>Sierra Leone has a routine immunization policy that requires four doses of OPV (see Appendix 7). However, supplementary immunization activities are also carried out in the country in order to maximize the immunization coverage: These consist of <i>national immunization days</i> (NIDs), <i>sub-NIDs</i> (mass campaigns similar to NIDs but covering smaller areas), and <i>mop-up campaigns</i>, during which two OPV doses are given at an interval of 1 month to all children under 5 years, preferably during the season of low transmission for enteroviruses (cooler season).</p> <p>In IDP/refugee camps, all children 0–59 months should be vaccinated on arrival.</p> <p>Any AFP case must be notified and investigated.</p>

Epidemic control	<p>In case of suspected outbreak, undertake:</p> <p><u>Investigation</u></p> <ul style="list-style-type: none">- Clinical and epidemiological investigation- Rapid virological investigation (2 stool samples within 14 days of onset of symptoms to be sent to a WHO-accredited laboratory) <p>Outbreak confirmation will be based on the isolation of wild poliovirus.</p> <p><u>Intervention</u></p> <p>A house-to-house mop-up campaign with OPV should be conducted in a wide geographical area (at least province involved and relevant neighbours) if no NIDs or sub-NIDs are planned to cover the area within the next 3 months. If NIDs or sub-NIDs are planned, focus should be set on ensuring that high-quality immunization activities are implemented in the area of the outbreak and adjacent districts.</p> <p>Surveillance should be enhanced through intensive monitoring of all reporting units, ensuring active surveillance and zero reporting, extensive retrospective record reviews, active case-finding in surrounding areas.</p>
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17. RABIES

DESCRIPTION

Infectious agent	Rabies virus, a Rhabdovirus of the genus <i>Lyssavirus</i> .
Case definition	<p><u>Clinical description</u></p> <p>Paresis or paralysis, delirium, convulsions.</p> <p>Without medical attention, death in about 6 days, usually due to respiratory paralysis.</p> <p><u>Clinical case definition</u></p> <p>An acute neurological syndrome (encephalitis), dominated by forms of hyperactivity (furious rabies) or paralytic syndrome (dumb rabies), that progresses toward coma and death, usually from respiratory failure, within 7–10 days after the first symptom. Bites or scratches from a suspected animal can usually be traced in the patient's medical history.</p> <p><u>Laboratory criteria</u></p> <p>One or more of the following:</p> <ul style="list-style-type: none"> • Detection of rabies viral antigens by direct fluorescent antibody (FA) in clinical specimens, preferably brain tissue (collected post mortem). • Detection by FA on skin or corneal smear (collected ante mortem). • FA positive after inoculation of brain tissue, saliva or CSF in cell culture, in mice or in suckling mice. • Detectable rabies-neutralizing antibody titre in the CSF of an unvaccinated person. • Identification of viral antigens by PCR on fixed tissue collected post mortem or in a clinical specimen (brain tissue or skin, saliva or urine). • Isolation of rabies virus from clinical specimens and confirmation of rabies viral antigens. <p><u>Case classification</u></p> <p>Human rabies</p> <p>Suspected: A case that is compatible with the clinical case definition.</p> <p>Probable: A suspected case plus history of contact with a suspected rabid animal.</p> <p>Confirmed: A suspected case that is laboratory confirmed.</p> <p>Human exposure to rabies</p> <p>Possibly exposed: A person who had close contact – usually a bite or a scratch – with a rabies-susceptible animal in (or originating from) a rabies-infected area.</p> <p>Exposed: A person who had close contact – usually a bite or a scratch – with a laboratory-confirmed rabid animal.</p>
Mode of transmission	<p>Usually through the bite of an infected mammalian species (dog, cat, fox, bat, etc.): bites or scratches introduce virus-laden saliva into the human body.</p> <p>No human-to-human transmission has been documented.</p>
Incubation	The incubation period usually ranges from 2 to 10 days but may be longer (up to 7 years).
Period of communicability	In dogs and cats, usually for 3–7 days before onset of clinical signs (rarely more than 4 days) and throughout the course of the disease. Longer periods of excretion before onset of clinical signs have been observed in other animals.

EPIDEMIOLOGY

Burden	No data available.
Geographical distribution	No data available.
Seasonality	No seasonality reported.
Alert threshold	One case in a susceptible animal species and/or human must lead to an alert.
Recent epidemics	No data available.

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	No	
Overcrowding	Yes	An infected animal has the opportunity to bite more people.
Poor access to health services	Yes	Prompt post-exposure administration of vaccine (plus immunoglobulin in case of heavy exposure) is the only way to prevent death of an infected person.
Food shortages	No	
Lack of safe water and poor sanitation	No	
Others	Yes	Availability of food sources for dogs and wild susceptible animals increases their number. Children of 5 to 15 years are the group at major risk.
Risk assessment conclusions		Rabies is endemic in most African countries. Overcrowded areas with poor environmental sanitation, especially urban settings where there are large concentrations of IDPs, are at greatest risk of exposure to infected animals. The risk of epidemics in humans is high if cases of rabies are reported in dogs or other susceptible animals in the same zone. The lack of information on incidence of rabies in many African countries including Sierra Leone is a result of limited and, or irregular surveillance. Surveillance of both human and animal rabies is essential to detect high-risk areas and outbreaks quickly.

PREVENTION AND CONTROL MEASURES

Case management	<p>There is no specific treatment for rabies, which is a fatal disease.</p> <p>The most effective way to prevent rabies after exposure is to wash and flush the wound or point of contact with soap and water, detergent or plain water, then apply ethanol or tincture or aqueous solution of iodine.</p> <p>Anti-rabies vaccine should be given as soon as possible for Category II and III exposures (see below), according to WHO recognized regimens. Anti-rabies immunoglobulin should be applied for Category III exposures only.</p> <p>Suturing should be postponed if possible; if it is necessary, immunoglobulin must be applied first. Where indicated, antitetanus treatment, antimicrobials and drugs should be administered to control infections other than rabies.</p> <p>Recommended treatments according to type of contact with suspect animal</p> <table border="1"> <thead> <tr> <th data-bbox="516 604 630 632">Category</th> <th data-bbox="659 604 902 659">Type of contact with suspected animal</th> <th data-bbox="1049 604 1349 632">Recommended treatment</th> </tr> </thead> <tbody> <tr> <td data-bbox="516 680 526 707">I</td> <td data-bbox="659 680 997 747">Touching or feeding an animal Licks on intact skin</td> <td data-bbox="1049 680 1386 735">None, if reliable case history is available</td> </tr> <tr> <td data-bbox="516 768 526 795">II</td> <td data-bbox="659 768 980 911">Nibbling of uncovered skin Minor scratches or abrasions without bleeding Licks on broken skin</td> <td data-bbox="1049 768 1414 911">Administer vaccine immediately, and stop if 10-day observation or laboratory techniques confirm suspect animal to be rabies-negative</td> </tr> <tr> <td data-bbox="516 932 526 959">III</td> <td data-bbox="659 932 992 1062">Single or multiple transdermal bites or scratches Contamination of mucous membrane with saliva</td> <td data-bbox="1049 932 1425 1054">Administer rabies immunoglobulin and vaccine immediately and stop if suspect animal confirmed to be rabies-negative</td> </tr> </tbody> </table> <p>If a person develops the disease, death is inevitable.</p> <p>Universal barrier-nursing practices are necessary for patients.</p>	Category	Type of contact with suspected animal	Recommended treatment	I	Touching or feeding an animal Licks on intact skin	None, if reliable case history is available	II	Nibbling of uncovered skin Minor scratches or abrasions without bleeding Licks on broken skin	Administer vaccine immediately, and stop if 10-day observation or laboratory techniques confirm suspect animal to be rabies-negative	III	Single or multiple transdermal bites or scratches Contamination of mucous membrane with saliva	Administer rabies immunoglobulin and vaccine immediately and stop if suspect animal confirmed to be rabies-negative
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III	Single or multiple transdermal bites or scratches Contamination of mucous membrane with saliva	Administer rabies immunoglobulin and vaccine immediately and stop if suspect animal confirmed to be rabies-negative											
Epidemic control	<p>Immediate notification if one or more suspected cases are identified.</p> <p>Confirm the outbreak, following WHO guidelines.</p> <p>Confirm diagnosis and ensure prompt management.</p>												
Prevention	<p>WHO promotes human rabies prevention through:</p> <ul style="list-style-type: none"> – well-targeted post-exposure treatment using modern vaccine types and, when appropriate, anti-rabies immunoglobulin; – advocating increased availability of modern rabies vaccine to susceptible populations; – elimination of dog rabies through mass vaccination of dogs and dog population management. 												
Immunization	<p>Preventive mass vaccination in humans is generally not recommended but can be considered under certain circumstances for the age group 5–15 years.</p>												

18. SCHISTOSOMIASIS (bilharziasis)

DESCRIPTION

Infectious agent	<p>Helminths: <i>Schistosoma haematobium</i> (causes urinary schistosomiasis) and <i>Schistosoma mansoni</i> (causes intestinal schistosomiasis), blood fluke worms belonging to the class Trematoda.</p> <p>Other <i>Schistosoma</i> species are not present in Sierra Leone.</p>
Case definition	<p><u>Urinary schistosomiasis</u></p> <p>1. Endemic areas (moderate or high prevalence) Suspected: not applicable. Probable: not applicable. Confirmed: a person with:</p> <ul style="list-style-type: none"> – visible haematuria or – positive reagent strip for haematuria or – <i>S. haematobium</i> eggs in urine (microscopy). <p>2. Non-endemic areas and areas of low prevalence Suspected: a person with:</p> <ul style="list-style-type: none"> – visible haematuria or – positive reagent strip for haematuria and – possible contact with infective water. <p>Probable: not applicable. Confirmed: a person with <i>S. haematobium</i> eggs in urine (microscopy).</p> <p><u>Intestinal schistosomiasis</u></p> <p>1. Endemic areas (moderate or high prevalence) Suspected: a person with nonspecific abdominal symptoms, blood in stool, hepato(spleno)megaly. Probable: a person who meets the criteria for presumptive treatment, according to the locally applicable diagnostic algorithms. Confirmed: a person with eggs of <i>S. mansoni</i> in stools (microscopy).</p> <p>2. Non-endemic areas and areas of low prevalence Suspected: a person with nonspecific abdominal symptoms, blood in stool, hepatosplenomegaly and possibly contact with infective water. Probable: not applicable. Confirmed: a person with eggs of <i>S. mansoni</i> in stools (microscopy).</p>
Mode of transmission	<p>Infection is acquired when schistosomes enter the body through the skin during contact with infested surface water, mainly among people engaged in agriculture and fishing.</p> <p>Parasite eggs are discharged in urine (if <i>S. haematobium</i>) or faeces (if <i>S. mansoni</i>) of patients with chronic schistosomiasis into a body of fresh water.</p> <p>In the water, the eggs liberate the larvae (miracidia) that penetrate suitable freshwater snails (<i>Bulinus globosus</i> and <i>Biomphalaria pfeifferi</i>), the intermediate host, to develop into larval worms (cercariae). The cercariae or schistosomes emerge from the snail and penetrate human skin, usually while the person is swimming, working or wading in water.</p> <p>In Sierra Leone, the snail intermediate hosts thrive in regions with the least shortage of rainfall in the dry season. Because of the relative salinity of surface water near sea level, the snail hosts are not found in the coastal plains. <i>Bulinus globosus</i> (host of <i>S. haematobium</i>) is very frequently found in the interior of Sierra Leone, while <i>Biomphalaria pfeifferi</i> (host of <i>S. mansoni</i>) is present in only a few sites in the north and east of Sierra Leone. Generally, <i>Bulinus globosus</i> is found at altitudes >100 metres, and <i>Biomphalaria pfeifferi</i> at altitudes >250 metres.</p>
Incubation	<p>Within 4 days: localized dermatitis at the site of cercarial penetration.</p> <p>Within 2–8 weeks: acute febrile reaction (Katayama fever; almost completely</p>

	absent in <i>S. haematobium</i> infection). From 3 months to several years: chronic illness manifestations
Period of communicability	As long as eggs are discharged by patients. This may be from 10–12 weeks to more than 10 years after infection.

EPIDEMIOLOGY

Burden	<p><i>S. haematobium</i>: prevalence of infection in selected areas and periods (where information available):</p> <ul style="list-style-type: none"> – Njala (Moyamba district): 1.1% (1994) – several locations in Moyamba district: 0.6% (1991) – Sefadu (Kono district): 75% (1970) – Mobai (Kailahun district): 60% (1970) – Boajibu (Kenema district): 61.5% (1970) – Giema (Kailahun district): 51.6% (1970) – Neama and Joru (Kailahun district): 54.5% (1970) – Bo (Bo district): 64.3% (1970) – Mandu (Kailahun district): 60% (1970) – Magburaka (Tonkolili district): 15.3% (1970) – Makali (Tonkolili district): 20% (1970) – Mamansu (Tonkolili district): 38.1% (1970) – Masingbi (Tonkolili district): 40% (1970) – Yele (Tonkolili district): 11.8% (1970) – Kabala (Koinadugu district): 22.7% (1970) – Kamakwie (Bombali district): 2.3% (1970) <p><i>S. mansoni</i>: prevalence of infection in selected areas and periods (where information available):</p> <ul style="list-style-type: none"> – Njala (Moyamba district): 0.8% (1994) – several locations in Moyamba district: 0.3% (1991) – Kabala (Koinadugu district): 40–45% among school-age children (1970)
Geographical distribution	<p>The foci of transmission of urinary schistosomiasis (<i>S. haematobium</i>) have been reported to be more than 100 km from the Atlantic coast. Eastern districts have the greatest number of affected localities, with the Moa river valley, less than 50 km from the border with Liberia, apparently the most extensively affected. Some foci are located in the northern area of Sierra Leone, with Tonkolili district the worst affected.</p> <p>Intestinal schistosomiasis (<i>S. mansoni</i>) is less widely distributed than urinary schistosomiasis; even where the disease is present, its prevalence is usually lower than that of <i>S. haematobium</i>.</p> <p>Prevalence of intestinal schistosomiasis tends to decrease from the north-east to the south-east of the country, while urinary schistosomiasis tends to increase. It has also been observed that, in each locality where <i>S. mansoni</i> was predominant, there was also a group of people infected with <i>S. haematobium</i> (and, usually, some people who were infected with both parasites). Conversely, <i>S. mansoni</i> was not found in many localities where <i>S. haematobium</i> was predominant.</p>
Seasonality	Peak transmission of both forms of schistosomiasis occurs at the beginning of the dry season, when water levels are falling and numerous marshes and ponds form in place of streams. Dry periods tend to increase transmission of the disease as a result of the higher cercarial densities in bodies of water.
Recent epidemics	Schistosomiasis is usually an endemic disease, with little likelihood of rapid changes in incidence. Surveys may identify areas of particularly high endemicity where mass treatment will be warranted.

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Population displacement can introduce schistosomiasis into previously non-endemic areas. It can also introduce the intestinal form into areas endemic for the urinary form (and vice versa).
Overcrowding	Yes	Higher population densities increase the chance of snails being penetrated and colonized by miracidia.
Poor access to health services	Yes	Regular treatment of cases has proved effective in reducing or preventing introduction of <i>Schistosoma</i> spp. into <i>Schistosoma</i> -free areas.
Food shortages	No	
Lack of safe water and poor sanitation	Yes	Use of surface water infested by cercariae and contamination of water by urination/defecation are essential for transmission of schistosomiasis.
Others	No	
Risk assessment conclusions		<p>Both <i>S. haematobium</i> and <i>S. mansoni</i> are endemic in Sierra Leone. Urinary schistosomiasis was first reported in the country in 1909, and intestinal schistosomiasis in 1934.</p> <p>Although rice cultivation is widespread, the rice fields do not appear to be ideal habitats for the snail intermediate hosts, because of low salinity of the land and the fact that, at certain times of the year, the water in the paddies is at a temperature of more than 40 °C, well above that tolerated by <i>Bulinus globosus</i> and <i>Biomphalaria pfeifferi</i>.</p> <p>Schistosomiasis is endemic in the diamond-mining areas along the Moa and Dewa rivers. The reservoirs and canals created by the large quantities of water used in the processing of ores, and the constant population migration at the extraction sites contribute to the high risk of transmission.</p> <p>Control of schistosomiasis should be a priority given the effect of this disease both on the general health of infected individuals and in increasing the severity of concomitant infections. However, no large-scale programmes are currently implemented in Sierra Leone. Schistosomiasis control should be built into case-management algorithms in primary health care services and into any health packages delivered through schools.</p>

PREVENTION AND CONTROL MEASURES

<p>Case management</p>	<p>Praziquantel is the drug of choice for all schistosomiasis parasites. A single oral dose of 40 mg/kg is generally sufficient to produce cure rates of 80–90% and dramatic reductions in the average number of eggs excreted.</p> <p>Praziquantel treatment for 1 person requires, on average, three tablets of 600 mg in one dose. The cost of a tablet is now less than US\$ 0.10, bringing the total drug cost of a treatment to about US\$ 0.30.</p> <p>A dose pole (for calculating dosage according to height) is available to facilitate the delivery of praziquantel in schools or for community-based delivery.</p>								
<p>Prevention</p>	<ul style="list-style-type: none"> • Community diagnosis (through primary school surveys) and regular treatment of individuals according to community prevalence categories (see below). • Creation of alternative, safe water sources to reduce contact with infective water. • Proper disposal of faeces and urine to prevent viable eggs from reaching bodies of water containing snail hosts. • Health education to promote early care-seeking behaviour, use of safe water (if available) and proper disposal of excreta. • Reduction of snail habitats and snail contact (through irrigation and agriculture practices), environmental management. • Treatment of snail-breeding sites with molluscicide (if costs permit). <table border="0" style="width: 100%;"> <thead> <tr> <th style="text-align: left;">Community category</th> <th style="text-align: left;">Prevalence</th> </tr> </thead> <tbody> <tr> <td style="vertical-align: top;">I (High prevalence)</td> <td> <p>≥30% visible haematuria (<i>S. haematobium</i>, by questionnaire)</p> <p>or</p> <p>≥50% infected (<i>S. mansoni</i>, <i>S. haematobium</i>, by parasitological methods)</p> </td> </tr> <tr> <td style="vertical-align: top;">II (Moderate prevalence)</td> <td> <p><30% visible haematuria (<i>S. haematobium</i>, by questionnaire)</p> <p>or</p> <p>≥10% but <50% infected (<i>S. mansoni</i>, <i>S. haematobium</i>, by parasitological methods)</p> </td> </tr> <tr> <td style="vertical-align: top;">III (Low prevalence)</td> <td> <p><10% infected (<i>S. haematobium</i>, <i>S. mansoni</i>, by parasitological methods).</p> </td> </tr> </tbody> </table>	Community category	Prevalence	I (High prevalence)	<p>≥30% visible haematuria (<i>S. haematobium</i>, by questionnaire)</p> <p>or</p> <p>≥50% infected (<i>S. mansoni</i>, <i>S. haematobium</i>, by parasitological methods)</p>	II (Moderate prevalence)	<p><30% visible haematuria (<i>S. haematobium</i>, by questionnaire)</p> <p>or</p> <p>≥10% but <50% infected (<i>S. mansoni</i>, <i>S. haematobium</i>, by parasitological methods)</p>	III (Low prevalence)	<p><10% infected (<i>S. haematobium</i>, <i>S. mansoni</i>, by parasitological methods).</p>
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III (Low prevalence)	<p><10% infected (<i>S. haematobium</i>, <i>S. mansoni</i>, by parasitological methods).</p>								

	<p><u>Category 1</u></p> <p>Intervention in schools (enrolled and non-enrolled children): Targeted treatment of school-age children, once a year.</p> <p>Health services and community-based intervention: Access to praziquantel for passive case-treatment + community-directed treatment for high-risk groups* recommended.</p> <p>*Such groups include pre-school children, school-age children, pregnant women and workers with occupations involving contact with fres water.</p> <p><u>Category 2</u></p> <p>Intervention in schools (enrolled and non-enrolled children): Targeted treatment of school-age children, once every 2 years.</p> <p>Health services and community-based intervention: Access to praziquantel for passive case treatment.</p> <p><u>Category 3</u></p> <p>Intervention in schools (enrolled and non-enrolled children): Targeted treatment of school-age children twice during primary schooling (once on entry, again on leaving). Community-based intervention: Access to praziquantel for passive case treatment.</p> <p>For the definition of classes of intensity and further information, see: <i>Prevention and control of schistosomiasis and soil-transmitted helminthiasis. Report of a WHO Expert Committee</i>. Geneva, WHO, 2002 (WHO Technical Report Series, No. 912).</p>
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19. SOIL-TRANSMITTED HELMINTHIASES (ascariasis, hookworm infection, trichuriasis)

DESCRIPTION

Infectious agent	Helminths: <i>Ascaris lumbricoides</i> , hookworm (<i>N. americanus</i>), <i>Trichuris trichiura</i>
Case definition	<p>Ascariasis Suspected: abdominal or respiratory symptoms and history of passing worms. Confirmed: suspected case and passage of <i>A. lumbricoides</i> (anus, mouth, and nose), or presence of <i>A. lumbricoides</i> eggs in stools (microscope examination).</p> <p>Hookworm infection Suspected: severe anaemia for which there is no other obvious cause. Confirmed: suspected case and presence of hookworm eggs in stools (microscope examination).</p> <p>Trichuriasis Suspected: bloody, mucoid stools. Confirmed: suspected case and presence of <i>T. trichiura</i> eggs in stools.</p>
Mode of transmission	<p><i>A. lumbricoides</i> and <i>T. trichiura</i>: ingestion of eggs, mainly as a contaminant of food.</p> <p>Hookworm: active penetration of skin by larvae in the soil.</p>
Incubation	<p><i>A. lumbricoides</i>: 4–8 weeks.</p> <p>Hookworm: a few weeks to many months.</p> <p><i>T. trichiura</i>: nonspecific.</p>
Period of communicability	<p><i>A. lumbricoides</i>: eggs appear in the faeces 45–75 days after ingestion and become infective in soil after 2–3 weeks. They can remain viable in soil for years. Infected people can contaminate soil as long as mature fertilized female worms live in the intestine (lifespan of adult worms can be 12–24 months).</p> <p>Hookworm: eggs appear in the faeces 6–7 weeks after infection. As larvae they become infective in soil after 7–10 days and can remain infective for several weeks. Infected people can contaminate soil for several years.</p> <p><i>T. trichiura</i>: eggs appear in the faeces 70–90 days after ingestion and become infective in soil after 10–14 days. Infected people can contaminate soil for several years.</p>

EPIDEMIOLOGY

Burden	Prevalence rates of the soil-transmitted helminthiases in different areas:			
		<i>A. lumbricoides</i>	Hookworm	<i>T. trichiura</i>
	Sierra Leone 1997 (various sites)	47%	21%	40%
	Njala schoolchildren 1994 (southern)	33.3%	10.4%	14.6%
	Moyamba district 1991 (southern)	13.7%	12.1%	9.3%
	Two villages near Bo town 1990 (southern)	39%	90%	15%
Geographical distribution	Soil-transmitted helminths are widespread in the country.			
Seasonality	No data available.			
Recent epidemics	Soil-transmitted helminthiases are usually endemic diseases, with little likelihood of rapid changes in incidence. Surveys may identify areas of particularly high endemicity where mass treatment will be warranted.			

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Strictly linked to inadequate sanitation facilities. Not a risk factor if people remain in the same place for a period shorter than that needed for eggs to be discharged by an infected patient and become infective themselves (at least 45–50 days).
Overcrowding	Yes	Linked to the number of people defecating and to unsafe disposal of faeces.
Poor access to health services	Yes	No treatment provided.
Food shortages	No	
Lack of safe water and poor sanitation	Yes	The number of people relative to available sanitation facilities is the most important risk factor.
Others	No	
Risk assessment conclusions		<p>Soil-transmitted helminths can be controlled with low-cost, highly effective interventions that can dramatically increase the quality of life of affected populations.</p> <p>No large-scale programmes for the control of soli-transmitted helminths are currently implemented in Sierra Leone.</p> <p>Control of helminthic infestations can play a major role in the reduction of the communicable disease burden borne by populations in complex emergency countries. Moreover, their simplicity and feasibility mean that control measures for intestinal helminth infections can represent a starting point for the reconstruction of health care systems in such countries.</p>

PREVENTION AND CONTROL MEASURES

Case management	<p>All soil-transmitted helminths (STH) compete with the host for nutrients, causing malabsorption of fats, proteins, carbohydrates and vitamins, and directly contributing to malnutrition. They can also cause growth retardation.</p> <p>A. lumbricoides infestation exacerbates vitamin A deficiency. Thus elimination of ascarids may result in rapid clinical improvement in night blindness and dryness around the eye. Infection from measles in a patient already infected with <i>A. lumbricoides</i> can result in a very severe disease.</p> <p>Hookworm infestation is strongly associated with chronic anaemia. Significant inverse correlations between intensity of worm infestation and haemoglobin level have been demonstrated.</p> <p>Heavy <i>T. trichiura</i> infection can cause diarrhoea and severe malabsorption.</p> <p>STH can be controlled with very cheap interventions. In a school distribution campaign the average cost of treatment (including drugs, distribution, and monitoring activities) is approximately US\$ 0.05 per child.</p> <p>For treatment, the following four drugs are recommended by WHO:</p> <ul style="list-style-type: none"> – 400 mg albendazole, or – 2.5 mg/kg levamisole, or – 500 mg mebendazole, or – 10 mg/kg pyrantel (less commonly used because it is less easy to administer). <p>Note 1: These drugs must not be given during the first trimester of pregnancy.</p> <p>Note 2: Where mass treatment with albendazole for filariasis is envisaged, chemotherapy of intestinal helminths will take place as part of the antifilarial chemoprophylaxis.</p> <p>Note 3: Iron supplementation is also recommended if required.</p>												
Prevention and control	<ul style="list-style-type: none"> • Personal hygiene and handwashing, appropriate disposal of faeces, hand-washing, and clean food • Improvements in sanitation standards (see Safe water and sanitation) • Community diagnosis (through primary school surveys) and community-wide treatment regimen for STH according to the following categories: 												
	<table border="1"> <thead> <tr> <th data-bbox="513 1192 829 1255">Community category</th> <th data-bbox="829 1192 1089 1255">Prevalence of any infection</th> <th data-bbox="1089 1192 1422 1287">% of moderate- to heavy-intensity infections</th> </tr> </thead> <tbody> <tr> <td data-bbox="513 1287 829 1392">I High prevalence, high intensity</td> <td data-bbox="829 1287 1089 1392">≥70%</td> <td data-bbox="1089 1287 1422 1392">≥10%</td> </tr> <tr> <td data-bbox="513 1392 829 1497">II High prevalence, low intensity</td> <td data-bbox="829 1392 1089 1497">≥50% but <70%</td> <td data-bbox="1089 1392 1422 1497"><10%</td> </tr> <tr> <td data-bbox="513 1497 829 1602">III Low prevalence, low intensity</td> <td data-bbox="829 1497 1089 1602"><50%</td> <td data-bbox="1089 1497 1422 1602"><10%</td> </tr> </tbody> </table>	Community category	Prevalence of any infection	% of moderate- to heavy-intensity infections	I High prevalence, high intensity	≥70%	≥10%	II High prevalence, low intensity	≥50% but <70%	<10%	III Low prevalence, low intensity	<50%	<10%
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I High prevalence, high intensity	≥70%	≥10%											
II High prevalence, low intensity	≥50% but <70%	<10%											
III Low prevalence, low intensity	<50%	<10%											

	<p><u>Category 1</u></p> <p>Intervention in schools (enrolled and non-enrolled children): Targeted treatment of school-age children, 2–3 times a year.</p> <p>Health services and community-based intervention: Systematic treatment of pre-school children and women of childbearing age in mother and child health programmes.</p> <p><u>Category 2</u></p> <p>Intervention in schools (enrolled and non-enrolled children): Targeted treatment of school-age children, once a year.</p> <p>Health services and community-based intervention: Systematic treatment of pre-school children and women of childbearing age in mother and child health programmes.</p> <p><u>Category 3</u></p> <p>Intervention in schools (enrolled and non-enrolled children): Selective treatment.</p> <p>Community based intervention: Selective treatment.</p> <p>For the definition of classes of intensity and further information, see: <i>Prevention and control of schistosomiasis and soil-transmitted helminthiasis. Report of a WHO Expert Committee</i>. Geneva, WHO, 2002 (WHO Technical Report Series, No. 912).</p> <p><u>In case of suspected or confirmed hookworm infection:</u></p> <ul style="list-style-type: none">– In highly endemic areas, wear shoes.– Consider drug treatment and iron supplementation in pregnancy.
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20. TUBERCULOSIS

DESCRIPTION

<p>Infectious agent</p>	<p>Bacterium: <i>Mycobacterium tuberculosis</i>. The <i>Mycobacterium tuberculosis</i> complex includes <i>M. tuberculosis</i> and <i>M. africanum</i> primarily from humans, and <i>M. bovis</i> primarily from cattle.</p>
<p>Case definition</p>	<p><u>Clinical description</u></p> <p>The most important symptoms in the selection of TB suspects in adults (over 15 years of age) are:</p> <ul style="list-style-type: none"> – productive cough for more than 3 weeks, and/or – haemoptysis and – significant weight loss. <p>Patients with TB may also have other symptoms (which are more common, but less suggestive) such as:</p> <ul style="list-style-type: none"> – chest pain – breathlessness – fever/night sweats – tiredness, and – loss of appetite. <p>In refugee and displaced populations, it is unusual to have ready access to X-ray facilities. It is the priority of the health services to detect the sources of infection by sputum microscopy, and cure them.</p> <p><u>Clinical case definition</u></p> <p>Tuberculosis suspect: any person who presents with symptoms or signs suggestive of TB, in particular cough of long duration (more than 3 weeks)</p> <p>Case of tuberculosis: a patient in whom TB has been bacteriologically confirmed or diagnosed by a clinician.</p> <p>Note: Any person given treatment for tuberculosis should be recorded as a case. Incomplete "trial" tuberculosis treatment should not be given as a method for diagnosis.</p> <p>Definite case of tuberculosis: a patient with positive culture for the <i>Mycobacterium tuberculosis</i> complex. (In countries where culture is not routinely available, a patient with two sputum smears positive for acid-fast bacilli (AFB) is also considered a "definite" case.)</p> <p><u>Laboratory criteria for diagnosis</u></p> <p>Each TB suspect should have 3 sputum samples examined by light binocular microscopy for AFB.</p> <p>The chances of finding TB organisms are greater with three sputum samples than with one or two samples. Secretions build up in the airways overnight, so that an early-morning sputum sample is more likely to contain the TB organism than a sample later in the day. In practice, a suspect provides sputum samples in the following manner:</p> <p>Day 1</p> <p>Sample 1 – Person suspected of TB provides an “on the spot” sample under supervision on presentation to the health facility. He/she is given a sputum container to take home for an early morning sample the following morning.</p> <p>Day 2</p> <p>Sample 2 – Person suspected of TB brings an early-morning sputum sample collected just after waking up.</p> <p>Sample 3 – Person suspected of TB provides another “on the spot” sample.</p> <p>If at least two sputum smears are positive</p> <p>Smears should be stained using the Ziehl-Neelsen method. Any TB suspect with</p>

	<p>two positive smears is a smear-positive TB patient, who must then be registered and started on anti-TB treatment.</p> <p>If only one initial sputum smear is positive</p> <p>A suggestive X-ray showing active pulmonary TB interpreted by an experienced medical officer may lead to a diagnosis of smear-positive TB. AFB microscopy may be repeated and, if at least one smear is again positive with compatible X-ray, the patient should be considered a smear-positive TB patient. In the absence of X-ray, one sputum smear with positive culture for <i>M. tuberculosis</i> is also classified as sputum-positive TB.</p> <p>If all three sputum smears are negative</p> <p>If the initial three smears are negative, but pulmonary TB is still suspected because of persistent symptoms, the suspect should be treated for acute respiratory infection with broad-spectrum antibiotics (e.g. amoxicillin or cotrimoxazole, but not rifampicin or any anti-TB drug) for at least one week. If there is no improvement, further sputum samples must be examined 2 weeks after the first sputum examination.</p> <p>Specific anti-TB medication should not be started unless the presence of AFB is confirmed in at least one sample (classed as smear-positive TB). 65–80% of all pulmonary TB cases are expected to be confirmed by positive sputum smear examination. X-ray lesions compatible with active TB should encourage further sputum examination if the three sputum smear examinations were negative. X-ray itself is not diagnosis tool for pulmonary TB.</p> <p>In <i>some</i> circumstances, a compatible X-ray together with symptoms consistent with TB will lead to the diagnosis of pulmonary TB in smear-negative cases. Thus, if all three samples are again negative after the trial of antibiotics, either a compatible X-ray interpreted by an experienced physician or, in the absence of X-ray facilities, the experienced physician's judgement alone will decide whether someone is categorised as having TB (classed as smear-negative TB).</p> <p>Additional cases of TB may be found among close contacts of known smear-positive cases, either family members or persons sleeping in the same shelter. Symptomatic contacts should be screened, using the procedures described above.</p> <p><u>TB in HIV-positive patients</u></p> <p>HIV-positive patients are more susceptible to TB infection, and HIV in a TB patient, is a potent cause of progression of tuberculosis infection to disease.</p> <p>The principles of TB control are the same even when there are many HIV/TB patients. In HIV-infected patients, pulmonary TB is still the commonest form of TB. The clinical presentation of TB depends on the degree of immunosuppression.</p> <p>Early in HIV infection, when immunity is good, the signs of TB are similar to those in an individual without HIV infection. As HIV infection progresses and immunity declines, the risk of TB dissemination increases. TB meningitis, miliary TB, and widespread TB lymphadenopathy occur.</p> <p>It is important to look systematically for signs or symptoms of TB in HIV-positive patients, start treatment without delay based on clinical, bacteriological and, in some circumstances, radiological evidence.</p>
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<p>Diagnostic criteria for classification of TB</p>	<p><u>Pulmonary tuberculosis (PTB)</u></p> <p>Pulmonary TB refers to disease involving the lung parenchyma. Tuberculous intrathoracic lymphadenopathy (mediastinal and/or hilar) or tuberculous pleural effusion, without radiographic abnormalities in the lungs, therefore constitutes a case of extrapulmonary TB. A patient with both pulmonary and extrapulmonary TB should be classified as a case of pulmonary TB.</p> <ul style="list-style-type: none"> • Smear-positive pulmonary TB <p>Either: A patient with at least two sputum specimens positive for AFB by microscopy; or: A patient with at least one sputum specimen positive for AFB by microscopy and radiographic abnormalities consistent with pulmonary TB; or: A patient with at least one sputum specimen positive for AFB by microscopy, which is culture-positive for <i>M. tuberculosis</i>.</p> • Smear-negative pulmonary TB <p>A case of PTB that does not meet the above definition for smear-positive TB. This group includes cases without smear result. This commonly occurs in children but is comparatively uncommon in adults.</p> <p>Diagnostic criteria for PTB-should include:</p> <ul style="list-style-type: none"> – at least three sputum specimens negative for AFB, and – no clinical response to a one-week course of broad-spectrum antibiotics, and – radiographic abnormalities consistent with active PTB, and – decision by a clinician to treat with a full course of anti-TB chemotherapy. <p>A patient whose initial sputum smears were negative and whose subsequent sputum culture result is positive is also considered as having smear-negative pulmonary TB.</p> <p><u>Extrapulmonary tuberculosis (EPTB)</u></p> <p>EPTB refers to TB of organs other than the lungs, e.g. pleura, lymph nodes, abdomen, genitourinary tract, skin, joints and bones, meninges. Diagnosis should be based on one culture-positive specimen, or histological or strong clinical evidence consistent with active EPTB, followed by a decision by a clinician to treat with a full course of anti-TB chemotherapy.</p> <p>The case definition of an EPTB case with several sites affected depends on the site representing the most severe form of disease.</p> <p>Some cases will be easy to diagnose with peripheral lymphadenitis, swelling of cervical or axillary lymph nodes, chronic evolution and/or production of caseous discharge. Other cases, such as severe, life-threatening forms (e.g. miliary TB, TB meningitis), TB of bone joints, TB peritonitis, TB laryngitis will be suspected but should be referred to a hospital for assessment.</p>
<p>Mode of transmission</p>	<p>Exposure to tubercle bacilli in airborne droplet nuclei produced by people with pulmonary or laryngeal TB during expiratory efforts, such as coughing and sneezing. Extrapulmonary tuberculosis (other than laryngeal) is usually non-infectious.</p> <p>Bovine TB results from exposure to tuberculous cattle, usually by ingestion of unpasteurized milk or dairy products and sometimes by airborne spread to farmers and animal handlers.</p>

Progression to active disease	<p>Progression to active disease can take weeks or years; latent infections may persist throughout life. The risk of TB occurrence is relatively high during the first year following TB infection, then progressively decreases by half within the next 4–5 years.</p> <p>Only 10% of infected people with normal immune systems will develop clinically evident TB at some point in life; half of these will have an early progression of the disease (primary TB); the remaining half will have a late progression of the disease (post-primary TB) after a period of initial containment.</p>
Period of communicability	As long as viable tuberculosis bacilli are being discharged in the sputum. Effective treatment usually eliminates communicability within 2 weeks.

EPIDEMIOLOGY

Burden	<p>Estimated number of new cases: 2001: 15 778 (of which 4673 (29.6%) were notified) 2000: 12 225 (of which 3 760 (30.7%) were notified)</p> <p>Estimated number of new cases of smear-positive TB: 2001: 6 924 (of which 2692 (38.8%) were notified) 2000: 5365 (of which 2472 (46.0%) were notified)</p>
Geographical distribution	Specific data are unavailable, but TB is known to be widespread in the country.
Seasonality	No specific seasonality is reported.
Alert threshold	An increase in the number of cases in crowded settings must lead to an alert.
Recent epidemics	No data available.

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Population displacement disrupts existing TB programmes, including identification and treatment of TB cases. This leads to increased numbers of infected individuals in the community and interruption of TB therapy with a consequent rise in the proportion of treatment failures.
Overcrowding	Yes	Overcrowding is recognized as one of the most important factors leading to increased risk of transmission.
Poor access to health services	Yes	<p>People affected by TB who cannot access health services and be treated remain infectious for a longer period.</p> <p>The case fatality rate is high (about 50%) without proper treatment.</p> <p>The interruption of treatment is the most important cause of development of multidrug-resistant TB (MDR-TB).</p>
Food shortages	No	However, poor nutritional status increases vulnerability to TB infection and development of active disease.
Lack of safe water and poor sanitation	No	
Others	No	

<p>Risk assessment conclusions</p>	<p>Sierra Leone is in a phase of expansion of DOTS – the internationally recommended strategy for TB control. DOTS was introduced in 1990, and the national DOTS coverage increased from 50% in 2000 to 90% in 2001.</p> <p>The smear-positive TB case-detection rate (new smear-positive cases notified/new smear-positive cases estimated) fell from 46.0% in 2000 to 38.8% in 2001. The detection rate for all TB cases (all cases notified/all cases estimated) fell from 30.7% to 29.6% in 2001.</p> <p>The global target is to detect 70% of all cases and cure 85% of them by 2005.</p> <p><u>BCG vaccination coverage:</u></p> <p>2001: 60% (official country estimates); 74% (WHO/UNICEF estimates) 2000: 39% (official country estimates); 74% (WHO/UNICEF survey database) 1999: 55% (official country estimates) 1998: 78% (official country estimates) 1997: 38% (official country estimates) 1990: 98% (official country estimates) 1980: 36% (official country estimates)</p>
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PREVENTION AND CONTROL MEASURES

<p>Case management</p>	<p>Once the diagnosis of TB has been made and before treatment starts, all patients must be questioned carefully as to whether or not they have ever taken anti-TB drugs before. Patients should be classified according to the following criteria:</p> <ul style="list-style-type: none"> – site of disease (pulmonary or extrapulmonary) – severity of disease – bacteriological status (assessed by sputum microscopy) – history of anti-TB treatment (new or previously treated). <p><u>New case</u></p> <p>A patient who has never had treatment for TB or who has taken anti-TB drugs for less than 4 weeks and has</p> <ul style="list-style-type: none"> – sputum smear-positive pulmonary TB or – sputum smear-negative pulmonary TB, and extrapulmonary TB. <p><u>Previously treated case</u></p> <p>A patient who has at any time received anti-TB treatment for more than 1 month. This group of patients comprises:</p> <ul style="list-style-type: none"> – Return after interruption: common among recent refugees or IDPs. – Failure: a patient who, while on treatment, remained, or became again, smear-positive, 5 months or later after starting treatment; also, a patient who was smear-negative before starting treatment and became smear-positive after the second month of treatment. – Relapse: a patient who has been declared cured of TB in the past by a physician after a full course of chemotherapy and who has become sputum smear-positive. – Chronic: a patient who remained, or became again, smear-positive after completing a fully supervised, standardized re-treatment regimen (very small number of previously treated cases). <p>Good case management includes directly observed therapy (DOT) during the intensive phase for all new sputum-smear positive cases, the continuation phase of rifampicin-containing regimens, and the whole of the re-treatment regimen.</p> <p>There are three main types of regimen: Category I for new smear-positive (infectious) pulmonary cases, Category II for re-treatment cases, and Category III for smear-negative pulmonary or extrapulmonary cases (see “Treatment categories” below).</p>
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	<p>The chemotherapeutic regimes are based on standardized combinations of five essential anti-TB drugs:</p> <ul style="list-style-type: none"> – rifampicin (R) – isoniazid (H) – pyrazinamide (P) – ethambutol (E), and – streptomycin (S)* <p>Each of the standardized chemotherapeutic regimens consists of 2 phases:</p> <p>1. Initial (intensive)</p> <ul style="list-style-type: none"> – 2–3 months, with 3–5 drugs given daily under direct observation, for maximum reduction in the number of TB organisms. – The number of drugs used relates to the risk of failure of treatment due to bacterial resistance. <p>2. Continuation</p> <ul style="list-style-type: none"> – 4–6 months, with 2–3 drugs given 3 times a week under direct observation, or in some cases (e.g. during repatriation of refugees), 2 drugs for 6 months given daily unsupervised, but in a fixed-dose combination form.* <p>*Regimens are written in short form with the number of months the medication is to be given in front of the letter and the doses per week written after the letter. If there is no number after the letter, a daily dosage is given. The oblique symbol (/) separates the different phases of the therapy, e.g. 2 RHZE / 4 H3R3 means that for the first 2 months of treatment, rifampicin, isoniazid, pyrazinamide and ethambutol are given daily. This is followed by 4 months of rifampicin and isoniazid given regularly but each given only 3 times per week.</p> <p>All doses of rifampicin-containing regimens are observed by staff.</p> <p>Actual swallowing of medication must be checked.</p> <p>Hospitalized patients should be kept in a separate ward for the first two weeks of treatment.</p> <p><u>HIV-positive patients</u></p> <p>Anti-TB drug treatment is the same for HIV-positive and HIV-negative patients, with one exception: Thiacetazone, an essential anti-TB drug which at times is used in combination with isoniazid when financial constraints preclude the use of ethambutol should not be given to HIV-positive TB patients as there is increased risk of severe and sometimes fatal skin reactions.</p> <p>Controlled clinical trial studies have shown that isoniazid preventive treatment (IPT) reduces the risk of TB disease in HIV-positive individuals with latent TB infection (shown by a positive tuberculin skin test).</p> <p>The use of IPT has shown to be more effective than other regimens used for prevention of latent TB infection. The decision to use IPT must be carefully evaluated, and requires first the exclusion of active TB in the patient.</p> <p>To manage the problem of the HIV-TB coinfection effectively, TB and HIV programmes should coordinate activities through a TB/HIV coordinating body.</p> <p>See :</p> <ul style="list-style-type: none"> – <i>Treatment of tuberculosis: guidelines for national programmes.</i> Geneva, WHO, 2003 (WHO/TB/2003.313). – <i>Tuberculosis control in refugee situations: an inter-agency field manual.</i> Geneva, WHO, 1997 (WHO/TB/97.22; to be updated in 2004). – <i>An expanded DOTS framework for effective tuberculosis control: stop TB communicable diseases.</i> Geneva, WHO, 2002 (WHO/CDS/TB/2002.297).
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<p>Treatment categories</p>	<p>Standardized short-course chemotherapy using regimens of 6 to 8 months. Treatment categories are essential for prioritization of TB treatment according to public health risk – Category I is the highest priority.</p> <p><u>Category I</u></p> <p>These patients are:</p> <ul style="list-style-type: none"> – smear-positive persons who have never previously been treated or have only received treatment for less than one month – severely ill patients with other forms of TB (new smear-negative pulmonary TB with extensive parenchymal involvement, and new cases of severe forms of TB1), and – children with a score of 7 or more on the score chart. <p>The recommended regimen lasts 6 months. The initial (intensive) phase of treatment lasts for 2 months; rifampicin, isoniazid, pyrazinamide and ethambutol are given daily, or three times a week (streptomycin may be used as a substitute for ethambutol), under direct supervision.</p> <p>At the end of the second month, most patients will have a negative result on sputum microscopy; they can then progress to the second stage of treatment – the continuation phase. This phase lasts for 4 months, with rifampicin and isoniazid given 3 times per week, under direct supervision.²</p> <p>If the sputum smear examination is positive at the end of the second month, whatever the reason, the initial phase is prolonged for a third month. The patient then starts the continuation phase. If the sputum smears are still positive at the end of the fifth month, the patient is classified a treatment failure. He or she is re-registered, and starts a full course of the re-treatment regimen as a Category II patient.</p> <p>Drug dose is adjusted for weight gain at the end of the initial phase (2nd or 3rd month).</p> <p>¹This category includes patients with TB meningitis, disseminated TB, pericarditis, peritonitis, bilateral or extensive pleurisy, vertebral disease with neurological complications, and intestinal and genitourinary disease.</p> <p>²Daily self-administered ethambutol and isoniazid may be used in the continuation phase for 6 months, so this treatment regimen takes a total of 8 months.</p> <p><u>Category II</u></p> <p>Patients who were previously treated and are now sputum smear positive include:</p> <ul style="list-style-type: none"> – treatment after interruption; – treatment failure; and – relapse after treatment. <p>These patients should receive a standardized re-treatment regimen, fully supervised throughout both phases of treatment.</p> <p>The initial phase of treatment lasts for 3 months; rifampicin, isoniazid, pyrazinamide and ethambutol are given daily and supplemented by streptomycin daily for the first 2 months.</p> <p>The continuation phase of this regimen is 5 months of rifampicin, isoniazid and ethambutol given 3 times per week.</p> <p>Sputum smear examination is performed at the end of the initial phase of treatment (i.e at the end of 3 months), during the continuation phase of treatment (at the end of the fifth month) and at the end of treatment (at the end of the eighth month). If the patient is sputum smear-positive at the end of the third month, the initial phase of treatment is extended for one more month. Patients who are still positive at the end of the fourth month progress to the continuation phase, regardless.</p> <p><u>Category III</u></p> <p>These patients include:</p> <ul style="list-style-type: none"> – those with smear-negative pulmonary disease (with limited parenchymal
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	<p>involvement)</p> <ul style="list-style-type: none"> – adults and children with non-serious extrapulmonary disease (including symptomatic primary disease). <p>All Category III patients should receive 2 months of rifampicin, isoniazid and pyrazinamide daily, followed by 4 months isoniazid and rifampicin every second day (if it is decided that treatment is to be started). These patients are not high priority, and should not be treated in the initial stages of the TB programme or if resources are scarce.</p> <p>When the continuation phase can not be carried out under direct observation, all patients should be given daily ethambutol and isoniazid in the continuation phase for 6 months.</p> <p>See:</p> <ul style="list-style-type: none"> – <i>Treatment of tuberculosis: guidelines for national programmes</i>. Geneva, WHO, 1997 (WHO/TB/97.220). – <i>Tuberculosis control in refugee situations: an inter-agency field manual</i>. Geneva, WHO, 1997 (WHO/TB/97.221; to be updated in 2004). – <i>An expanded DOTS framework for effective tuberculosis control</i>. Geneva, WHO, 2002 (WHO/CDS/TB/2002.297).
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Treatment in Children	<p>The drug regimens used for children are the same as for adults. Drug dosages must be calculated according to the child's weight. Adjustments may have to be made during the course of the treatment as the child may rapidly regain lost weight.</p> <p>For infants of newly diagnosed smear-positive mothers, breastfeeding should continue. The infant should not be separated from the mother. Transmission is likely to have occurred already and the infant is at greater risk of dying from other causes if breastfeeding is stopped. If the infant is well, isoniazid prophylaxis should be given for 6 months and requires regular follow-up, for example one in every two months.</p> <p>See: Precautions for use of streptomycin and ethambutol in children: WHO/CDS/TB 2003.313, Page 64)</p>
Prevention	<p>Detection and treatment of smear-positive (infectious) TB cases is the most effective means of preventing the transmission of TB.</p> <p>Directly Observed Treatment Strategy (DOTS) programmes</p> <p>To ensure the appropriate treatment and cure of TB patients, strict implementation of the DOTS strategy is important. DOTS is the recommended strategy for TB control and has the following components:</p> <ul style="list-style-type: none"> – government commitment to ensuring sustained, comprehensive TB control activities; – case detection by sputum smear microscopy among symptomatic patients self-reporting to health services; – standardized short-course chemotherapy using regimens of 6–8 months, for at least all confirmed smear-positive cases (see "Case management" above); – a regular, uninterrupted supply of all essential anti-TB drugs; – a standardized recording and reporting system that allows assessment of case-finding and treatment results for each patient and of the overall performance of the TB control programme. <p>Complementary control strategies</p> <ul style="list-style-type: none"> – Health education to improve awareness and reduce stigma – Maintaining good ventilation and reducing overcrowding in health clinics, and ensuring that hospitalized patients are kept in a separate ward for the first 2 weeks of treatment. – Particular care must be taken to separate infectious TB patients from HIV-positive individuals. – BCG has been shown to be effective in preventing severe forms of TB such as meningitis in children (see below). <p>Isoniazid prophylaxis is not recommended in complex emergency situations, except for children being breastfed by smear-positive mothers. If the child is well, BCG vaccination should be postponed and isoniazid should be given to the child for 6 months. In the event of a sudden disruption to the programme, isoniazid may be stopped, and BCG should be given before the child leaves the programme (preferably after a 1-week interval).</p>
Immunization	<p>BCG has been shown to be effective in preventing severe forms of TB such as meningitis in children. As overcrowding and malnutrition are common in many refugee and displaced populations, the risk of TB transmission to children is increased.</p> <p>BCG is strongly recommended for all newborn children and any children up to the age of 5 years who have not already received it. The vaccination of newborns should be incorporated into routine immunization programmes for all children. Re-vaccination is not recommended.</p>

<p>Health education</p>	<p>Key elements of community education:</p> <ul style="list-style-type: none"> – avoiding stigmatization of TB patients – TB disease is curable – early (self) referral of TB suspects – importance of adherence to treatment – contact tracing. <p>The most important messages to teach:</p> <ul style="list-style-type: none"> – TB in an adult should be suspected when the person has a productive cough lasting more than 3 weeks, and/or blood in the sputum, with significant weight loss. – Cover the mouth whenever coughing or sneezing to prevent the spread of lung diseases. – Anyone may contract TB. – TB is curable. – Early treatment is important for best results and to prevent spread, especially to family members. – Children are especially at risk if not treated and may develop severe, even fatal, disease. – Good treatment is the best prevention. – All patients must take the full course of treatment. – Treatment makes patients non-infectious in 2 weeks, but cure takes 6–8 months. – Treatment must be completed even though the patient may feel better sooner. – Failure to complete the treatment may result in a recurrence which may be impossible to treat and spread of serious disease to others, especially to children. – All patients should be treated sympathetically and with respect. – Controlling TB is a community responsibility. <p>Note: Diagrams should be used as much as possible – a high literacy should not be assumed. Cured patients are often helpful teachers and supporters of new patients.</p>
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21. TYPHOID FEVER

DESCRIPTION

Infectious agent	Bacterium: <i>Salmonella enterica</i> serovar S. Typhi (older nomenclature was <i>Salmonella typhi</i> or <i>S.typhi</i>)
Case definition	<p>Clinical case definition</p> <p>Clinical diagnosis is difficult. In absence of laboratory confirmation, any case with fever of at least 38 °C for 3 or more days is considered suspect if the epidemiological context is conducive.</p> <p>Confirmed case</p> <p>Isolation of S.Typhi from blood or stool cultures.</p>
Mode of transmission	Faecal–oral route, particularly through contaminated water and food.
Incubation	Incubation period is usually 8–14 days but may be from 3 days up to 1 month.
Period of communicability	For 2 weeks from the onset of symptoms. Additionally, 2–5% of infected cases remain carriers for several months. Chronic carriers contribute significantly to the spread of the disease

EPIDEMIOLOGY

Burden	2003 (Jan-Jun): 644 cases 2002: 2303 cases 2001: 963 cases 2000: NA
Geographical distribution	Countrywide
Seasonality	No data available.
Alert threshold	Two or more linked cases.
Recent epidemics	None.

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Dissemination of multidrug-resistant strains of S. Typhi.
Overcrowding	Yes	Very important.
Poor access to health services	Yes	Early detection and containment of cases are paramount in reducing dissemination. Case fatality rate is high (10-20%) in absence of a proper treatment.
Food shortages	No	
Lack of safe water and poor sanitation	Yes	The most important risk factor.
Others	Yes	Multidrug-resistant strains of S. Typhi, including resistance to ciprofloxacin. Milk and dairy products are an important source of infection
Risk assessment conclusions		<p>In the general population, the risk is related to the availability of safe food and water. There is a high risk of epidemics, particularly in complex emergency settings where the above risk factors are common.</p> <p>Rapid diagnosis, determination of antibiotic sensitivity and institution of control measures are key factors in containing the risk.</p>

PREVENTION AND CONTROL MEASURES

Case Management	Early antimicrobial treatment, selected according to the antimicrobial resistance
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	<p>pattern of the strain.</p> <p>Quinolones (e.g. ciprofloxacin), co-trimoxazole, chloramphenicol and ampicillin are usually used for typhoid fever.</p> <p>Prevention of dehydration and case management using oral rehydration solution also play an important role.</p>
Epidemic control	<p>Inform the health authorities if one or more suspected cases are identified.</p> <p>Confirm the outbreak, following WHO guidelines.</p> <p>Confirm the diagnosis and ensure prompt treatment.</p>
Prevention	<p>See "Prevention" under Section 5 "Diarrhoeal diseases (others)", and Appendix 3, "Safe water and sanitation".</p>
Immunization	<p>Mass immunization may be an adjunct for the control of typhoid fever during a sustained, high-incidence epidemic. This is especially true when access to well functioning medical services is not possible or in the case of a multidrug-resistant strain.</p> <p>A parenteral vaccine containing the polysaccharide Vi antigen is the vaccine of choice among displaced populations. An oral, live vaccine using <i>S. Typhi</i> strain Ty21a is also available.</p> <p>Neither the polysaccharide vaccine nor the Ty21a vaccine is licensed for children under 2 years old. The Ty21a vaccine should not be used in patients receiving antibiotics.</p>

22. YAWS

DESCRIPTION

Infectious agent	<i>Treponema pallidum</i> , subspecies <i>pertenue</i> , a spirochaete.
Case definition	<p>A chronic, relapsing, non-venereal treponematosi s, characterized by contagious, early cutaneous lesions (papilloma/ulcers on the face or extremities) and non-contagious, late destructive lesions (skin and bones).</p> <p>Clinical description</p> <p>Primary lesion is typically a painless papilloma on the face or extremities (usually the leg) that persists for several weeks or months (mother yaw); it is usually painless unless there is secondary infection). This proliferates slowly and may form a raspberry lesion or undergo ulceration (ulceropapilloma).</p> <p>Secondary disseminated or satellite papillomata appear before or shortly after the initial lesion heals. Secondary lesions occur in successive crops, are often accompanied by mild constitutional symptoms, periostitis of the long bones (saber shin) and fingers (polydactylitis). Papillomata and hyperkeratosis may appear on palms and soles. These lesions are very painful and usually disabling. The lesions heal spontaneously, but relapses may occur at other sites.</p> <p>Tertiary or late stages occur 5 or more years after the primary infection occurs in about 10-20% of untreated patients. This stage is characterized by destructive lesions of skin and bone. Painful papillomata and hyperkeratosis on palms and soles may appear in this stage as well.</p> <p>The infection is rarely fatal, but can be very disfiguring and disabling.</p> <p>Laboratory diagnosis</p> <p>Diagnosis confirmed by dark-field or direct fluorescent antibody microscopic examination of exudates from lesions.</p> <p>Non-treponemal serological tests for syphilis (e.g. VDRL) are reactive in the initial phases, and become non-reactive after many years of latency. Treponemal serological tests (e.g. fluorescent treponemal antibody absorbed (FTA-ABS), microhemagglutination assay for antibody to <i>T. pallidum</i> (MHA-TP)) usually remain reactive through life.</p>
Mode of transmission	<p>Direct contact with exudates of early skin lesions of infected persons.</p> <p>Indirect transmission, through skin contamination from scratching, skin piercing articles and flies on open wounds, is probable.</p>
Incubation	From 2 weeks to 3 months.
Period of communicability	Variable; may extend intermittently over several years while moist lesions are present. The infectious agent is not usually present in late ulcerative lesions.

EPIDEMIOLOGY

Burden	93 cases reported between January and June 2003.
Geographical distribution	Remote rural communities of Bombali and Port Loko districts in the northern region.
Seasonality	Not determined.
Alert threshold	Every case should be notified to local health authorities.
Recent epidemics	Re-emergence of cases in late 1990s at the height of the conflict after elimination of the disease in the 1980s.

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Previously unexposed people moving into established foci of the disease.
Overcrowding	Yes	Many household members and siblings sharing the same sleeping space.

Poor access to health services	Yes	Poor surveillance, lack of health education, and untimely intervention. Lack of proximal water sources for bathing and washing of clothes.
Lack of safe water and poor sanitation	Yes	Poor personal hygiene due to lack of water for bathing, poverty and lack of health education to promote bathing.
Poverty and illiteracy	Yes	Ignorance about the disease and inability to access health care facility in times of need.

PREVENTION AND CONTROL MEASURES

Case management	<p>Specific treatment is with benzathine benzylpenicillin: for patients aged 10 years or more with active disease and contacts, a single injection of benzathine benzylpenicillin, 1.2 million units IM; for patients under 10 years of age, a single injection of benzathine benzylpenicillin, 0.6 million units.</p> <p>Concurrent disinfection, careful disposal of discharges and contaminated articles. Avoid intimate contact and contamination of the environment unless lesions are healed.</p> <p>Treat disfiguring and incapacitating late manifestations with appropriate topical and surgical care.</p> <p>All familial contacts should be treated; those with no active disease should be regarded as latent cases.</p>
Prevention	<p>General health education of population about treponematosi s, with emphasis on the value of better sanitation, and liberal use of soap.</p> <p>All cases should be reported to local health authorities. Intensive control activities should be organized at the community level, suited to the local problem. In areas of low prevalence, all active cases, all children and close contacts of infectious cases should be treated.</p> <p>Periodic clinical and serological surveys for latent cases, especially in children, should be conducted to prevent relapses and development of infective lesions that maintain the disease in the community. Continuous surveillance is essential for success. Treatment of the entire population should be considered when the prevalence of active disease exceeds 10%.</p> <p>As part of the national plan for mass control, health facilities should have the capacity for early diagnosis and treatment.</p>
Epidemic control	<p>Institute active mass treatment programmes in areas of high prevalence.</p> <p>Examine a high percentage of the population through field surveys. Conduct surveys at intervals of 1–3 years as part of national rural public health activities.</p> <p>Treat all active cases, including family contacts and community contacts.</p> <p>Displaced populations in endemic areas without hygienic facilities are potentially at greater risk.</p> <p>Where active mass treatment programmes are in place, protect countries or communities at risk of reinfection by instituting suitable public health and supervision measures against yaws in adjacent countries.</p>

23. YELLOW FEVER

DESCRIPTION

Infectious agent	Yellow fever virus, belonging to Flavivirus group
Case definition	<p><u>Clinical description</u></p> <p>Characterized by acute onset of fever followed by jaundice within 2 weeks of onset of first symptoms. Haemorrhagic manifestations and signs of renal failure may occur.</p> <p>There are 2 disease phases for yellow fever.</p> <p>Acute phase</p> <p>While some infections cause no symptoms at all, this first phase is normally characterized by fever, muscle pain (with prominent backache), headache, shivers, loss of appetite, nausea and/or vomiting. Often, the high fever is paradoxically associated with a slow pulse (Faget's sign). Most patients improve after 3–4 days and their symptoms disappear, but 15% enter the toxic phase.</p> <p>Toxic phase</p> <p>Fever reappears, the patient rapidly develops jaundice and complains of abdominal pain with vomiting. Bleeding can occur from mouth, nose, eyes and/or stomach. Once this happens, blood appears in the vomit and faeces. Kidney function deteriorates; this can range from abnormal protein levels in the urine (albuminuria) to complete renal failure with no urine production (anuria). Half the patients in the toxic phase die within 10–14 days. The remainder recover without significant organ damage.</p> <p><u>Laboratory criteria</u></p> <p>Isolation of yellow fever virus, or Presence of yellow-fever-specific IgM or a 4-fold or greater rise in serum IgG levels in paired sera (acute and convalescent), or Positive post-mortem liver histopathology, or Detection of yellow fever antigen in tissues by immunohistochemistry, or Detection of yellow fever virus genomic sequences in blood or organs by polymerase chain reaction.</p> <p><u>Case classification</u></p> <p>Suspected: a case that is compatible with the clinical description.</p> <p>Probable: not applicable.</p> <p>Confirmed: a suspected case that is laboratory-confirmed (national reference laboratory) or epidemiologically linked to a confirmed case or outbreak.</p>
Mode of transmission	<p>Bite of infective mosquitoes.</p> <p>The vectors of yellow fever in forest areas in Africa are <i>Aedes africanus</i> and other <i>Aedes</i> species. In urban areas, the vector is <i>Aedes aegypti</i> (all-day biting species).</p>
Incubation	From 3 to 6 days.
Period of communicability	Blood of patients is infective for mosquitoes shortly before onset of fever and for the first 3–5 days of illness. The disease is highly communicable where many susceptible people and abundant vector mosquitoes coexist. It is not transmitted by contact or other common means of disease transmission. Once infected, mosquitoes remain so for life.

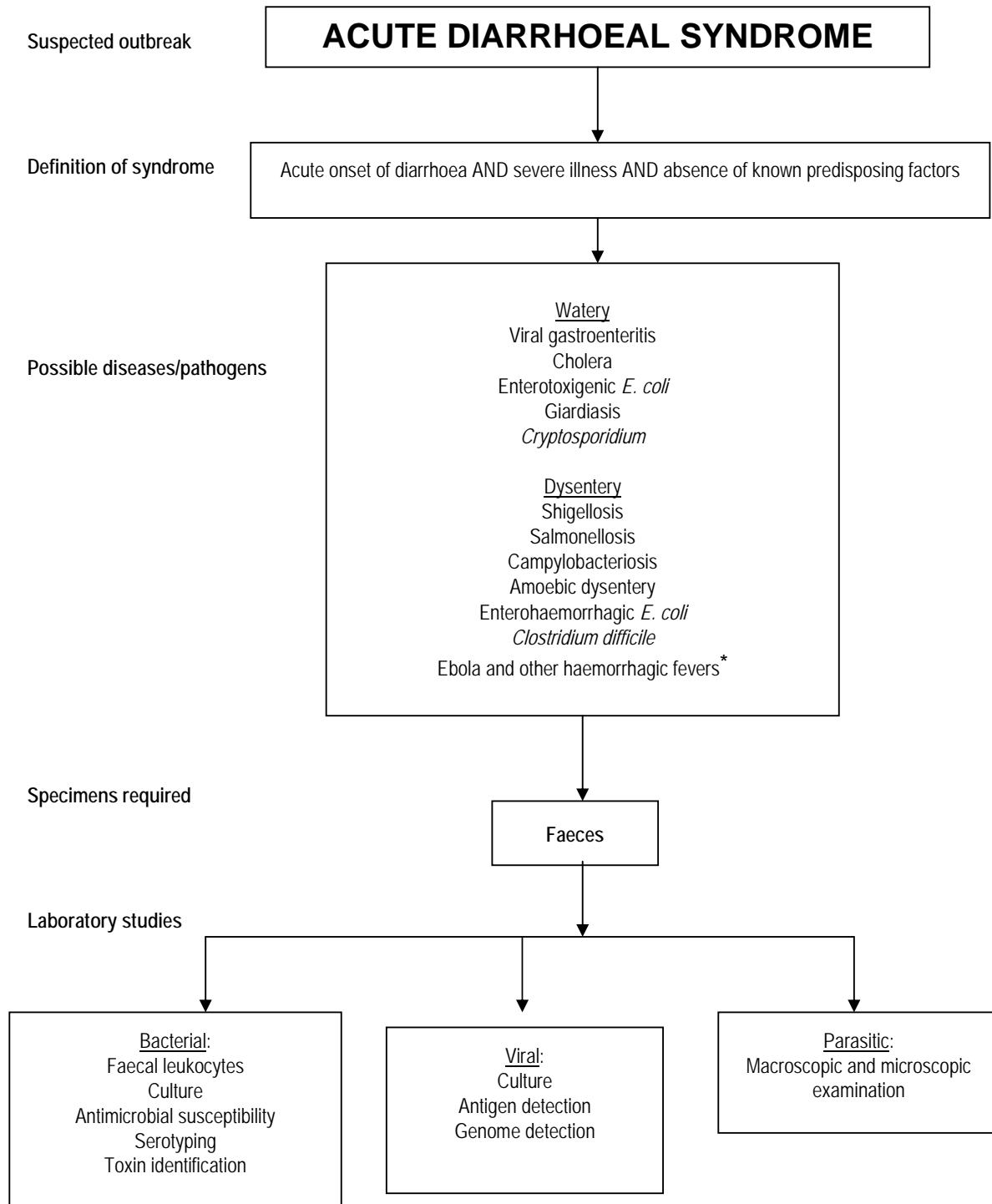
EPIDEMIOLOGY

Burden	<p><u>Cases notified to WHO/CDS</u></p> <p>2003: 90 cases</p> <p>2002: no data available</p>
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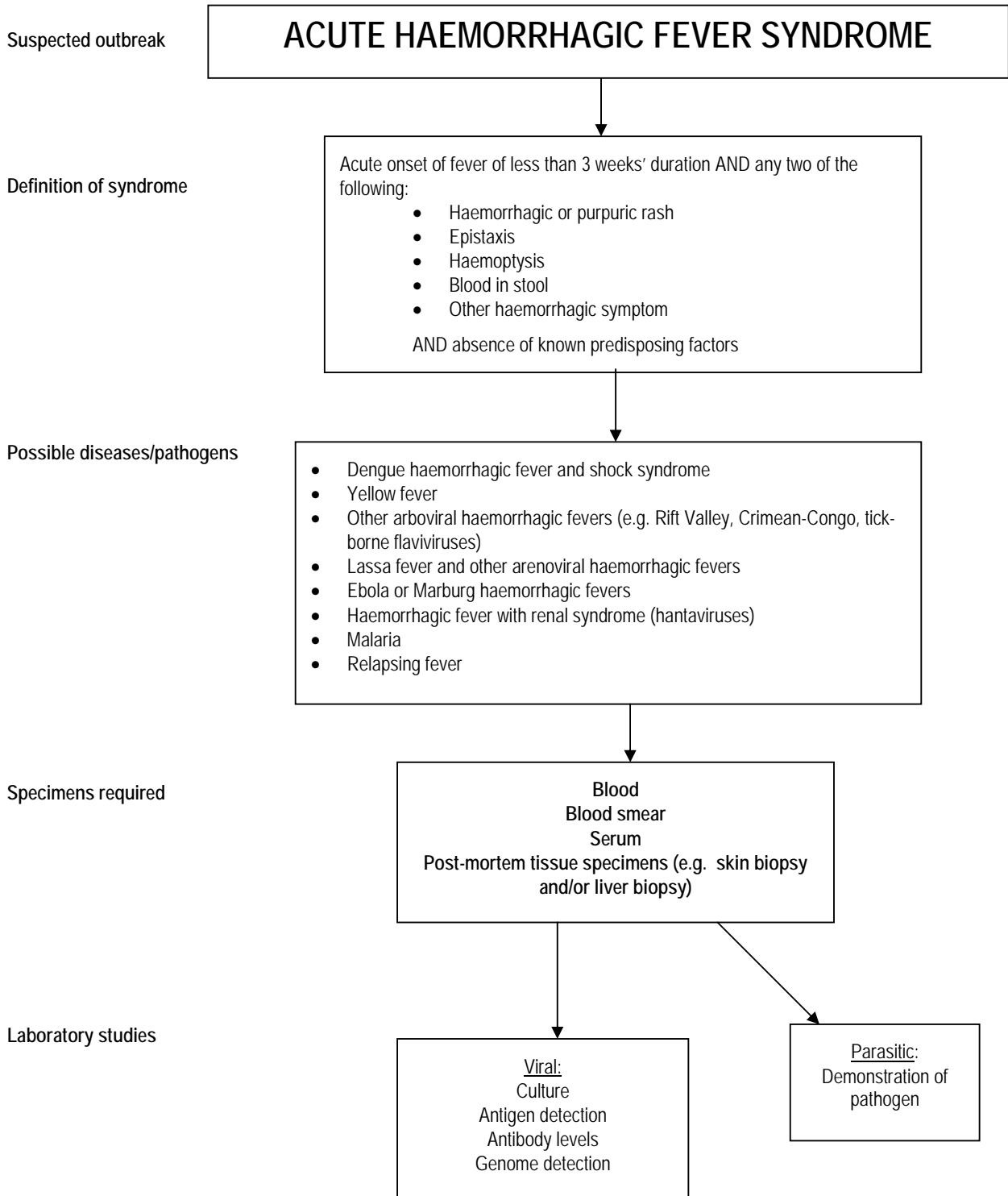
	<p>2001: no data available 2000: 1 case 1999: no data available 1998: no data available 1997: no data available 1996: 4 cases 1995: 1 case 1990: no data available 1980: no data available Since 1950, a total of 236 cases have been notified to WHO. <u>Deaths notified to WHO/CDS</u> 2003: 10 deaths No yellow fever deaths have previously been reported to WHO. (Data - 2004)</p>
Geographical distribution	The southern half of the country was officially endemic in the year 2000.
Seasonality	<p>In forest areas, where the yellow fever virus circulates between mosquitoes and monkeys or chimpanzees, the disease is present throughout the year.</p> <p>In field or savannah areas, the virus remains dormant in infected mosquito eggs throughout the dry season and emerges in the rainy season when eggs hatch.</p>
Alert threshold	<p>One confirmed case must lead to alert.</p> <p>An outbreak of yellow fever is at least one confirmed case.</p>
Recent epidemics	In 2003 there were 4 confirmed cases in Tonkolili. A total of 90 cases were notified to WHO in 2003.

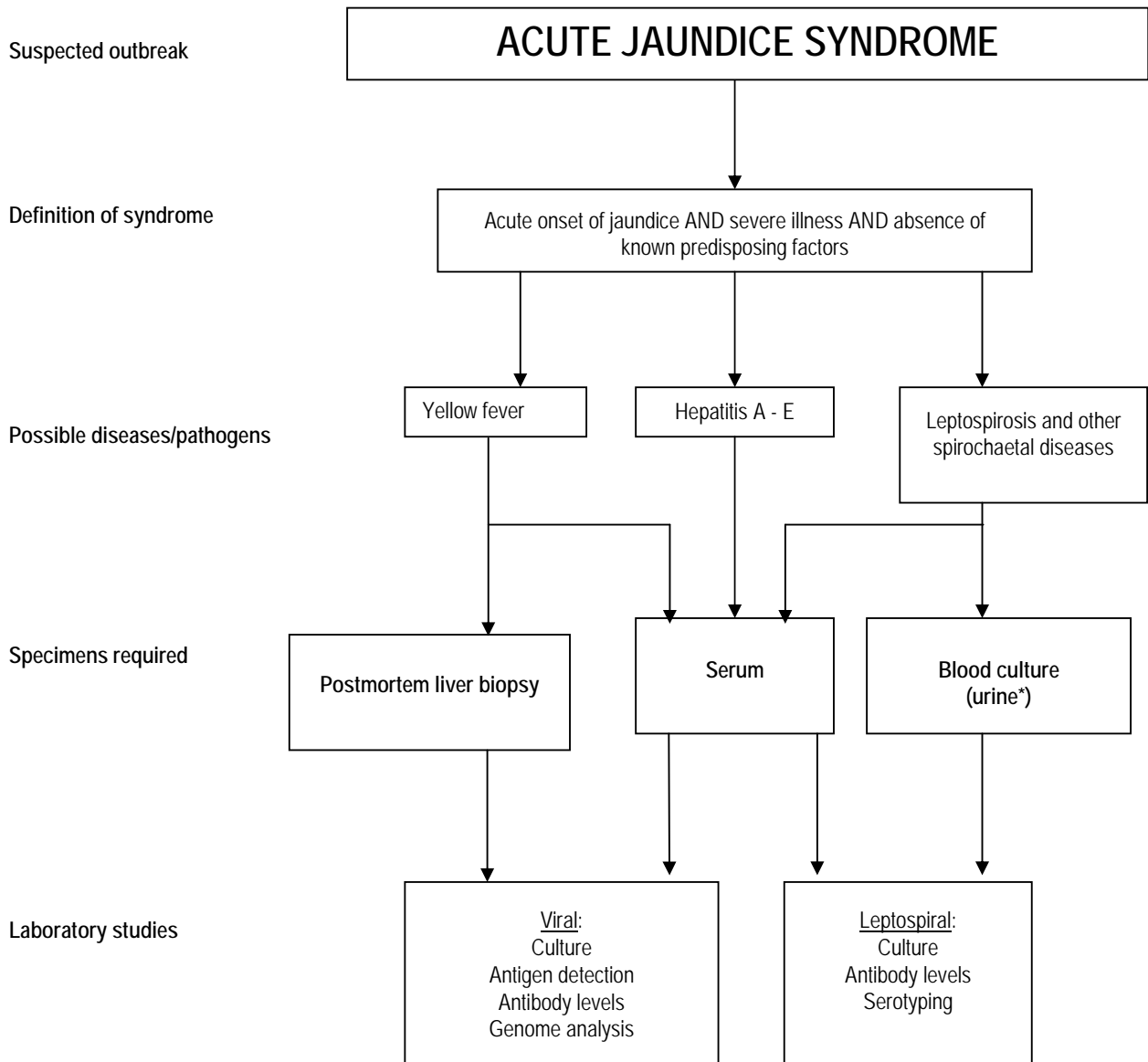
	<ul style="list-style-type: none"> — mass vaccination with YFV; — emergency mosquito control measures: <ul style="list-style-type: none"> · eliminating potential mosquito breeding sites (the most important mosquito control measure for YF control) · spraying to kill adult mosquitoes (less important because of limited impact) · use of ITNs.
<p>Prevention</p>	<p>Vaccination is the single most important measure for preventing yellow fever</p> <p>In endemic areas, vaccination must be done routinely through the incorporation of YFV in routine child immunization programmes and mass preventive campaigns. YFV is not recommended for symptomatic HIV-infected persons or other immunosuppressed individuals; for theoretical reasons, it is not recommended for pregnant women.</p> <p>Recommended strategies: vaccinating the population over 9 months in districts where coverage is less than 80%; if funds are limited, a lower cost intervention would be to vaccinate children between 9 months and 14 years to reach at least 50% of the population; yellow fever vaccination should be integrated in routine EPI activities.</p> <p>Routine mosquito control measures Potential mosquito breeding sites must be eliminated.</p>

APPENDIX 1: FLOWCHARTS FOR THE DIAGNOSIS OF COMMUNICABLE DISEASES

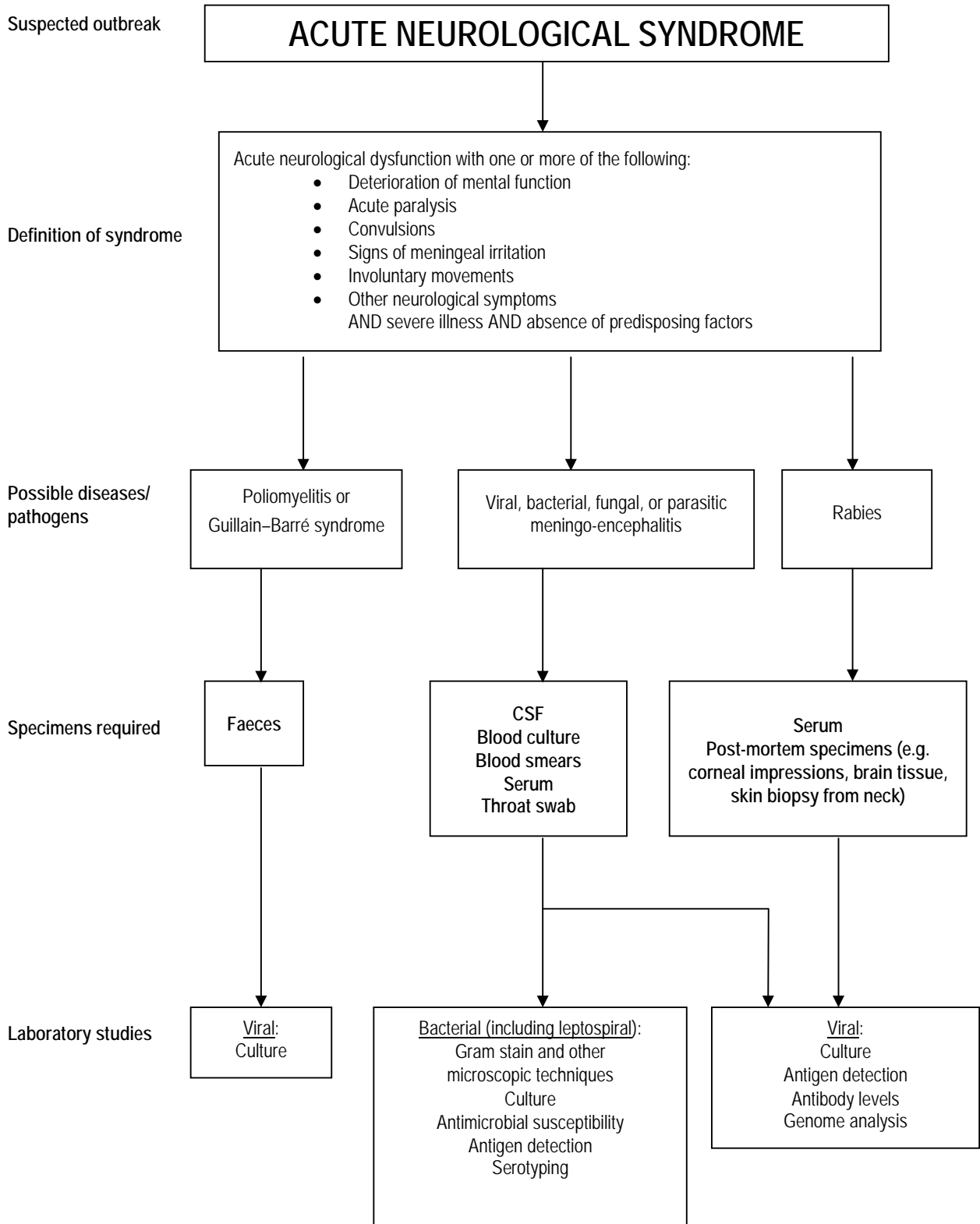


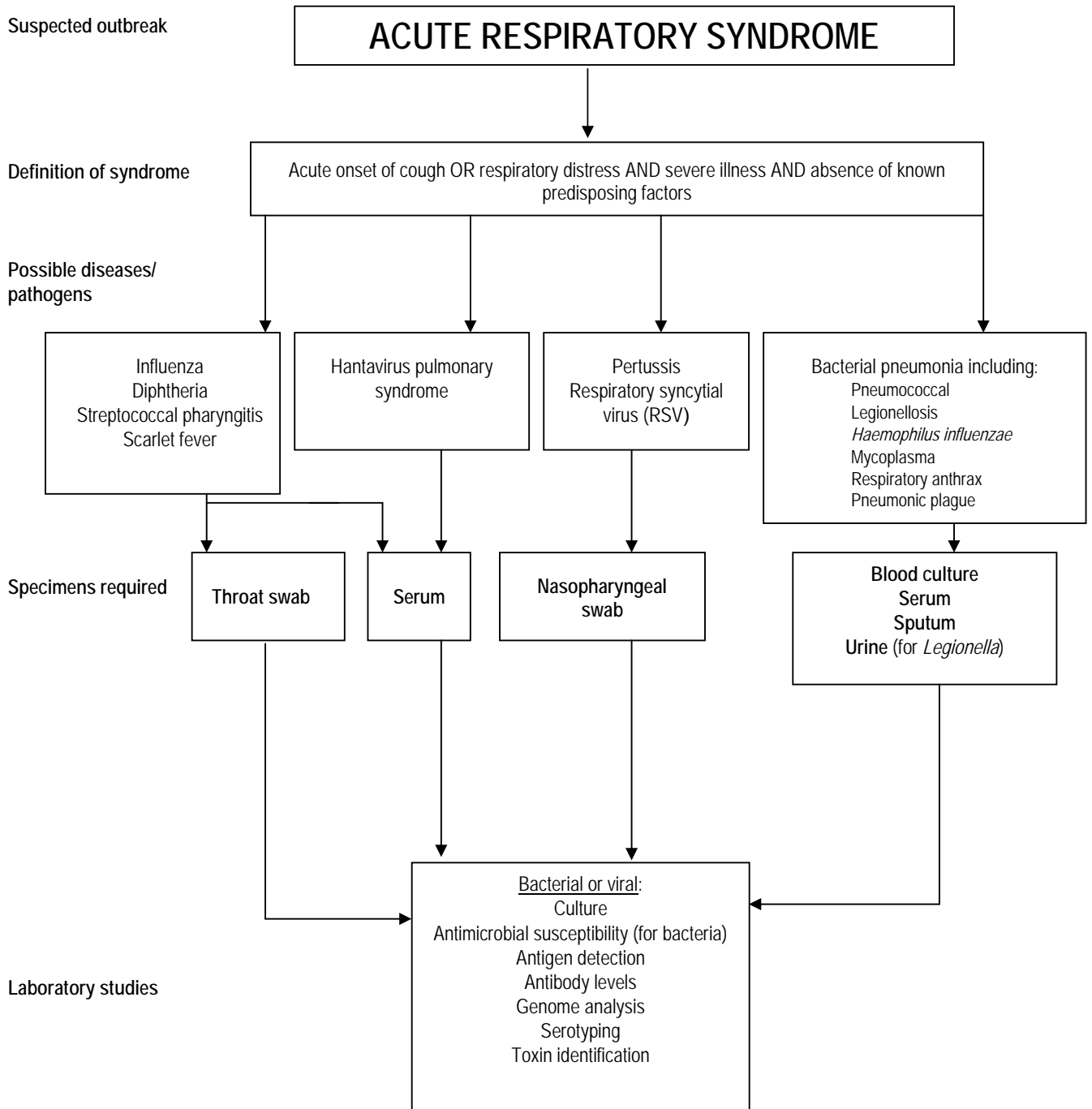
* Ebola and other haemorrhagic fevers may initially present as bloody diarrhoea. If such an etiology is suspected, refer to "Acute Haemorrhagic Fever Syndrome" for appropriate specimen collection guidelines. (See: *Infection control for Viral Haemorrhagic fevers in the African Health Care setting*, WHO/emc/lesr/98.2)





* Requires specialized media and handling procedures. See Annex 7 *Guidelines for collection of specimens for laboratory testing* in this toolkit.





Adapted from: *Guidelines for the collection of clinical specimens during field investigation of outbreaks*. Geneva, WHO, 2000 (WHO/CDS/CSR/EDC/2000.4).

APPENDIX 2 : STEPS IN OUTBREAK MANAGEMENT

PREPARATION

- Health coordination meetings
- Surveillance system – weekly health reports to WHO
- Stockpiles – specimen kits, appropriate antibiotics, IV fluids
- Epidemic investigation kits
- Contingency plans for isolation wards in hospitals
- Laboratory support

DETECTION

If a certain number of cases of any of the following diseases/syndromes is diagnosed (i.e. alert threshold is passed):

- Acute watery diarrhoea in over-5-year-olds
- Bloody diarrhoea
- Suspected cholera
- Measles
- Meningitis
- Acute haemorrhagic fever syndrome
- Acute jaundice syndrome
- Suspected polio (acute flaccid paralysis)
- Cluster of deaths of unknown origin

(diseases/syndromes in list to be modified according to country's up-to-date epidemiological profile).

Inform your health coordinator as soon as possible. The health coordinator should inform the Ministry of Health and Sanitation and WHO.

RESPONSE

Confirmation

- The lead health agency should investigate reported cases to confirm the outbreak situation – number of cases higher than that expected for same period of year and population. Clinical specimens will be sent for testing.
- The lead health agency should activate an outbreak control team with membership from relevant organizations: Ministry of Health, WHO and other United Nations organizations, nongovernmental organizations in the fields of health and water and sanitation, veterinary experts.

Investigation

- Confirm diagnosis (laboratory testing of samples).
- Define outbreak case definition.
- Count number of cases and determine size of population (to calculate attack rate).
- Collect/analyse descriptive data to date (e.g. time/date of onset, place/location of cases, and individual characteristics such as age/sex).
- Follow up cases and contacts.
- Determine the at-risk population.
- Formulate hypothesis for pathogen/source/transmission.
- Conduct further investigation/epidemiological studies (e.g. to clarify mode of transmission, carrier, infectious dose required, better definition of risk factors for disease and at-risk groups).
- Write an investigation report (investigation results and recommendations for action).

Control

- Implement control measures specific for the disease and prevent exposure (e.g. isolation of cases in viral haemorrhagic fever outbreak).
- Prevent infection (e.g. immunization in measles outbreak).
- Treat cases as recommended in WHO guidelines.

EVALUATION

- Assess timeliness of outbreak detection and response, cost.
- Change public health policy if indicated (e.g. preparedness).
- Write outbreak report and disseminate.

APPENDIX 3 : SAFE WATER AND SANITATION

The following are effective methods to obtain safe drinking water:

BOILING

To make water safe for drinking and hygiene purposes, bring it to a vigorous, rolling boil and keep it boiling for 1 minute. This will kill, or inactivate, most of the organisms that cause diarrhoea.

HOUSEHOLD FILTRATION

Household filtration should considerably reduce the pathogens in the water. It should be followed by disinfection through chlorination or boiling.

DISINFECTION THROUGH CHLORINATION

The following guidelines should be translated into messages that take into account locally available products and measuring devices. To make water safe by chlorination, the first step is to make a stock solution of chlorine. This can be prepared by adding the following products to one litre of water:

Product (% concentration by weight of available chlorine)	Amount for 1 litre
Calcium hypochlorite (70 %); or	15 g
Bleaching powder or chlorinated lime (30%); or	33 g
Sodium hypochlorite (5%); or	250 ml
Sodium hypochlorite (10 %); or	110 ml

The stock solution must be stored in a closed container, in a cool, dark place, and used within one month. It should be used to prepare safe water as follows:

Stock solution	Added volume of water
0.6 ml or 3 drops	1 litre
6 ml	10 litres
60 ml	100 litres

Mix by stirring and allow the chlorinated water to stand for at least 30 minutes before using it. The free residual chlorine level after 30 minutes should be between 0.1 and 0.5 mg/litre. If the free residual chlorine is not within this range, the number of drops of the stock solution should be adjusted so that the final product falls within this range.

If the water is cloudy or turbid it must either be filtered before chlorination or boiled vigorously rather than chlorinated. Chlorination of turbid water might not make it safe.

See:

- *Guidelines for cholera control*. Geneva, World Health Organization, 1993.
- *Cholera and other epidemic diarrhoeal diseases control - Technical cards on environmental sanitation*, World Health Organization, 1997. WHO/EMC/DIS/97.6

SANITATION

Good sanitation can markedly reduce the risk of transmission of intestinal pathogens, especially where its absence may lead to contamination of clean water sources. High priority should be given to observing the basic principles of sanitary human waste disposal, as well as to ensuring the availability of safe water supplies.

Appropriate facilities for human waste disposal are a basic need of all communities; in the absence of such facilities there is a high risk of water-related diseases. Sanitary systems that are appropriate for the local conditions should be constructed with the cooperation of the community.

People will need to be taught how to use latrines, about the dangers of defecating on the ground, or in or near water, and about the importance of thorough hand-washing with soap or ash after any contact with excreta. The disposal of children's excreta in latrines needs to be emphasized.

See:

- *Fact sheets on environmental sanitation for cholera control*. Geneva, WHO, 1996.
- Franceys R, Pickford J, Reed R. *A guide to the development of on-site sanitation*. Geneva, WHO, 1992.

APPENDIX 4 : INJECTION SAFETY

Analysis of data collected as part of the Comparative Risk Assessment component of the Global Burden of Disease study suggests that the WHO Africa region, in which Sierra Leone is situated, faces substantial challenges in terms of unsafe injection practices and transmission of bloodborne pathogens through injections. In this region, the proportions of new infections with hepatitis B, hepatitis C and HIV that are attributable to unsafe injections practices are 10.9%, 16.4% and 2.5% respectively. Thus, in any relief efforts to assist the population and the refugees in this region of the world, safe and appropriate use of injections should be ensured through the following actions:

PATIENTS

- State a preference for oral medications when visiting health care facilities.
- Insist upon a new, single-use syringe for every injection.

HEALTH WORKERS

- Avoid prescribing injectable medication whenever possible.
- Use a new, single-use syringe for every injection.
- Do not recap syringes, and discard them immediately in a sharps box to prevent needle stick injury.
- Dispose of by open-air incineration and burial of full sharp boxes.

IMMUNIZATION SERVICES

- Deliver vaccines with matching quantities of auto-disable syringes and sharps boxes.
- Make sterile syringes and sharps boxes available in every health care facility.

ESSENTIAL DRUGS:

- Build rational use of injections into the national drug policy.
- Make single-use syringes available in quantities that match injectable drugs in every health care facility.

HIV-AIDS PREVENTION

- Communicate the risk of HIV infection associated with unsafe injections.

HEALTH CARE SYSTEM

- Monitor safety of injections as a critical quality indicator for health care delivery.

MINISTRY OF HEALTH

- Coordinate safe and appropriate national policies with appropriate costing, budgeting and financing.

REMEMBER:

- Observe the "ONE SYRINGE – ONE NEEDLE SET – ONE INJECTION" rule.
- A safe injection is one that:
 - does no harm to the recipient
 - does not expose the health worker to avoidable risk
 - does not result in waste that puts other people at risk.
- An unsterile injection is usually caused by:
 - reusable syringes that are not properly sterilized before use
 - single-use syringes that are used more than once
 - used syringes and needles which are not disposed of properly.

APPENDIX 5 : KEY CONTACTS FOR SIERRA LEONE

Table 1: World Health Organization – Sierra Leone

<p>Office of the WHO Representative PO Box 529 Freetown – Sierra Leone</p>	<p>Dr J. Saweka <i>WHO Representative</i> Location 21 A & B Riverside Drive, Off King Harman Road Brookfield, Freetown. Sierra Leone.</p> <p>Tel: +232 22 223188 +232 22 241259 +232 22 229806 (direct line) Fax: +232 22 235215 E-mail: sawekaj@who-sl.org , swrsl@who-sl.org</p>
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Table 2: Relevant WHO Regional Offices and Headquarters Technical Staff

Areas of work	AFRO contact	HQ contact
Communicable disease surveillance and control	Dr Paul Lusamba lusambap@whoafr.org	Dr Máire Connolly connollyma@who.int Dr Michelle Gayer gayerm@who.int Dr Pamela Mbabazi mbabazip@who.int
Outbreak alert and response	Dr Paul Lusamba lusambap@whoafr.org	Dr Mike Ryan ryanm@who.int Mr Pat Drury druryp@who.int
Acute lower respiratory infections		Dr Shamim Qazi gazis@who.int
African trypanosomiasis		Dr Jean Jannin janninj@who.int
Bacillary dysentery – cholera – typhoid fever – other diarrhoeal diseases		Dr Claire-Lise Chaignat chaignatc@who.int
Diphtheria		Dr Julian Bilous bilousj@who.int
Dracunculiasis		Dr Ahmed Tayeh tayeha@who.int
HIV/AIDS		Dr Michel Tailhardes tailhardesm@who.int
Leishmaniasis		Dr François-Xavier Meslin meslinf@who.int
Leprosy		Dr Denis Daumerie daumeried@who.int Dr Myo Thet Htoon

		htoonm@who.int
Lymphatic filariasis		Dr Francesco Rio riof@who.int Dr Sergio Yactayo yactayos@who.int
Malaria		Dr Aafje Rietveld rietvelda@who.int Dr Jose Nkuni nkunij@who.int
Measles		Dr Brad Hersh hershbr@who.int
Meningococcal disease		Dr William Perea peraw@who.int Dr Eric Bertherat bertherate@who.int
Onchocerciasis		Dr Markus Behrend behrendm@who.int
Pertussis (whooping cough)		Dr Philippe Duclos duclosp@who.int
Poliomyelitis		Mr Chris Maher maherc@who.int Ms Claire Chauvin chauvinc@who.int
Rabies		Dr François-Xavier Meslin meslinf@who.int
Schistosomiasis		Dr Lorenzo Savioli saviolil@who.int Dr Dirk Engels engelsd@who.int
Soil-transmitted helminths		Dr Lorenzo Savioli saviolil@who.int Dr Dirk Engels engelsd@who.int
Tuberculosis		Dr Salah-Eddine Ottmani ottmanis@who.int Dr Giuliani Gargione gargioniq@who.int
Viral Haemorrhagic fevers		Dr Cathy Roth rothc@who.int

		Mr Pierre Formenty formentyp@who.int
Yellow Fever		Dr Sylvie Briand briands@who.int
Health aspects of biological agents		Dr Ottorino Cosivio cosivio@who.int
Injection safety		Dr Yvan Hutin hutiny@who.int
Safe water		Mr Jose Hueb hueb@who.int

APPENDIX 6 : LIST OF WHO GUIDELINES ON COMMUNICABLE DISEASES

Title	Publication No./Date
FACT SHEETS	
Anthrax	Fact Sheet No. 264 October 2001 http://www.who.int/mediacentre/factsheets/fs264/en/
Cholera	Fact Sheet No. 107 Revised March 2000 http://www.who.int/mediacentre/factsheets/fs107/en/
Crimean-Congo haemorrhagic fever	Fact Sheet No. 208 December 1998 http://www.who.int/mediacentre/factsheets/fs208/en/
Dengue and dengue haemorrhagic fever	Fact Sheet No. 117 Revised November 1998 http://www.who.int/mediacentre/factsheets/fs117/en/
Diphtheria	Fact Sheet No. 89 Revised September 2000 http://www.who.int/mediacentre/factsheets/fs089/en/
Epidemic dysentery	Fact Sheet No. 108 Revised October 1996 (Being updated)
<i>Escherichia coli</i> O157:H7	Fact sheet No. 103 Revised December 2000 (Being updated)
Food safety and foodborne illness	Fact Sheet No. 237 revised January 2002 http://www.who.int/mediacentre/factsheets/fs237/en/
Hepatitis B	Fact Sheet No. 204 Revised October 2000 http://www.who.int/mediacentre/factsheets/fs204/en/
Hepatitis C	Fact Sheet No. 164 Revised October 2000 http://www.who.int/mediacentre/factsheets/fs164/en/
Influenza	Fact Sheet No. 211 February 1999 http://www.who.int/mediacentre/factsheets/fs211/en/
Influenza A(H5N1)	Fact Sheet 15 th January 2004 http://www.who.int/csr/don/2004_01_15/en/
Injection safety: background	Fact Sheet No. 231 Revised April 2002 http://www.who.int/mediacentre/factsheets/fs231/en/
Injection safety: facts & figures	Fact Sheet No. 232 October 1999 (Being updated)
Injection safety: a glossary	Fact Sheet No. 233 October 1999 http://www.who.int/inf-fs/en/fact233.html
Injection safety: questions & answers	Fact Sheet No. 234 May 2000 (Being updated)
Leishmaniasis	Fact Sheet No. 116 Revised May 2000 http://www.who.int/mediacentre/factsheets/fs116/en/
Malaria	Fact Sheet No. 94 http://www.who.int/mediacentre/factsheets/fs094/en/
Plague	Fact Sheet No. 267 January 2002 http://www.who.int/mediacentre/factsheets/fs267/en/
Poliomyelitis	Fact Sheet No. 114 Revised August 2002 http://www.who.int/mediacentre/factsheets/fs114/en/

Rabies	Fact Sheet No. 99 Revised June 2001 http://www.who.int/mediacentre/factsheets/fs099/en/
Salmonella	Fact Sheet No. 139 January 1997 (Being updated)
Smallpox	Fact sheet October 2001 http://www.who.int/mediacentre/factsheets/smallpox/en/
Tuberculosis	Fact Sheet No. 104 Revised August 2002 http://www.who.int/mediacentre/factsheets/who104/en/
Typhoid Fever	WHO/Water Sanitation and Health http://www.who.int/water_sanitation_health/diseases/typhoid/en/
<u>GUIDELINES/PUBLICATIONS/REPORTS</u>	
First steps for managing an outbreak of acute diarrhoea http://www.who.int/csr/resources/publications/cholera/WHOCDSCSRNCS20037.pdf	WHO/CDS/CSR/NCS/2003.7 English only
Protocol for the assessment of national communicable disease surveillance and response systems. Guidelines for assessment teams http://www.who.int/emc-documents/surveillance/whocdscsrnr20012c.html	WHO/CDS/CSR/ISR/2001.2 English only
Strengthening implementation of the Global Strategy for Dengue Fever/Dengue Haemorrhagic Fever Prevention and Control http://www.who.int/emc-documents/dengue/whocdsdenic20001c.html	WHO/CDS/(DEN)/IC/2000.1 English only
WHO report on global surveillance of epidemic-prone infectious diseases http://www.who.int/emc-documents/surveillance/whocdscsrnr20001c.html	WHO/CDS/CSR/ISR/2000.1 English only
Guidelines for the collection of clinical specimens during field investigation of outbreaks http://www.who.int/emc-documents/surveillance/docs/whocdscsredc2004.pdf	WHO/EDC/2000.4 English only
Hepatitis A http://www.who.int/emc-documents/hepatitis/whocdscsredc20007c.html	WHO/CDS/EDC/2000.7 English only
Potential use of oral cholera vaccines in emergency situations. Report of a WHO meeting. Geneva, Switzerland, 12–13 May 1999 http://www.who.int/csr/resources/publications/cholera/whocdscsredc994.pdf	WHO/CDS/CSR/EDC/99.4 English only
WHO guidelines for epidemic preparedness and response to measles outbreaks http://www.who.int/emc-documents/measles/whocdscsrnr991c.html	WHO/CDS/CSR/ISR/99.1 English only
Influenza pandemic preparedness plan. The role of WHO and guidelines for national and regional planning http://www.who.int/emc-documents/influenza/whocdscsredc991c.html	WHO/CDS/CSR/EDC/99.1 English only
Plague manual: epidemiology, distribution, surveillance and control http://www.who.int/emc-documents/plague/whocdscsredc992c.html	WHO/CDS/CSR/EDC/99.2 English and French
Laboratory methods for the diagnosis of meningitis caused by <i>Neisseria meningitidis</i> , <i>Streptococcus pneumoniae</i> , and <i>Haemophilus influenzae</i> http://www.who.int/emc-documents/meningitis/whocdscsredc997c.html	WHO/CDS/CSR/EDC/99.7 English and French
Laboratory methods for the diagnosis of epidemic dysentery and cholera http://www.who.int/emc/diseases/cholera.html	WHO/CDS/CSR/EDC//99.8 English and French
Infection control for viral haemorrhagic fevers in the African health care setting http://www.who.int/emc-documents/haem_fevers/whoemcesr982c.html	WHO/EMC/ESR/98.2 English and French

Control of epidemic meningococcal disease. WHO practical guidelines, 2nd ed. http://www.who.int/emc-documents/meningitis/whoemcbac983c.html	WHO/EMC/BAC/98.3 English and French
Guidelines for the surveillance and control of anthrax in humans and animals, 3rd ed. Weekly epidemiological record, 20 th October 2001 http://www.who.int/wer	WHO/EMC/ZDI/98.6
Cholera and other epidemic diarrhoeal diseases control. Technical cards on environmental sanitation http://www.who.int/emc-documents/cholera/whoemcdis976c.html	WHO/EMC/DIS/97.6
Epidemic diarrhoeal disease preparedness and response. Training and practice. Participant's manual http://www.who.int/emc-documents/cholera/whoemcdis973c.html	WHO/EMC/97.3 Rev.1 English, French and Spanish
Epidemic diarrhoeal disease preparedness and response. Training and practice. Facilitator's guide http://www.who.int/emc-documents/cholera/whoemcdis974c.html	WHO/EMC/97.4 Rev.1 English, French and Spanish
Dengue haemorrhagic fever: diagnosis, treatment, prevention and control, 2nd ed. http://www.who.int/emc/diseases/ebola/Denguepublication/index.html	1997 English only
Guidelines for the control of epidemics due to <i>Shigella dysenteriae</i> type 1 http://www.who.int/emc-documents/cholera/whocdr954c.html	WHO/CDR/95.4 English only
Guidelines for cholera control http://www.who.int/emc/diseases/cholera.htm	1993 English and French
VIDEOS	
Protecting ourselves and our communities from cholera (41 min) http://www.who.int/emc/diseases/cholera/videos.html	2000 English and French
WEB SITES	
WHO	http://www.who.int
WHO AFRO	http://www.afro.who.int/
WHO Communicable Disease Surveillance and Response)	http://www.who.int/csr/
WHO Roll Back Malaria	http://www.rbm.who.int/
WHO Stop TB	http://www.stoptb.org/

APPENDIX 7: IMMUNIZATION SCHEDULE FOR SIERRA LEONE

VACCINE	SCHEDULE
BCG	Birth
DTwP	6, 10, 14 weeks
OPV	Birth, 6, 10, 14 weeks
Measles*	9 months
Vitamin A	9-59 months
Yellow Fever	9 months
Tetanus Toxoid	In pregnant women and WCBA : 1st contact; +1, +6 months; +1, +1 year

WCBA = women of child-bearing age

* Administered with Vitamin A (from 6-59 months)

APPENDIX 8: MAP OF SIERRA LEONE



APPENDIX 9: POPULATION OF SIERRA LEONE, 1995

Western Area			
DISTRICT	CAPITAL	AREA (km ²)	Population, 1985
Western Area	Freetown	600	554 000
Subtotal		600	554 000
Northern Province			
DISTRICT	CAPITAL	AREA (km ²)	Population, 1985
Bombali	Makeni	7 900	318 000
Kambia	Kambia	3 100	186 000
Koinadugu	Kabala	12 100	186 000
Port Loko	Port Loko	5 700	329 000
Tonkolili	Magburaka	7 000	240 000
Subtotal		35 800	1 259 000
Eastern Province			
DISTRICT	CAPITAL	AREA (km ²)	Population, 1985
Kailahun	Kailahun	3 900	234 000
Kenema	Kenema	6 100	337 000
Kono	Sefadu	5 600	390 000
Subtotal		12 600	961 000
Southern Province			
DISTRICT	CAPITAL	AREA (km ²)	Population, 1985
Bo	Bo	5 200	269 000
Bonthe	Bonthe	3 500	98 000
Moyamba	Moyamba	6 900	250 000
Pujehun	Pujehun	4 100	117 000
Sherbro, urban	Bonthe	10	7 000
Subtotal		19 710	741 000
Grand total		68 710	3 515 000^a

^aThe actual population (1995) was estimated at 4 081 000 (United Nations Population Division <http://esa.un.org/unpp/p2k0data.asp>)

APPENDIX 10: POPULATION DATA OF SIERRA LEONE, 2005*

Total population	5 340 000
Population density	74 per square km
Urban population (%)	2 148 000 (40.2%)
Rural population (%)	3 192 000 (59.8%)
Total male population	2 624 000
Total female population	2 716 000

* Data source: United Nations Population Division. World Population Prospects: The 2002 Revision Population Database.

APPENDIX 11: BASIC HEALTH INDICATORS IN SIERRA LEONE

Life expectancy at birth (years)	36 male, 39 female (1999) 34.2 (2000–2005)
Infant mortality rate	182 deaths per 1000 live births (2001)
Under-5 mortality rate	316 deaths per 1000 live births (2001)
Maternal mortality rate	1800 deaths per 100 000 live births (1995–2000)
Crude birth rate	45 live births per 1000 population (1999)
Annual population growth rate	1.5% (1995–2000) 2.4% (2000–2015)
Access to improved sanitation	66% of population (2000)
Access to an improved water source	57 % of population (2000)

Sources: UNICEF, World Bank, United Nations Development Programme.