

COMMUNICABLE DISEASE TOOLKIT

LIBERIA

1. COMMUNICABLE DISEASE PROFILE



World Health Organization
2004

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The WHO Regional Office for Africa (AFRO)
WHO Office, Liberia*

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ACKNOWLEDGMENTS

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This Profile 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 Liberia. 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).

The following people were involved in the development and review of this document and their contribution is gratefully acknowledged:

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We would like thank the Government of Ireland and the Office of US Foreign Disaster Assistance (OFDA) of the US Agency for International Development for their support in development of this document.

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INTRODUCTION

The purpose of this document is to provide public health professionals working in Liberia with up to date information on the major communicable disease threats that the population is facing. The list of endemic and epidemic diseases has been selected on the basis of the burden of morbidity and mortality. Diseases which have global eradication or elimination goals are also included. Due to the many years of civil unrest and conflict, data on Liberia are very limited. However, the document tries to outline the burden of communicable diseases in Liberia, provides data on reported outbreaks in the country, and presents disease-specific guidelines on the prevention and control of these diseases.

The control of communicable diseases represents a major challenge to those providing health care services in Liberia. It is hoped that this document will facilitate the co-ordination 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 lesser extent). Several respiratory viruses
Case definition	<p><u>Clinical description</u></p> <p>ALRI are bronchitis, bronchiolitis, and pneumonia. Pneumonia is the most severe and it is fatal in 10–20% of cases if inappropriately treated.</p> <p>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.</p> <p>Severe pneumonia Cough or difficult breathing and any general danger sign or Chest indrawing or stridor in a calm child.</p> <p>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.</p> <p>General danger signs For children aged 2 months to 5 years: inability to drink or breastfeed; vomiting; convulsions; lethargy or unconsciousness.</p>
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	<p>Most affected populations are the extreme age groups: children under 5 years old (particularly infants) and elderly people (>60 years old). Within these age groups mortality rates can be very high when appropriate management is not initiated promptly.</p> <p>In Liberia ALRI is one of the most common causes of morbidity and mortality in children under 1 year old and one of the three major causes of morbidity in all age groups. A prevalence survey in 2000 showed 29% prevalence rate of pneumonia among children under five years old (Liberia Democratic Households Survey) In 2001, according to the MoH outpatient morbidity report, 12.2% of all cases were ALRI.</p> <p>In Monrovia alone, from 18 August 2003 to 21 September 2003 (5 epidemiological weeks), 9727 cases of ALRI were reported (average 1945 cases/week). This is based on data collected from medical NGOs (data that represent only the population that have access to health centres run by NGOs). This period corresponds to the rainy season, when the incidence of ALRI is expected to be higher.</p>
Geographical distribution	ALRI is considered to have a widespread distribution. However, climatic conditions with lower temperatures (rainy season or altitude) could give rise to a higher incidence, especially where there is overcrowding and when people lack proper shelter, as is commonly the case with large population displacement.
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 increase the 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>ALRI are among the three major causes of morbidity in all age groups in Liberia.</p> <p>In other complex emergencies it has been observed that food insecurity, inadequate feeding practices, malnutrition and limited access to good-quality health care are likely to increase children's risk for illness and death, especially among rural populations and the poor.</p> <p>In this scenario, with high rates of malnutrition and low birth weight, a child's risk of dying of pneumonia is substantially increased.</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 cases in under-fives classified as pneumonia is co-trimoxazole; second-line antibiotic is amoxicillin.</p> <p>Pre-referral antibiotics for severe cases who cannot tolerate oral antibiotics or treatment for severe cases that cannot be referred:</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 also be treated for malaria, according to malaria laboratory results (blood film or rapid test) 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 must 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 must 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 immunization is effective in reducing the impact of ALRI. Immunization coverage rates for these antigens are not currently satisfactory in Liberia (measles-containing vaccine or MCV = 57% and DTP3 = 51% in 2002).</p>

2. AFRICAN TRYPANOSOMIASIS (African sleeping sickness)

DESCRIPTION

Infectious agent	Protozoa in Liberia,: <i>Trypanosoma brucei gambiense</i> .
Case definition	<p><u>Clinical description</u></p> <p>1st stage (haemolympathic involvement): Possibly fever, intense headache, sleeping disturbance, painless cervical lymphadenopathy, anaemia, local oedema and rash.</p> <p>2nd stage (neurological involvement): Parasites cross the blood–brain barrier and attack the central nervous system.</p> <p>Cachexia, sleeping disturbance and signs of central nervous system involvement.</p> <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><u>Laboratory criteria</u></p> <p>Serological: Card agglutination trypanosomiasis test (CATT): for <i>T. b. gambiense</i> only. A negative CATT result indicates that there is no disease; a positive result must be confirmed by microscopy.</p> <p>Parasitological: Detection (microscopy) of trypanosomes in blood or lymph node aspirates.</p> <p><u>Case classification</u></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 the infecting 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 (life span: 1–6 months).

EPIDEMIOLOGY

<p>Burden</p>	<p>The epidemiological profile of trypanosomiasis in Liberia is poorly understood.</p> <p>It is extremely difficult to measure accurately the incidence rates of gambiense sleeping sickness because of the long (and variable) asymptomatic period of the disease.</p> <p>Since most deaths take place in rural areas with poor or non-existent registration systems, there is little or no information on detected cases and mortality outside hospitals. This indicates that, in the current situation, reported data would underestimate the real problem.</p> <p>The current status of sleeping sickness in Liberia is unclear, but historical data confirm the presence of the disease. At the beginning of the last century, sleeping sickness was restricted to a few endemic areas in Liberia. The development of roads (with subsequent population movements) and plantations led to an increase of the disease.</p> <p>Over the period 1941–1943, Kissi in northern Liberia was an important focus of sleeping sickness, with a prevalence of 15% – similar to that in adjacent regions in neighbouring Guinea and Sierra Leone.</p> <p>According to reports from 1987, control activities reduced the prevalence in this area but no further control measures were instituted and the epidemiological profile in Kissi is currently unknown.</p> <p>Another focus has been described in the population north-east of Ganta, Nimba county, between the borders of Guinea and Côte d'Ivoire, with a prevalence of 1.5% reported during 1944–1953.</p> <p>A focus has been described in the Gbarnga area of Bong County, where 78% of the population in nine villages were screened and a prevalence of 1.1% was found (Hamburg Institute of Tropical Medicine, 1981). This indicates that <i>T. b. gambiense</i> has survived over decades in restricted areas.</p> <p>Reports from 1978 show the existence of an extremely active focus of Trypanosomiasis (more than 400 cases/year) in neighbouring Côte d'Ivoire, just 250 km from the Liberian border.</p>
<p>Geographical distribution</p>	<p>Foci of sleeping sickness have been reported in at least three counties in Liberia (Lofa, Bong and Nimba). These are the Kissi foci in northern Liberia, Gbarnga and Suakoko areas in Bong county, areas north-east of Ganta and Saniquellie in Nimba county between the borders of Guinea and Côte d'Ivoire, and the Foya area in Lofa county.</p>
<p>Seasonality</p>	<p>The disease has no clearly evident seasonal pattern.</p>
<p>Recent epidemics</p>	<p>The only reported epidemic occurred during 1940–1945 in Lofa county (Foya area) and reached a high prevalence (60%, unconfirmed).</p> <p>There is no epidemic threshold for sleeping sickness; any report of cases in a region where endemicity is unknown should lead to further investigation.</p>

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Risk of settlement in a high-transmission area.
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	
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 aren't 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 in recent years. Nevertheless, the close contact between humans, animals and the vector, as well as the proximity of the Guinean foci, makes it likely that such foci exist in Liberia.</p> <p>Moreover, the current internal conflict has led to large population movements to and from the neighbouring countries of Guinea and Côte d'Ivoire. These have been classified as highly endemic countries where the disease still poses a serious problem.</p> <p>The lack of surveillance for the disease and of technical expertise for testing and treating patients at peripheral health facilities means that most cases are unlikely to be detected – and therefore untreated and fatal.</p> <p>Sleeping sickness is also one of the few communicable diseases for which systematic population screening is necessary, and this must be re-established (along with appropriate treatment) in high-risk areas as soon as the security situation allows.</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 in four daily slow IV infusions (lasting approximately 2 hours). Infusions are given every 6 hours, representing 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% <i>treatment failure rate</i>, 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 by 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 periodic 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 consists of 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).
<p>Epidemic control</p>	<p>Mass surveys to identify affected areas.</p> <p>Early identification of infection in the community, followed by treatment.</p> <p>Urgent implementation of tsetse fly control measures (e.g. aerosol insecticides sprayed by helicopter and fixed-wing aircraft).</p>

3. BACILLARY DYSENTRY (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: Diarrhoea 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	Due to the collapse of the disease surveillance system there is no exact information on the extent of the disease. From January to July 2003, the Ministry of Health reported 2002 cases of bloody diarrhoea in the country (average 286 per month), although many counties did not report because of insecurity. From 11 August to 21 September 2003, just after crisis of June–July, 674 cases of acute bloody diarrhoea were reported in Monrovia (data coming from facilities run by NGOs).
Geographical distribution	Not known
Seasonality	In neighbouring countries 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	<p>There is limited information on cases of <i>Shigella</i> in previous years in Liberia. Apart from under-reporting, this may be partly the result of cases being treated with antibiotics before samples are collected.</p> <p>In 1998: April–June: 81 cases reported in Nimba county (Ganta district). November: 10 cases and 6 deaths reported (<i>S. dysenteriae</i> confirmed) in Bong county (Baila district). December: 63 cases in Zoe Geh, Nimba county; unknown number of cases and 3 deaths among patients admitted to hospital in Yekepa district, Nimba county (at least one case was confirmed <i>S. dysenteriae</i> type 1).</p> <p>In 2003: from 11 August to 27 October, 1857 cases (186 per week) were reported to the epidemic committee (data from 45 health facilities located in Montserrado, and some areas in Margibi, and Bong counties); <i>Shigella</i> (but not <i>dysenteriae</i> type 1), sensitive to nalidixic acid and ciprofloxacin, was confirmed by laboratory.</p>
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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 the cases are paramount in reducing transmission.</p> <p>In the absence of appropriate 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	However, malnutrition increases both gastrointestinal tract susceptibility to invasiveness of the organism and severity of disease.
Lack of safe water and poor sanitation	Yes	The most important risk factor.
Others	Yes	Contaminated food, lack of soap, and poor hygiene are also very important risk factors.
Risk assessment conclusions		<p>Risk of epidemics of <i>S. dysenteriae</i> type 1 is high in 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. Given that only 32% of the population have access to safe drinking-water (urban access 55%, rural access 10% – Liberia Demographic Health Survey, 2000), this risk is very high.</p>

PREVENTION AND CONTROL MEASURES

<p>Case management</p>	<p>Early and appropriate therapy is very important: treatment with an effective antimicrobial is the mainstay of therapy.</p> <p>Selection of the appropriate antibiotic depends on resistance patterns of the bacteria and drug availability. The problem of rapid acquisition of antimicrobial 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 reduce the risk of early development of resistance to this antibiotic.</p> <p>Resistance patterns may vary during the outbreak; regular stool sampling is therefore required.</p> <p>Supportive treatment using ORS, continued feeding (frequent, small meals) and use of antipyretics to reduce high fever are also essential.</p> <p><i>S. dysenteriae</i> type 1 is often more severe or fatal in young children, the elderly, and malnourished individuals, and prompt treatment with antibiotics is essential. If antibiotics are in short supply, they should be reserved for such high-risk groups.</p>
<p>Epidemic control</p>	<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, allow for timely mobilization of resources needed for appropriate case management and control.</p> <p>Confirm the outbreak. See “<i>Guidelines for outbreak control</i>” in this Toolkit.</p> <p>Rectal swabs should be collected from suspected cases and shipped, refrigerated, in an appropriate medium (e.g. Cary-Blair medium) to laboratories for culture to confirm the diagnosis of <i>S. dysenteriae</i> type 1. (The viability of bacteria in this medium when refrigerated is generally 1–3 days but is very 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, laboratory confirmation for every patient is unnecessary.</p> <p>Testing of <i>S. dysenteriae</i> type 1 isolates for antimicrobial sensitivity should be done at regular intervals to determine whether treatment remains appropriate. International reference laboratories are available to assist in the identification of the organism and confirmation of the antimicrobial resistance pattern.</p>
<p>Prevention</p>	<p>See:</p> <ul style="list-style-type: none"> – “Prevention” in Section 5, “Diarrhoeal diseases (others)”, and Annex 3, “Safe water and sanitation”. – <i>Guidelines for the control of epidemics due to Shigella dysenteriae type 1</i>. Geneva, WHO, 1995 (WHO/CDR/95.4, available at: http://www.who.int/emc-documents/cholera/whocdr954c.html)

4. CHOLERA

DESCRIPTION

Infectious agent	Bacteria: <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> <p>1. Person to person</p> <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). <p>2. Drinking contaminated water</p> <p>3. Eating food (fruits and vegetables) contaminated through</p> <ul style="list-style-type: none"> – water – soil – contamination <i>during</i> preparation (rice, millet, food from street vendors) – contaminated seafood. <p>4. Indirect contamination (hands)</p>
Incubation	Incubation period is usually a few hours to 5 days
Period of communicability	<p>During the symptomatic phase until 2–3 days after recovery. Very rarely for months.</p> <p>Asymptomatic carriers are common.</p>

EPIDEMIOLOGY

Burden	<p>Between July and October 2003 (4 months), more than 20 000 cases of cholera were recorded in Monrovia (data reported from NGO-run facilities). Cholera was laboratory-confirmed in Monrovia, also in Buchanan and Tubmanburg (see “Recent epidemics” below for details).</p> <p>Cases reported in Monrovia (source Médecins Sans Frontières – Belgium):</p> <table border="1"> <thead> <tr> <th>Year</th> <th>Total cases</th> <th>Attack rate</th> </tr> </thead> <tbody> <tr> <td>2002</td> <td>1617</td> <td>0.16</td> </tr> <tr> <td>2001</td> <td></td> <td></td> </tr> <tr> <td>2000</td> <td>1876</td> <td>0.18</td> </tr> <tr> <td>1999</td> <td>2305</td> <td>0.23</td> </tr> <tr> <td>1998</td> <td>4849</td> <td>0.48</td> </tr> <tr> <td>1997</td> <td>2282</td> <td>0.22</td> </tr> <tr> <td>1996</td> <td>5932</td> <td>0.59</td> </tr> </tbody> </table>	Year	Total cases	Attack rate	2002	1617	0.16	2001			2000	1876	0.18	1999	2305	0.23	1998	4849	0.48	1997	2282	0.22	1996	5932	0.59
Year	Total cases	Attack rate																							
2002	1617	0.16																							
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2000	1876	0.18																							
1999	2305	0.23																							
1998	4849	0.48																							
1997	2282	0.22																							
1996	5932	0.59																							
Geographical distribution	Cases have been reported from the most populous counties – Montserrado, Bong Bomi and Grand Bassa.																								
Seasonality	Beginning of the rainy season in April, in August and September, and the beginning of the dry season in November.																								

Alert threshold	This should be determined according to baseline data, if available. If baseline data are not available, any cluster of cases that match the above case definition must be investigated.
Recent epidemics	<p>In June 1998, 222 cases of severe diarrhoea with 8 deaths, clinically highly suspicious of cholera, were notified in Nimba county. According to the report, cholera was laboratory-confirmed, although no specifications were given regarding the laboratory or tests performed. At the same time, 138 cases and 4 deaths from severe diarrhoea were notified from Margibi county (no information on laboratory results).</p> <p>In October 2000, 31 clinically suspected cases and 8 deaths highly suspected of cholera were reported from Greenville, Sinoe County. However, cholera could not be confirmed because of antibiotic treatment before the investigation. The case-fatality rate was extremely high at 25.8%.</p> <p>In June–July 2003, during the attack by rebels on Monrovia, massive population displacement led to more than 250 000 people living in over 90 IDP sites in Monrovia. Combined with the lack of adequate water supply, sanitation and waste disposal, this situation provided ideal conditions for a cholera outbreak.</p> <p>Between early July and the end of October, 26 815 cases were recorded by health facilities run by NGOs, with an average of 2000 cases per week compared with 30 cases per week in the same period of previous years.</p> <p>Note: These figures are reported cases only and are an underestimation – security conditions prevented many people from reaching health facilities.</p>

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	Yes	More severe cases in malnourished population.
Lack of safe water and poor sanitation	Yes	The most important risk factor.
Others	Yes	Poor hygiene and lack of soap. Funerals and cultural practices that involve contact with the dead body (cholera case) could become important sources of the spread of disease.
Risk assessment conclusions		<p>Cholera outbreaks are expected to recur (superimposed on the continuing underlying incidence), given that displaced populations are living in overcrowded conditions, insufficient safe water is available and the general sanitation is very poor. There is also the risk of spread the disease to other regions in Liberia through population movement.</p> <p>The high prevalence of acute and chronic malnutrition in Liberia increases susceptibility to more severe disease.</p>

PREVENTION AND CONTROL MEASURES

<p>Case management</p>	<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 can be extremely high (5–40%) in the absence of proper treatment. With appropriate case management, the rate may be less than 1%.</p>
<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 available. 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.</p> <p>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 rehydration 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"> – “Guidelines for collection of specimens” in this Toolkit. – 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 Annex 3, “Safe Water and Sanitation”. – <i>Guidelines for cholera control</i>. Geneva, WHO, 1993.
<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 – the killed cholera vaccine (WC/rBS, two doses) and the attenuated live vaccine (CVD103-HgR, single dose) – and have been licensed in some countries.</p> <p>See:</p> <ul style="list-style-type: none"> – <i>Cholera vaccines: a new public health tool? Report, WHO meeting, 10–11 December 2002, Geneva, Switzerland</i>. 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>Salmonella</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	<u>Clinical case definition for acute diarrhoea</u> 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	According to the Liberia Demographic Health Survey in 2000, the diarrhoea prevalence rate increased slightly from 22.5% in 1997 to 23% in 2000. The MoH outpatient morbidity report of 2001– based on outpatient morbidity data from 11 counties – indicates that diarrhoea cases represent 6.9% of all outpatient cases.
Geographical distribution	Not known
Seasonality	The “diarrhoea season” in Liberia is at the beginning of the rainy season in April and from August to September, also at the beginning of the dry season during November.
Alert threshold	An increase in the number of cases above the expected level compared with previous years or baseline data.
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 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	Poor hygiene; lack of soap; contaminated food items.
Risk assessment conclusions		<p>The diarrhoea prevalence rate of 6.9% (MoH outpatient morbidity data) is likely to be underestimated; the prevalence is probably much higher given the poor access to health care.</p> <p>Lack of safe water and adequate sanitation, overcrowded living conditions and poor hygiene practices in Liberia increase the risk of diarrhoea. In addition, the hot, humid climate and inadequate fluid intake can aggravate the dehydration due to diarrhoea.</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 the deaths usually occur among children under 2 years old.</p>

PREVENTION AND CONTROL MEASURES

Case management	<p>Reduction of mortality due to diarrhoeal disease depends primarily on the correct management of dehydration, particularly in children</p> <p><u>Prevention</u></p> <p>Give recommended home fluid (RHF) and oral rehydration salts (ORS).</p> <p><u>Treatment of dehydration</u></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 of infants and young children.</p> <p>Resume feeding 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></p> <p>Provision of an adequate and safe supply, collection and storage system. Provision of information on the importance of clean water, including system maintenance and household storage (see Annex 3, "Safe water and sanitation".)</p> <p><u>Safe disposal of human excreta</u></p> <p>Provision of adequate facilities for the disposal of human waste. Provision of information on the importance of human waste disposal, including use and maintenance of the facilities.</p> <p><u>Food safety</u></p> <p>Provision of adequate storage facilities for food (both uncooked and cooked), cooking utensils, adequate quantities of water and fuel to allow for cooking and reheating of food. Health education on the importance of food safety and safe food handling.</p> <p><u>Hand-washing with soap</u></p> <p>Provision of sufficient soap 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></p> <p>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 criteria</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.</p> <p>Note: persons with positive <i>C. diphtheriae</i> cultures who do not meet the clinical description (e.g. asymptomatic carriers) should 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>There is no information about cases since 1980 (WHO's Immunization Profile – Liberia, Vaccines, Immunization and Biologicals), which probably reflects the under-reporting during this period of political instability in the country.</p> <p>The disease affects mainly non-immunized children under 15 years of age but it can also often affect adults, particularly in populations not vaccinated as children.</p>
Geographical distribution	Unknown
Seasonality	In tropical countries such as Liberia, seasonal trends are less distinct than in temperate regions.
Alert threshold	One case that meets the clinical description (probable 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 management of cases are paramount in reducing transmission
Food shortages	No	
Lack of safe water and poor sanitation	No	
Others	Yes	Lack of complete immunization.
Risk assessment conclusions		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. <u>DTP3 coverage in Liberia</u> 2002: 51% 2001: 62% 2000: 48% 1999: 23% (WHO/UNICEF estimates)

PREVENTION AND CONTROL MEASURES

Introduction	The control of diphtheria is based on 3 measures: – ensuring high population immunity through vaccination (primary prevention) – rapid investigation and treatment of contacts (secondary prevention of spread) – early diagnosis and appropriate case management (tertiary prevention of complications and deaths).
Immunization	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. Diphtheria-toxoid-containing vaccine (preferably a vaccine with reduced diphtheria content (Td) should be given. To ensure injection safety during immunization, auto-disable syringes and safety boxes are recommended. Safe disposal of used sharps should be ensured.

<p>Case management</p>	<p>Diphtheria antitoxin and antibiotic therapy are the cornerstones of therapy for diphtheria. The antibodies neutralize toxin only <i>before</i> its entry into cells. It is therefore critical that diphtheria antitoxin is 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 a total of 14 days.</p> <p><u>Close contacts</u>¹</p> <p>Surveillance for 7 days for close contacts, regardless of vaccination status, and throat cultures.</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 G IM (600 000 units for children under 6 years; 1.2 million units for those age 6 and older).</p> <p><i>Note:</i> 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

Epidemic control	<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 whether it fulfils the case definition, and record the 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 they prove not to be carriers.</p> <p>Implement outbreak response measures. Give priority to case management and immunization of populations in areas not yet affected but to which 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. HIV/AIDS

DESCRIPTION

Infectious agent	Human immunodeficiency virus (HIV). Two types have been identified, HIV-1 and HIV-2, have been identified. They have similar epidemiological characteristics, but HIV-2 is less pathogenic than HIV-1.
Case definition	<p><u>AIDS case definition</u></p> <p>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</p> <ol style="list-style-type: none"> 1. Asymptomatic 2. Persistent generalized lymphadenopathy (PGL) <p>Performance Scale 1: <i>asymptomatic, normal activity</i></p> <p>Stage 2</p> <ol style="list-style-type: none"> 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 last 5 years 6. Recurrent upper respiratory tract infections (e.g. bacterial sinusitis) <p>And/or Performance Scale 2: <i>symptomatic, normal activity</i></p> <p>Stage 3</p> <ol style="list-style-type: none"> 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) <p>And/or Performance Scale 3: <i>bedridden <50% of the day during the last month</i></p> <p>Stage 4</p> <ol style="list-style-type: none"> 14. HIV wasting syndrome, as defined by the Centres 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, coccidioidomycosis) 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><i>Note: both definitive and presumptive diagnoses are acceptable</i></p> <p>(a) HIV wasting syndrome: weight loss of >10% of body weight, plus either unexplained chronic diarrhoea (>1 month) <i>or</i> chronic weakness and unexplained prolonged fever (>1 month).</p>

	<p>(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.</p> <p>Expanded WHO case definition for AIDS surveillance*</p> <p>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:</p> <ol style="list-style-type: none"> 1. >10% body weight loss or cachexia, with diarrhoea or fever, or both, 2. intermittent or constant, for at least 1 month, not known to be due to a condition unrelated to HIV 3. Cryptococcal meningitis 4. Pulmonary or extrapulmonary tuberculosis 5. Kaposi sarcoma 6. 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) 7. Candidiasis of the oesophagus (which may be presumptively diagnosed based on the presence of oral candidiasis accompanied by dysphagia) 8. Clinically diagnosed life-threatening or recurrent episodes of pneumonia, with or without etiological confirmation 9. Invasive cervical cancer <p><i>*Weekly Epidemiological Record, 1994, 69:273–275.</i></p> <p>Laboratory evidence of HIV</p> <p>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.</p> <p>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.</p> <p>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 in serum/plasma, and testing may therefore be performed by non-laboratory personnel with adequate training and supervision.</p>
<p>Mode of transmission</p>	<p>Sexual intercourse (vaginal or anal) with an infected partner, especially in the presence of a concurrent ulcerative or non-ulcerative sexually transmitted infection (STI).</p> <p>From infected mother to her child during pregnancy, labour and delivery or through breastfeeding.</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> <p>Transfusion of infected blood or blood products</p>
<p>Incubation</p>	<p>Variable. On average, time from HIV infection to clinical AIDS is 8–10 years, although clinical AIDS may manifest in less than 2 years or be delayed in onset beyond 10 years from time of HIV infection. Incubation times are shortened in resource-poor settings and in older patients. They can be prolonged by primary prophylaxis for opportunistic infections or by 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>
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EPIDEMIOLOGY

Burden	<p>The first case of AIDS was reported in Liberia in 1986. The HIV/AIDS epidemiological surveillance report from the National AIDS and STI Control Programme (NACP), shows an overall HIV prevalence rate of 8.1%; data were collected from 24 sentinel surveillance sites in 11 counties from January to December 2002. These data are not truly representative of the whole country since not all counties have sentinel sites or laboratory facilities.</p> <p>Among all age groups, women had a higher HIV infection rate than men. The sources for these data were anonymously tested blood donors, volunteers, TB patients, antenatal clinic (ANC) attendees, and inpatients, as well as outpatients (diagnostic).</p> <p>According to surveillance data from the NACP, Liberia had an HIV/AIDS prevalence of less than 1% in the late 1980s, before the start of the civil conflict. In 2000, prevalence was estimated by the NACP at 8.2%. Most AIDS cases reported during 1994–2002 were from Montserrado county. The highest number of confirmed AIDS cases in a single year was 88 cases in 2002, followed by 87 cases in 1998 (surveillance report/NACP). However, these figures are the result of limited and irregular surveillance in several populations. The prevalence of HIV in TB patients was 11.6% outside major urban areas (UNAIDS/UNICEF/WHO, 1998).</p> <p>The limited information available on HIV prevalence among ANC attendees in Liberia is based on health facility HIV sero-surveys. In Monrovia, the major urban area, 4% of ANC attendees tested in 1992 and 1993 were positive for HIV. In 1996 and 1997, HIV testing at various sites found no evidence of HIV infection among ANC attendees. In 1998, 10% of ANC attendees from an unspecified site were HIV-positive, and in 1999, 12.7% of the ANC attendees tested across the country were HIV-positive. There is no information on HIV prevalence among ANC attendees in 2000 and 2001 (UNAIDS/WHO Fact Sheet, 2002).</p>												
Geographical distribution	<p>The NACP categorizes data according to major urban areas and non-major urban areas. However, because of ongoing conflict over years, these data have been collected irregularly and geographical differences in prevalence cannot be accurately determined.</p> <p>The surveillance report from the NACP indicates that Montserrado County had the highest infection rate at 6.3%, followed by Margibi County with 5.5% (2002).</p>												
Seasonality	Not applicable												
Alert threshold	One suspected case must be investigated.												
Recent epidemics	<p>Number of AIDS cases by year of reporting (UNAIDS/WHO Fact Sheet, 2002):</p> <table style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 50%;">1991: 10</td> <td style="width: 50%;">1997: 59</td> </tr> <tr> <td>1992: 9</td> <td>1998: 114</td> </tr> <tr> <td>1993: 4</td> <td>1999: 79</td> </tr> <tr> <td>1994: 13</td> <td>2000: 81</td> </tr> <tr> <td>1995: 11</td> <td>2001: 52</td> </tr> <tr> <td>1996: 8</td> <td></td> </tr> </table> <p>Total number of confirmed cases: 532 (last report: November 15, 2001).</p>	1991: 10	1997: 59	1992: 9	1998: 114	1993: 4	1999: 79	1994: 13	2000: 81	1995: 11	2001: 52	1996: 8	
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1995: 11	2001: 52												
1996: 8													

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	<p>Movement of population to areas of higher risk (to and from Côte d'Ivoire, Guinea, Sierra Leone). Population movement also results in:</p> <ul style="list-style-type: none"> – breakdown of family and social ties – economic strain on families, prostitution as income source – erosion of traditional values and coping strategies, which can result in higher-risk sexual behaviour that increases the risk of HIV spread – Influence of illicit drug trafficking and drug use, increasing risk of HIV transmission through injecting drug use.
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 shelter, where there is greater potential for sexual contact.</p> <p>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 supplies for HIV prevention, particularly condoms, are likely to be lacking in complex emergency countries.</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, or extra food, 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>If injecting drug use is practised, complex emergency situations increase the risk that needles will be shared.</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 regular transfusion services have broken down.</p>

	<p><u>Adolescent health</u></p> <p>Children in complex emergencies may have little to occupy themselves, and this may lead them to experiment with sex earlier than children in other situations.</p> <p><u>Lack of regular supplies</u></p> <p>Lack of laboratory reagents for screening and testing particularly for blood transfusions.</p> <p>Lack of condoms.</p>
<p>Risk assessment conclusions</p>	<p>Because of 14 years of conflict, there are limited data on the number of people infected with HIV/AIDS. The sentinel site surveillance system formerly in place is functioning only partially, if at all.</p> <p>The overall prevalence rates of HIV/AIDS in Liberia appear to be increasing and may be 8% and higher in certain risk groups, such as pregnant women. The TB/HIV co-infection is also worrying (with a prevalence of 11.8% in 1998).</p> <p>The war has exacerbated the risk of HIV/STI transmission through increases in sexual violence against women and in prostitution among girls and women seeking to meet their basic needs, but also through unsafe abortions, low condom use and risky sexual behaviour (USAID).</p> <p>The following must be addressed:</p> <ul style="list-style-type: none"> – The population’s lack of awareness and knowledge of HIV/AIDS transmission (education, media messages). – All stakeholders involved in humanitarian activities must be sensitized to the importance of addressing HIV in tandem with all other activities. – Activities should include HIV prevention and care, and support for people living with HIV/AIDS. – Vulnerable populations must be reached, with particular focus on the needs of women and children. – Test kits for HIV screening to ensure safe transfusions are needed and condoms must be available. – The establishment of voluntary counselling and testing (VCT) services should be considered when relative stability is restored.

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; told how and where to acquire free condoms and medical attention if necessary; and given information on basic hygiene. – <i>Condom promotion</i>, including ensuring the availability of good-quality condoms. – <i>STI management</i>, including for sex workers, using the syndromic STI management approach, with partner notification, treatment 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. – Avoidance of unintended pregnancies 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 invasive obstetric procedures, such as artificial rupture of membranes or episiotomy; and – modifying infant feeding practices (replacement feeding given with a cup when culturally acceptable, feasible, affordable, sustainable and safe; otherwise exclusive breastfeeding for the first six months of life is recommended). <p><u>Blood safety</u></p> <ul style="list-style-type: none"> – Avoidance of non-essential blood transfusion. – HIV testing of all transfused blood. – 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 safe 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. <p>Universal precautions</p> <ul style="list-style-type: none"> – Thorough hand-washing 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. Populations that are displaced may often be coerced into testing, when they are suffering acute or post-traumatic stress disorders.</p> <p>As refugees are often tested before resettlement in other countries, it is critical that they receive counselling on the legal and social implications of the test. Often, migration or temporary residence status is dependent on the applicant's having a seronegative (HIV antibody) 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 becomes an important part of post-test counselling in the context of a complex emergency.</p> <p>Orphaned minors should be tested only when 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 before 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 BCG or yellow fever vaccines.</p>

8. LASSA FEVER

DESCRIPTION

Infectious agent	Lassa virus (genus <i>Arenavirus</i> , family Arenaviridae)
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) 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 (VHFs), 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 complication 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>, <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 air 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 it is 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 known to be endemic in Liberia but no data are available on numbers of cases from recent years.</p> <p>The number of Lassa virus infections per year in west Africa is estimated at 300 cases/year – mostly in Sierra Leone – with approximately 1.5% mortality (2001 data). 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	Most cases have been reported in the western part of the country.
Seasonality	Disease rates peak from November to April (the dry season).
Alert threshold	One probable case must lead to an alert.
Recent epidemics	<p>A female patient originating from Ganta, Nimba county, who was highly suspected for Lassa fever died in 2001. No laboratory specimen was obtained.</p> <p>Epidemics in neighbouring Sierra Leone have been reported in recent years. Three outbreaks occurred between 1996 and 2000 in the Sierra Leonian district of Kenema, bordering with Liberia.</p> <p>The long conflict has disrupted disease surveillance and no reliable data are available on recent epidemics or numbers of cases.</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 seen among 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>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 public health problem in Sierra Leone and some outbreaks have occurred close to the Liberian border. The disease is also endemic in Guinea. There may be a risk of Lassa fever outbreaks in Liberia given the large population displacements between Sierra Leone and Liberia over the years of conflict in west Africa and the reported cases of Lassa fever among Liberian refugees in camps in Sierra Leone, who will one day return to Liberia.</p> <p>In addition, these populations are living in overcrowded settings with poor hygiene, poor sanitary conditions and with very limited access to health care in some areas.</p> <p>Hospitals in Liberia 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.</p> <p>Prevention activities can be implemented among all vulnerable populations, including IDP and refugee camps), by improving general sanitation in the area (proper waste and excreta management), improving access to safe water and introducing health education messages regarding safe water and food storage and hygiene measures.</p> <p>Rodent control activities are important in reducing the population of rats (thereby contributing to the control and prevention of the disease) even though it is impossible to eliminate them totally.</p>

PREVENTION AND CONTROL MEASURES

Case management	<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 IV 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 is not available; however, oral ribavirin therapy <i>may</i> be attempted when IV therapy is not feasible. General supportive treatment, as well as treatment of any other complicating infection, is also very important in the management of Lassa patients.</p> <p>Side-effects of ribavirin are restricted largely to reversible haemolysis. Plasma</p>
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	<p>transfusion has not shown to be beneficial during Lassa fever convalescence and is not recommended, especially because of the potential for transmitting other viruses such as HIV, HBV, and others.</p> <p><u>Intravenous ribavirin treatment</u></p> <p>The threshold number of cases at which IV 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 17 mg/kg IV (max 1g 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 (maximum, 500 mg per dose) every 8 hours for 6 days. <p>* If there is delay in starting treatment, a loading dose of 30 mg/kg (IV) (max. 2 g) may be necessary.</p> <p>Pregnant women</p> <p>Same as for adults. Ribavirin is generally 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. <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 foetuses of infected pregnant women, about 95% of which 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> <p>– “VHF outbreak control” in <i>Guidelines for Outbreak Control</i>, in this Toolkit.</p>
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	<ul style="list-style-type: none"> - <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, or when disposing of waste material, needles, or other sharp instruments.</p> <p>See:</p> <ul style="list-style-type: none"> - "Prevention" in Section 7, "HIV/AIDS". - Annex 8 in <i>Guidelines for Collection of Specimens for Laboratory Testing</i>, in this Toolkit.
<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. Though studies that have involved trapping and destruction of rodents (Trap-out studies) on a large scale have been effective in neighbouring 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 that rats cannot contaminate them. - Personal hygiene, environmental sanitation and hand-washing. - Elimination of rat habitats to minimize 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, etc.) during surgery or midwifery.</p>

9. 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 terms of 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 spp.</i> , also <i>Culex spp.</i>) 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 <i>microfilariae</i> (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	<p>Lymphatic filariasis is known to be endemic in most of the warm, humid regions of Africa. Liberia is included in the African tropical endemic region and up to 2.9 million of the country’s population may be considered as being at risk of lymphatic filariasis.</p> <p>Measuring the impact of disease control activities is very slow because of the long incubation periods and chronic course of the disease.</p>
Geographical distribution	Elephantiasis is more prevalent in the coastal counties, especially those in the south-east Liberia.
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.
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 cause by difficulties in accessing health services (geographical, financial, security) increases the risk of transmission.
Food shortages	No	
Lack of safe water and poor sanitation	Yes	Providing safe water is a means of secondary prevention (of disease progression, not of the infection) since it facilitates some of the hygiene measures recommended for affected body parts. Poor sanitation may contribute to creation of breeding sites for mosquito vectors (especially <i>Culex</i> spp.).
Others	Yes	There is an established link between the degree of poverty and the prevalence of lymphatic filariasis.
Risk assessment conclusions		The whole country was reported as endemic, but this needs to be confirmed through endemicity mapping. The complex emergency situation in Liberia is one of the reasons that this country has not so far been included in the Global Programme to Eliminate Lymphatic Filariasis (GPELF). Efforts to control lymphatic filariasis in Liberia may therefore be suboptimal, and the situation poses a risk for elimination of LF in neighbouring countries (Côte d'Ivoire, Guinea and Sierra Leone) as Liberia could represent a source of ongoing transmission. The introduction of GPELF in Liberia would provide benefits "beyond filariasis"; for example, albendazole is also an effective and safe drug for treating soil-transmitted helminth infections, and ivermectin is effective against many intestinal parasites, scabies and lice.

PREVENTION AND CONTROL MEASURES

Case management	<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 months if necessary. – Alternatively, ivermectin and albendazole can be used. Ivermectin, though very effective in reducing 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.
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<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 Liberia 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 test (ICT) card test. 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 the 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 given:</p> <p>Areas with concurrent onchocerciasis:</p> <ul style="list-style-type: none"> – Albendazole 400 mg + Ivermectin 150 µ/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>Reduce 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.
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	<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 carrying out other simple, low-cost procedures at home (see “Case management” for details).</p> <p>B. <u>Individual level</u></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.

10. MALARIA

DESCRIPTION

Infectious agent	In Liberia, <i>Plasmodium falciparum</i> accounts for more than 90% of all malaria infections. <i>P. malariae</i> and <i>P. ovale</i> , alone or mixed with <i>P. falciparum</i> , are responsible for the remaining malaria burden.
Clinical case definitions	<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 and 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 Liberia, children with fever and no general danger sign or stiff neck should be classified as having malaria. Although a substantial 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. In Liberia, the primary malaria vectors are <i>An. gambiae</i>. <i>An. melas</i> (usually only found in the coastal regions) and <i>An. funestus</i>.</p> <p>Malaria may also be transmitted by injection of infected blood.</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> . 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

Disease burden	<p>At least 90% of the population have no access to adequate health care services: most of the health services have broken down and only a third of the country is accessible to humanitarian agencies. Historical surveys have shown overall malaria prevalence rates of up to 80%.</p> <p>Data from the 1990s indicate that, among those who access health services, malaria is the leading cause of outpatient attendance (40–45%) and inpatient deaths. The under-five mortality is 235/1000, and at least 17.8% of these deaths are attributable to malaria (MCD, Routine Malaria Surveillance Data, 1993-1999). The overall prevalence rate of malaria has increased from 34.6% in 1987 to 50% in 2000 (LDHS, 2000).</p> <p>In 2003, malaria contributed 55% of the morbidity of diseases under surveillance in Liberia in August–November, according to weekly surveillance reports. The disease affects children disproportionately: in a survey of five clinics in Monrovia in November, 41% of malaria patients were children under 5 years of age, who make up an estimated 16% of the total population.</p> <p>Laboratory confirmation of diagnosis is rare. The quality of locally available antimalarial drugs is unknown.</p> <p><u>Antimalarial drug resistance</u></p> <p>Recent studies in Harper (south-eastern Liberia) by Checchi et al. (2002) showed:</p> <ul style="list-style-type: none"> – chloroquine 74.0% failure rate after 14 days of follow-up (95% confidence interval 59.7–85.4%) – sulfadoxine–pyrimethamine (SP) 48.5% failure rate (95% CI 36.2–61.0%) after 14 days of follow-up, and 51.5% (95% CI 38.9–64.0%) after 28 days (corrected by polymerase chain reaction to exclude reinfections) – amodiaquine 19.8% failure rate in 28-day test (95% CI 11.7–30.1%), corrected by polymerase chain reaction genotyping to distinguish recrudescence from reinfection.
Geographical distribution	Risk of transmission is high in the whole country, including cities.
Seasonality	The climate is tropical, hot and humid, with ideal temperatures throughout the year for malaria transmission. A true rainy season runs from May to October, with some rains occurring throughout the year. Depending upon the amount of rainfall, malaria transmission may show some seasonal variation in the forest/savannah, but slight or no seasonal variation in coastal areas. A study conducted in 1995 in the Mount Nimba region, close to the border with Côte d'Ivoire and Guinea, found that mosquito densities were seasonal, with 0.5 infectious bites per person per night during the rainy season and 0.02 infectious bites per person per night late in the dry season.
Alert threshold	Not strictly applicable as malaria is highly endemic throughout the country and data from previous years are limited, inaccurate and/or irrelevant to the current situation. Nevertheless, reasons for concern include: <ul style="list-style-type: none"> – case numbers are very much higher than at the same time in the previous year; – case numbers rising very rapidly in the past few (2–4) weeks; – a high case–fatality rate from malaria-like illness; – a rising slide positivity rate in adults with symptoms of malaria; and/or – unusually high consumption of antimalarial drugs in the past 2 weeks.
Recent epidemics	No data available.

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Mass population movement results in increased vulnerability of displaced populations because of malnutrition, concomitant diseases, settlement in marginal areas close to mosquito breeding sites, housing in temporary shelters with increased exposure to mosquito bites, increased population density promoting malaria transmission.
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 malaria burden.
Risk assessment conclusions		<p>Malaria is hyperendemic in Liberia and transmitted throughout the year. Despite the fact that Liberia has reduced tariffs and taxes on nets and insecticides and has established a malaria steering committee, malaria control remains difficult given the massive population displacement, potential for ongoing conflict, lack of regular supply of medicines and diagnostics, and the increasing resistance of <i>P. falciparum</i> to the commonly used antimalarial drugs.</p> <p>According to surveillance data (which are limited by the lack of access to health care in many counties), malaria is the leading cause of outpatient attendance (40–45%) and mortality.</p> <p>Priority activities for malaria control in Liberia include:</p> <ul style="list-style-type: none"> – studies on the current level of resistance to antimalarial drugs in the country, followed by update and standardization of the national treatment policy; – making the malaria surveillance system functional nationwide, encouraging laboratory confirmation of cases, and integrating this data source into the national surveillance system to increase the number of <i>confirmed</i> malaria cases reported; – implementing appropriate and sustainable vector control activities.

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: chloroquine (first line) – Treatment failure: sulfadoxine–pyrimethamine (second line) – Severe malaria: quinine. <p>In light of the reported resistance of <i>P. falciparum</i> to currently recommended drugs (chloroquine and SP), alternative first-line antimalarial regimens would be either artemether–lumefantrine (Coartem®) or another artemisinin-based combination therapy. A review of the national treatment policy review is under way.</p> <p>Where the operational situation does not allow for continuous, high-quality nursing care, artemether, IM once daily, is preferable to IV quinine for the treatment of severe malaria.</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 be requested from the manufacturer before purchase.</p>
<p>Prevention and control</p>	<p>Intermittent preventive treatment (IPT) with SP during pregnancy has recently been approved for use in the country.</p> <p>No vector control strategy has been developed so far in Liberia, but there has been some distribution of insecticide-treated mosquito nets (ITNs). The proportion of households with pregnant women and children under 5 years of age protected with vector control methods such as indoor residual spraying and ITNs is unknown but expected to be very low. The proportion of households where pregnant women and children under 5 years actually sleep under ITNs is also unknown. Other methods of vector control, such as insecticide-treated plastic sheeting for temporary shelters, are still under evaluation.</p> <p>Controlling malaria through ensuring a clean environment has been a priority message advocated by many NGOs through community workers in IDP camps. It is <u>not</u> recommended by WHO as a priority measure for the control of malaria.</p>

11. MEASLES

DESCRIPTION

Infectious agent	Measles virus (genus <i>Morbillivirus</i> , family Paramyxoviridae)
Case definition	<p><u>Clinical case definition</u></p> <p>Any person with: Fever and Maculopapular (i.e. non-vesicular) rash, and Cough or coryza (i.e. runny nose) or conjunctivitis (i.e. red eyes). or Any person in whom a clinical health worker suspects measles infection</p> <p><u>Laboratory criteria</u></p> <p>Presence of measles-specific IgM antibodies.</p> <p><u>Case classification</u></p> <p>Clinically-confirmed: case that meets the clinical case definition Laboratory-confirmed (only for outbreak confirmation and during the outbreak prevention/elimination phase):</p> <ul style="list-style-type: none"> – case that meets the clinical case definition and is laboratory-confirmed or – case meeting clinical definition and epidemiologically linked by direct contact to a laboratory-confirmed case in which rash onset occurred 7–18 days earlier.
Mode of transmission	Airborne by droplet spread; or Direct contact with the nasal and throat secretions of infected persons or via objects (e.g. toys) that have been in close contact with an infected person.
Incubation	After infection there is an asymptomatic incubation period of 10–12 days, with a range of 7 to 18 days from exposure to the onset of fever
Period of communicability	Measles is most infectious from 4 days before the rash until 1–2 days after the onset of rash.

EPIDEMIOLOGY

Burden	<p>Number of cases reported :</p> <p>2002: 131 2001: 1379 2000: 5977 1999: 1679 1998: 1436 1997: 2961 1990: no data 1980: no data</p> <p>These details do not reflect the full extent of cases as reporting has been irregular and security has prevented access to health facilities – the only reporting sites.</p>
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Geographical distribution	Measles is highly endemic throughout the region and the expected number of measles cases is high.
Seasonality	Higher incidence during the colder months .
Alert threshold	One case must lead to an alert. Laboratory confirmation of all cases is not required. Only a few cases from each outbreak need be laboratory-confirmed.
Recent epidemics	No data are available about recent measles epidemics in Liberia; however, an outbreak was reported in the bordering Kailahun district of Sierra Leone in June–July 2003.

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Facilitates transmission between refugees, displaced people and non-immune communities.
Overcrowding	Yes	Crowded conditions facilitate transmission.
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 in the host area.

Risk assessment conclusions	<p>There is a great risk of measles in Liberia as immunization coverage has been low in past years:</p> <p><u>MCV (measles-containing vaccine) coverage:</u></p> <table> <tr> <td>2002: 57%</td> <td>1998: 31.0%</td> </tr> <tr> <td>2001: 78%</td> <td>1997: no data available</td> </tr> <tr> <td>2000: 52%</td> <td>1990: no data available</td> </tr> <tr> <td>1999: 34.9%</td> <td>1980: no data available</td> </tr> </table> <p>(Figures for some counties are unknown or even lower because of conflict-related inaccessibility).</p> <p>In some areas, especially in north-western Liberia, there has been no immunization for more than 3 years.</p> <p>Displaced populations, with malnourished children living in overcrowded settings and poor sanitary conditions where measles has the potential to spread rapidly, are at higher risk of measles outbreaks. To address this problem, UNICEF, WHO, the Liberian MoH and NGOs are carrying out emergency measles vaccination campaigns. On 29 August 2003, UNICEF/WHO Liberia launched the first mass measles immunization campaign following the cessation of hostilities in Bomi County.</p> <p>A second campaign was carried out in Grand Cape Mount and Gbarpolu counties. The aim is to immunize children aged 6 months to 15 years (in IDP/refugee camps as well as among communities) against measles and to provide vitamin A supplements. UNICEF/WHO will be expanding the emergency measles immunization campaigns as access to more parts of the country becomes possible.</p> <p>Between August and December 2003, close to 630 000 children were immunized against measles.</p>	2002: 57%	1998: 31.0%	2001: 78%	1997: no data available	2000: 52%	1990: no data available	1999: 34.9%	1980: no data available
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PREVENTION AND CONTROL MEASURES

<p>Introduction</p>	<p>National EPI policy in Liberia: single dose at 9 months. Supplementary measles immunization campaigns are planned (2003 onwards).</p>
<p>Immunization in emergency and post-emergency phases</p>	<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 in this age group.</p> <p>Children who are vaccinated against measles before 9 months of age must receive a second measles vaccination 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 of age should also receive prophylactic vitamin A supplementation. If there is evidence of clinical vitamin A deficiency in older age groups, treatment with vitamin A should be initiated in accordance with 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>

<p>Outbreak response</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 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 years)* 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. – Isolation is not indicated, and children should not be withdrawn from feeding programmes. <p>*This range can be reduced (e.g. 6 months to 12 years or 6 months to 5 years) if resources are limited.</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). – A third dose of vitamin A should be given 2 weeks later. – 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 signs,* 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 2 weeks later. <p>* Inability to drink or breastfeed, vomiting everything, convulsions, lethargy or unconsciousness.</p>

12. MENINGOCOCCAL DISEASE (MENINGITIS AND MENINGOCOCCAL SEPTICAEMIA)

DESCRIPTION

Infectious agent	Bacterium: <i>Neisseria meningitides</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 until 24 hours after the institution of therapy, but the most important source of infection is asymptomatic carriers.

EPIDEMIOLOGY

Burden	2003: 799 cases reported (weeks 1 to 25). 2002: data unavailable 2001: data unavailable 1998: 111 cases reported 1999: 114 cases reported
Geographical distribution	Not known.
Seasonality	Not known.

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 in 1 week or – the number of cases in a 3-week period doubles 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 have been established from data in meningitis belt countries and have not been validated in countries outside the belt.</p>
Recent epidemics	No data available.

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	A 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.
Poor access to health services	Yes	Case identification is crucial for the rapid implementation of control measures. Case-fatality rate in the absence of treatment can be very high (50%).
Food shortages	No	
Lack of safe water and poor sanitation	No	

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

Others	No	Concurrent upper respiratory tract infections may increase the risk of transmission and help to propagate outbreaks. Dry and windy/dusty seasons increase transmission of the disease.
Risk assessment conclusions		Liberia is not situated in the African meningitis belt and large outbreaks are therefore not expected. However population displacement, with consequent overcrowding and high incidence of respiratory infections, makes the occurrence of epidemics more likely. Moreover, the increasing incidence of meningococcal epidemics outside the meningitis belt (Burundi, Rwanda, United Republic of Tanzania in 2002) in recent years is worrying.

PREVENTION AND CONTROL MEASURES

Case management	<p>Meningococcal disease (either meningitis or septicaemia) is potentially fatal and should always be considered as a medical emergency.</p> <p>Non-epidemic conditions</p> <ul style="list-style-type: none"> Admission to a hospital or health centre is necessary for diagnosis (lumbar puncture and CSF examination). Lumbar puncture must be done as soon as meningitis is suspected, before antimicrobial therapy is started. As infectivity of patients is moderate and disappears quickly following antimicrobial treatment, isolation of the patient is not necessary. Antimicrobial therapy must be instituted as soon as possible after lumbar puncture (without waiting for laboratory results), and should be combined with supportive treatment. Initial antimicrobial therapy should be effective against the three major causes of bacterial meningitis until bacteriological results and sensitivity are available: <table border="1"> <thead> <tr> <th>Age group</th> <th>Probable pathogens</th> <th>Antibiotic, first choice</th> <th>Alternative therapy</th> </tr> </thead> <tbody> <tr> <td>Adults</td> <td><i>S. pneumoniae</i></td> <td>Benzylpenicillin</td> <td>Ampicillin or Amoxicillin</td> </tr> <tr> <td>Children >5 yrs</td> <td></td> <td></td> <td>Chloramphenicol Ceftriaxone or cefotaxime</td> </tr> <tr> <td>Children 1 month – 5 years</td> <td><i>H. influenzae</i> <i>S. pneumoniae</i> <i>N. meningitidis</i></td> <td>Ampicillin or amoxicillin^a</td> <td>Ceftriaxone or Cefotaxime</td> </tr> <tr> <td>Neonates (<1 month)</td> <td>Gram-negative bacteria Group B <i>streptococci</i> <i>Listeria</i></td> <td>Ampicillin and gentamicin</td> <td>Ceftriaxone or cefotaxime^b Chloramphenicol (at reduced doses)</td> </tr> </tbody> </table> <p>^aIf <i>H. influenzae</i> is highly resistant to ampicillin, chloramphenicol should be given with ampicillin.</p> <p>^bNo effect on <i>Listeria</i>.</p> <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. 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 is also effective. <p>Epidemic conditions</p>			Age group	Probable pathogens	Antibiotic, first choice	Alternative therapy	Adults	<i>S. pneumoniae</i>	Benzylpenicillin	Ampicillin or Amoxicillin	Children >5 yrs			Chloramphenicol Ceftriaxone or cefotaxime	Children 1 month – 5 years	<i>H. influenzae</i> <i>S. pneumoniae</i> <i>N. meningitidis</i>	Ampicillin or amoxicillin ^a	Ceftriaxone or Cefotaxime	Neonates (<1 month)	Gram-negative bacteria Group B <i>streptococci</i> <i>Listeria</i>	Ampicillin and gentamicin	Ceftriaxone or cefotaxime ^b Chloramphenicol (at reduced doses)
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	<p>During epidemics of confirmed meningococcal disease, case management needs to be simplified to permit the health system to respond to rapidly expanding 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 <i>orally chloramphenicol</i> (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> <ul style="list-style-type: none"> • 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. • 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: <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 any of the W135 meningococcal polysaccharide-containing vaccines available (trivalent ACW or tetravalent ACWY if serogroup 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), trivalent (ACW) or tetravalent (A/C/Y/W135) vaccine. At risk populations (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 (from epidemiological data of age groups affected, for example). <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.

13. MONKEYPOX

DESCRIPTION

Infectious agent	Monkeypox virus (MPV), of the orthopoxvirus group.
Case definition [Case definition and case classification may vary according to circumstances locally identified in the field.]	<p><u>Clinical case definition</u></p> <p>The disease is very similar to smallpox (pustular rash, fever, respiratory symptoms), except for marked lymphadenopathy (sometimes only in the neck or inguinal region, but more often generalized), often observed at the time of onset of fever, usually 1–3 days before the appearance of rash. Monkeypox is clinically milder than smallpox.</p> <p><u>Laboratory criteria</u></p> <p>Virus isolation in lesion material (PCR, antigen capture) or serology (ELISA, Western blot). Must be correlated with clinical and epidemiological data.</p> <p><u>Case classification</u></p> <p>Confirmed case: based on the results of analysis of biological specimens and correlation with clinical data.</p> <p>Suspected case: the occurrence, in a resident in the outbreak zone, of fever and a vesicular-pustular rash similar to a WHO reference photograph.</p> <p>The latter case definition is highly sensitive and therefore likely to include cases with other causes of an illness with vesicular rash such as varicella-zoster.</p>
Mode of transmission	<p>Monkeypox is a rare sporadic zoonosis that is enzootic among mammals including monkeys and squirrels in the rainforests of central and west Africa.</p> <p>Both animal-to-person (non-human primates and squirrels seem to be the most important reservoir species) and person-to-person transmission occurs. Animal contact (through handling or eating dead monkeys or squirrels) is the most probable mode of transmission to humans. The exact means of transmission is unclear and still needs to be determined.</p> <p>Person-to-person transmission is believed to occur primarily through direct contact and also through respiratory droplet spread. Transmission of monkeypox within hospitals has been described, but rarely. There is no evidence to date that person-to-person transmission alone can sustain monkeypox in the human population.</p>
Incubation	12 days (range 7–21 days)
Period of communicability	For humans, may be from 1 day before the onset of rash up to 21 days after the onset of rash or illness, or when all rash lesions have scabbed over.

EPIDEMIOLOGY

Burden	<p>Most cases occur in remote villages of central and west Africa, close to tropical rainforests where there is frequent contact with infected animals. In Liberia, four cases of human monkeypox were reported between September 1970 and May 1971; two were laboratory-confirmation and two were epidemiologically-linked.</p> <p>Human monkeypox, although sporadic, is a life-threatening disease.</p>
Geographical distribution	Most cases were found in Grand Gedeh county, and one in the Agebu region.
Seasonality	Unknown.
Alert threshold	<p>One confirmed case must lead to alert.</p> <p>See also outbreak control section in <i>Communicable disease control in emergencies – a field manual</i>. Geneva, WHO (in press).</p>

Recent epidemics	No cases have been officially reported since 1971. Outbreaks of monkeypox and VZV (chickenpox) can occur concomitantly (co-circulation and/or co-infection with VZV, chickenpox virus).
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RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Displaced populations moving to endemic areas are at risk.
Overcrowding	Yes	Contact with an infected individual may increase the possibility of human-to-human transmission.
Poor access to health services	Yes	Collapse of general health infrastructure and disease surveillance activities.
Food shortages	No	
Lack of safe water and poor sanitation	No	
Others	Yes	Contact with wild animals. Cases occur mainly among villagers who are engaged at least part-time as hunters or gatherers. Civil unrest and economic collapse may result in more frequent and deeper penetration of people into rainforests in search of food, thus increasing the risk of contact with animal reservoirs of the monkeypox virus.
Risk assessment conclusions		The four cases reported in Liberia in early 1970s were among the first reported cases of monkeypox in west Africa. No cases have been officially reported since 1971. However, the disease may still be a threat in Liberia in view not only of the years of civil war that have resulted in disruption of health services and disease surveillance but also of the massive displacement of populations in the region. Vaccinia (smallpox) vaccination ceased in 1983 after global smallpox eradication. This has led to an increase in the numbers of people susceptible to monkeypox and an increased risk of monkeypox outbreaks (since the virus will continue to be introduced into human communities from animal sources).

PREVENTION AND CONTROL MEASURES

Case management	Symptomatic treatment is the mainstay. Measures to avoid secondary infection of pustules (hygiene and antibiotics) may be necessary. Cidofovir [(S)-1-(3-hydroxy-2-phosphonylmethoxypropyl)cytosine, HPMPIC], is the first promising anti-orthopoxviral drug. It has been licensed since 1996 for clinical use in the treatment of cytomegalovirus (CMV) retinitis in AIDS patients. Laboratory data show that cidofovir should be effective in the therapy of monkeypox in humans. Cidofovir has significant toxicity and should be considered only for the treatment of severe monkeypox infections and not for prophylactic use.
Epidemic control	Epidemiological surveillance is key in controlling the disease. Early detection and notification of cases allow for timely mobilization of resources needed for epidemic control. See <i>Guidelines for Outbreak Control</i> in this Toolkit for Liberia.
Prevention	Vaccination of personnel at risk should be considered. Vaccinia vaccine is about

	<p>85% effective in preventing human monkeypox.</p> <p>Vaccination is contraindicated if there is immunosuppression (e.g, in HIV/AIDS).</p> <p>Direct and prolonged contact with possible reservoir animals in endemic areas should be avoided.</p>
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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 description</u></p> <p>In an endemic area, any individual presenting with fibrous nodules in subcutaneous tissues. These must be distinguished from lymph nodes or ganglia. Persons suffering from onchocerciasis may experience:</p> <ul style="list-style-type: none"> • Skin lesions Dermal changes are secondary to tissue reaction to motile larvae as they migrate subcutaneously, or due 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, this may persist as the only symptom. – Rashes The rash usually consists of many raised papules, 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 chambers of the eye can occur. The more serious lesions lead to significant 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”. Adults 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 pathological 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

<p>Burden</p>	<p>The African Programme for Onchocerciasis Control (APOC) estimated the total infected population as 600 000 in 1996.</p> <p>Rapid Epidemiological Mapping of Onchocerciasis (REMO) in Liberia, from 26 December 1998 to 9 February 1999, showed that human onchocerciasis is widespread and present in virtually all counties in the country, though at different levels of endemicity. Similar observations have come from other sources (e.g. Burch, 1955; Gratama, 1966; Frentzel-Beyme, 1975).</p> <p>In the south of Liberia, towards the Atlantic coast, onchocerciasis intensity and endemicity are lower. Few cases of onchocercal nodules were observed in coastal or near-coastal communities during the REMO assessment. The sluggish, swampy, salty and polluted water in this zone is not suitable for breeding of the vector flies and hence transmission does not occur.</p>
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Geographical distribution	In the REMO assessment (see “Burden”, above), the distribution and endemicity levels of onchocerciasis in Liberia did not coincide with any administrative or political boundaries in the country. Rather, they were governed by the location of major rivers and tributaries (the breeding sites of vectors).
Seasonality	Vector breeding and disease transmission are perennial in locations where there rivers that are fast-flowing throughout the year.
Recent epidemics	Not applicable

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Migration can lead to the establishment of new foci
Overcrowding	Yes	Increases 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 some 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, are associated with increased risk of exposure to blackfly bites in forest areas.
Risk assessment conclusions		<p>Onchocerciasis (a common cause of blindness) is endemic in Liberia and has a widespread distribution in all 15 counties.</p> <p>Given the security situation in Liberia over the past decade, it is likely that the prevalence of onchocerciasis has increased as a result of difficulties in implementing and sustaining control programmes. In addition, displaced populations may have been exposed to the vector when fishing or farming in areas of high risk.</p> <p>As the security situation and geographical access improve throughout Liberia, a comprehensive REMO assessment of onchocerciasis endemicity needs to be undertaken, particularly to identify communities likely to be at highest risk. These communities would then need to be screened for clinical signs of the disease.</p> <p>This epidemiological assessment forms the basis of any national control programme and is recommended to all countries by APOC (see “Prevention” below).</p>

PREVENTION AND CONTROL MEASURES

<p>Case management</p>	<p>Administration of ivermectin once a year over a period of at least 15–20 years significantly reduces infection and prevents the appearance of clinical manifestations. The recommended dosage is 150 µg/kg body weight. 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 of 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, started in 1996 to cover 19 African countries including Liberia, 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 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., 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 were in the OCP area.</p> <p>APOC administers ivermectin to communities in high-risk areas as determined by REMO assessment and through use of geographical information systems (GIS). Continued annual distribution of ivermectin will control onchocerciasis to the 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 of 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. The initial catarrhal stage 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. 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	Average 9–10 days (range 6–20 days).
Period of communicability	Highly communicable in the early catarrhal stage and at the beginning of the paroxysmal cough stage (first 2 weeks). Thereafter, communicability gradually decreases and becomes negligible in about 3 weeks, despite persisting spasmodic cough with whoop. Untreated patients may be contagious for up to 3 weeks after the onset of paroxysmal cough in the absence of treatment, or for up to 5 days after onset of treatment.

EPIDEMIOLOGY

Burden	Number of cases reported: 2002: 379 2001: 131 2000: 281 1999: 394 1998: 1114 1990: no cases reported 1980: 62 These are likely to be underestimations because of the limited and irregular surveillance activities during years of conflict.
Geographical distribution	Pertussis is an endemic disease common to children everywhere, regardless of ethnicity, climate or geographical location.
Seasonality	Pertussis activity may increase in the summer and autumn.
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	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	No	

<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 they have 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), plus lack of access to health services in many areas combined with overcrowded living conditions put the Liberian population at high risk for this disease.</p> <p>DTP3 coverage: (global summary statistics WHO/IVB). 2002: 51% 2001: 62% 2000: 55% Most recent coverage survey, 1999: 44% (UNICEF/WHO estimates).</p> <p>To reduce disease risk, it is essential to improve access to health services and increase 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>	<ul style="list-style-type: none"> • 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. • Drug administration both modifies the course of illness (if initiated early), and eradicates the organism from secretions, thereby reducing communicability but does not reduce symptoms except when given during the catarrhal stage or early in the paroxysmal stage. • Symptomatic treatment and supportive case-management are important.
<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 Liberia.</p> <p>In general, pertussis vaccine (wP) is not given to persons aged 7 years or over, 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>

Epidemic control	<ul style="list-style-type: none">• The highly contagious nature of pertussis leads to large numbers of secondary cases among non-immune contacts. Prophylactic antibiotic treatment (with erythromycin) in the early incubation period may prevent disease, is limited to selected individual cases because of the difficulties of early diagnosis, the costs involved and concerns about the occurrence of drug resistance.• Priority must be given to:<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.• The strategy relies on chemoprophylaxis of contacts within a maximum delay of 14 days following the first contact with the index case.• 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.• All contact cases must have their immunization status verified and brought up to date.
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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 nonspecific 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 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.

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 – antispasmodic 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 only by 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 IPV.</p> <p><u>Immunization policy in Liberia</u></p> <p>Liberia has a routine immunization policy that requires four doses of OPV (see Annex 7). However, supplementary immunization activities are also carried out 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 low transmission season for enteroviruses (cooler season).</p> <p>Supplementary immunization activities in Liberia</p> <p>1999: 2 rounds of NIDs 2000: 5 rounds of NIDs 2001: 2 rounds of NIDs 2002: 2 rounds of NIDs and 2 rounds of sub-NIDs (synchronized with all countries in the west African block) 2003: no supplementary immunization activities</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 a suspected outbreak:</p> <p><u>Investigation</u></p> <ul style="list-style-type: none">- Clinical and epidemiological investigation.- Rapid virological investigation (2 stool samples taken 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> <ul style="list-style-type: none"> • Paresis or paralysis, delirium, convulsions. • Without medical attention, death in about 6 days, usually due to respiratory paralysis. <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 by 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. • Specific FA staining of frozen skin sections from the back of the neck at the hairline • Isolation of rabies virus from clinical specimens and confirmation of rabies viral antigens. <p><u>Case classification</u></p> <ul style="list-style-type: none"> • Human rabies Suspected: A case that is compatible with the clinical case definition. Probable: A suspected case plus history of contact with a suspected rabid animal. Confirmed: A suspected case that is laboratory-confirmed. • Human exposure to rabies 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. Exposed: A person who had close contact – usually a bite or a scratch – with a laboratory-confirmed rabid animal.
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 (rarely more than 4 days) before onset of clinical signs and throughout the course of the disease. Longer periods of excretion before onset of clinical signs have been observed in other animals.

EPIDEMIOLOGY

Burden	Two cases of human rabies were reported in 1992.
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 outbreaks have been reported.

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 (food scraps; poor environmental sanitation and waste disposal). Children aged 5–15 years are the major group at risk.
Risk assessment conclusions		The lack of information on incidence of rabies in Liberia is a result of limited and irregular surveillance during years of conflict and insecurity. Rabies is endemic in Liberia as it is 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. Surveillance of both human and animal rabies is essential to detect high-risk areas and outbreaks quickly.

PREVENTION AND CONTROL MEASURES

<p>Case management</p>	<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 given for Category III exposures only.</p> <p>Suturing should be postponed if possible; if it is necessary, immunoglobulin must be applied first. Where indicated, anti-tetanus treatment and antimicrobials 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" data-bbox="410 703 1302 1291"> <thead> <tr> <th data-bbox="410 703 698 766">Category</th> <th data-bbox="706 703 1006 766">Type of contact with suspected animal</th> <th data-bbox="1015 703 1302 766">Recommended treatment</th> </tr> </thead> <tbody> <tr> <td data-bbox="410 777 698 882">I</td> <td data-bbox="706 777 1006 882">Touching or feeding an animal Licks on intact skin</td> <td data-bbox="1015 777 1302 882">None, if reliable case history is available</td> </tr> <tr> <td data-bbox="410 892 698 1102">II</td> <td data-bbox="706 892 1006 1102">Nibbling of uncovered skin Minor scratches or abrasions without bleeding Licks on broken skin</td> <td data-bbox="1015 892 1302 1102">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="410 1113 698 1291">III</td> <td data-bbox="706 1113 1006 1291">Single or multiple trans-dermal bites or scratches Contamination of mucous membrane with saliva</td> <td data-bbox="1015 1113 1302 1291">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 trans-dermal 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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<p>Epidemic control</p>	<p>Immediate notification if one or more suspected cases are identified.</p> <p>Confirm the outbreak, following WHO guidelines.</p> <p>Confirm diagnosis and insure prompt management.</p>												
<p>Prevention</p>	<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 for increased availability of modern rabies vaccine to susceptible populations – elimination of dog rabies through mass vaccination of dogs and dog population management. 												
<p>Immunization</p>	<p>Preventive mass vaccination in humans is generally not recommended but can be considered under certain circumstances for the 5–15-year age group.</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 Liberia.</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 possible 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 (for <i>S. haematobium</i>) or faeces (for <i>S. mansoni</i>) of patients with chronic schistosomiasis into a body of freshwater.</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>
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 absent in <i>S. haematobium</i> infection).</p> <p>From 3 months to several years: chronic illness manifestations.</p>
Period of communicability	<p>As long as eggs are discharged by patients. This may be from 10–12 weeks to more than 10 years after infection.</p>

EPIDEMIOLOGY

Burden	Human infection with <i>S. haematobium</i> and/or <i>S. mansoni</i> is known to be widespread in central Liberia (Lofa, Bong and Nimba counties). Prevalence data are not available.
Geographical distribution	Central Liberia, especially around paddy fields. A south-to-north stratification of schistosomal infection prevalence has been observed, similar to the west-to-east gradient reported by Saladin et al. (1980).
Seasonality	In a study in Liberia in 1980, the snail population density and infection rate were shown to fluctuate with the season, being higher in the dry season and lower during periods of heavy rainfall. More water sites contained infected snails during December to February than at any other time of the year. When water levels are falling, numerous marshes and ponds tend to form in place of streams. Dry periods tend to increase transmission of the disease because of the higher densities of cercariae in these 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 or school treatment would 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 previously endemic only for the urinary form (and vice versa).
Overcrowding	Yes	Higher human densities increase the chance of snails being penetrated and colonized by the larvae (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		Both <i>S. haematobium</i> and <i>S. mansoni</i> are endemic in Liberia. Given years of population displacement, overcrowding and poor access to health services, schistosomiasis is likely to be highly prevalent in many areas. Control of schistosomiasis should be a priority because of the effect of this disease both on the general health status of infected individuals and in increasing the severity of concomitant infections. However, no large-scale programmes are currently implemented in Liberia. Schistosomiasis control should be built into case-management algorithms in primary health care services and into any health packages delivered through schools.

PREVENTION AND CONTROL MEASURES

Case management	Praziquantel is the drug of choice for all schistosomiasis parasites. A single oral dose of 40 mg/kg is generally sufficient to give cure rates of 80–90% and dramatic reductions in the average number of eggs excreted. 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.
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	A dose pole (for calculating dosage according to height) is available to facilitate the delivery of praziquantel in schools or for community-based delivery.								
Prevention	<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 habitat and snail contact (in irrigation and agriculture practices), environmental management. • Treatment of snail-breeding sites with molluscicide (if costs permit). <table border="0"> <thead> <tr> <th style="text-align: left;">Community category</th> <th style="text-align: left;">Prevalence</th> </tr> </thead> <tbody> <tr> <td>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>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>III (Low prevalence)</td> <td><10% infected (<i>S. haematobium</i>, <i>S. mansoni</i>, by parasitological methods).</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)	<10% infected (<i>S. haematobium</i> , <i>S. mansoni</i> , by parasitological methods).
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	<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 freshwater.</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).</p> <p>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 HELMINTHIASIS (ASCARIASIS, HOOKWORM INFECTION, TRICHURIASIS)

DESCRIPTION

Infectious agent	Helminths: <i>Ascaris lumbricoides</i> , hookworms (<i>N. americanus</i>), <i>Trichuris trichiura</i>
Case definition	<p>Ascariasis</p> <p>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</p> <p>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</p> <p>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>: non-specific</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	<p><i>A. lumbricoides</i>: unknown Hookworm (<i>N. americanus</i>): unknown <i>T. trichiura</i>: unknown</p> <p>Since most helminths (<i>A. lumbricoides</i>, <i>N. americanus</i>, <i>T. trichiura</i>) are transmitted by the faecal–oral route, prevalence rates of infection are higher in places where hygienic practices are not followed and especially where there are poor sanitary conditions, e.g. latrines not used, inadequate excreta disposal.</p> <p>Large population displacement results in a high prevalence of these parasites not only as a result of overcrowding and lack of safe water, but also because good hygiene practices are less likely in such circumstances.</p>
Geographical distribution	Soil-transmitted helminths are thought to be 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.
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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 and transmission cycle is therefore perpetuated.
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 improve the quality of life of affected populations.</p> <p>No large-scale programmes for the control of soil-transmitted helminths are currently implemented in Liberia.</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 means that control activities for intestinal helminth infections can represent a starting point for the reconstruction of health care systems in such countries.</p>

PREVENTION AND CONTROL MEASURES

<p>Case management</p>	<p>All soil-transmitted helminthiases (STH) compete with the host for nutrients, causing malabsorption of fats, proteins, carbohydrates and vitamins, and contributing directly 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. Measles infection 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>												
<p>Prevention and control</p>	<p>Personal hygiene and hand-washing, appropriate disposal of faeces, and clean food</p> <p>Improvements in sanitation standards (see Annex 3, “Safe water and sanitation”).</p> <p>Community diagnosis (through primary school surveys) and community-wide treatment regimen for STH according to the following categories:</p> <table border="1" data-bbox="410 1333 1310 1732"> <thead> <tr> <th>Community category</th> <th>Prevalence of any infection</th> <th>% of moderate- to heavy-intensity infections</th> </tr> </thead> <tbody> <tr> <td>I High prevalence, high intensity</td> <td>≥70%</td> <td>≥10%</td> </tr> <tr> <td>II High prevalence, low intensity</td> <td>≥50% but <70%</td> <td><10%</td> </tr> <tr> <td>III Low prevalence, low intensity</td> <td><50%</td> <td><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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<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

Diagnosis in adults	<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 2 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 three 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 taken 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 day.</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>At least two sputum smears are positive</p>
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	<p>Smears should be stained using the Ziehl-Neelsen method. Any TB suspect with 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 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 positive 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 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 co-trimoxazole, but not rifampicin or any anti-TB drug) for at least one week. If there is no improvement, sputum samples must be re-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 a diagnostic 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 a patient is categorized 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 from TB infection to disease. 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 and to start treatment without delay based on clinical, bacteriological and, in some circumstances, radiological evidence.</p>
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<p>Diagnosis in children</p>	<p>TB in children is a general disease, which may affect any part of the body. Children rarely have smear-positive TB, so they are rarely infectious. In complex emergency situations with a large number of children, extrapulmonary forms of TB should be suspected, diagnosed and treated appropriately. This may often require referral to a hospital for X-ray and special examinations (e.g. lumbar puncture).</p> <p>In children with headache, change of temperament, recent squint or ocular muscle paralysis, or dyspnoea, meningitis should be suspected. TB is one cause of meningitis, although rare – meningococcal meningitis is more common in complex emergency settings. Definitive diagnosis requires hospital referral.</p> <p>Children with high fevers, dyspnoea, gastrointestinal symptoms, confusion (i.e. those with suspicion of acute miliary TB) must also be referred to hospital for assessment and diagnosis. Suspected bone and joint TB, or pleural effusions also require referral.</p> <p>Commoner forms of extrapulmonary disease (e.g. cervical or auxiliary lymphadenitis, peritonitis with ascites) can be diagnosed and treated in a camp situation.</p> <p>The diagnosis of TB in children should be carefully considered in a child if there is:</p> <ul style="list-style-type: none"> – illness lasting for more than 10 days – history of close contact with a TB patient – poor response to antibiotic therapy – poor response to one month of nutritional rehabilitation – weight loss or abnormally slow growth – loss of energy, or – increasing irritability and drowsiness over 2 weeks. <p>A score sheet has been developed to improve the diagnosis of childhood TB (see chart, p. 40, in <i>Tuberculosis control in refugee situations: an inter-agency field manual</i>. Geneva, WHO, 1997 (WHO/TB/97.221)). According to the score obtained, either the child is classified as having TB, with appropriate treatment being recommended, or a 7-day course of antibiotics is recommended (which is repeated if there is no clinical improvement). If there has been no improvement when response is assessed after the second week of antibiotics, anti-TB treatment is recommended.</p> <p>Nutritional support and rehabilitation should be given for at least 1 month to a child in whom TB is suspected.</p> <p>Note: The considerations explained above for the diagnosis of TB in HIV-positive adults also apply in to children.</p>
<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;</p> <p>or: A patient with at least one sputum specimen positive for AFB by microscopy and radiographic abnormalities consistent with pulmonary TB;</p> <p>or: A patient with at least one sputum specimen positive for AFB by microscopy, which is culture-positive for <i>M. tuberculosis</i>.</p>

	<ul style="list-style-type: none"> • 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 to have smear-negative pulmonary TB.</p> <p>Extrapulmonary tuberculosis (EPTB)</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 on 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 axially 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>
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EPIDEMIOLOGY

Burden	<p>Estimated TB in Liberia, 2001 (WHO Global Tuberculosis Control Report, 2003): New sputum smear-positive (ss+) cases: 3426 (110/100 000 population) Total cases: 7800 (250/100 000 population) No data available on DOTS.</p> <p>WHO mission report, produced in October 2000: New smear-positive (ss+) cases: 1693 New smear-negative cases: 477 Extrapulmonary cases: 292 Total cases: 2462</p>
Geographical distribution	Even if specific data are not available, tuberculosis 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, leading 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 increase transmission.

Poor access to health services	Yes	People affected by TB who cannot access health services and be treated, remain infectious for a longer period. The case fatality rate is high (about 50%) without proper treatment. The interruption of treatment is the most important cause of development of multidrug resistant-TB (MDR-TB).								
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	Yes	HIV is the most potent factor known to increase the risk of TB.								
Risk assessment conclusions		<p>The Directly Observed Treatment Strategy (DOTS) for TB control was introduced in Liberia in October 1999 and initially set up in seven counties. The main constraint to DOTS expansion in Liberia has been the continuing conflict, which has led directly and indirectly to destruction of health care facilities, shortage of qualified staff, low salaries – paid irregularly or not at all, lack of drugs, reagents, equipment, running water and electricity, poor means of communications, and lack of financial resources for logistics .</p> <p><u>BCG vaccination coverage</u></p> <table> <tr> <td>2002: 67%</td> <td>1998: 28%</td> </tr> <tr> <td>2001: 77%</td> <td>1997: No data</td> </tr> <tr> <td>2000: 45%</td> <td>1996: 84%</td> </tr> <tr> <td>1999: 43%</td> <td>1995: 44%</td> </tr> </table> <p>A random survey of HIV among sputum-positive TB cases (sample size 395) carried out in 1998 showed that 12% of all patients were positive for HIV, although the co-infection percentage may be much higher and poses an increased burden on any TB control programme.</p> <p>Certain considerations are essential before starting any TB programme in countries such as Liberia with complex emergencies. The WHO manual “<i>TB control in refugee situations</i>” (under revision) contains information on the minimal conditions that must be fulfilled from both sides, beneficiaries and implementing agency, when starting a DOTS programme.</p>	2002: 67%	1998: 28%	2001: 77%	1997: No data	2000: 45%	1996: 84%	1999: 43%	1995: 44%
2002: 67%	1998: 28%									
2001: 77%	1997: No data									
2000: 45%	1996: 84%									
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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 extra-pulmonary) – severity of disease – bacteriological status (assessed by sputum microscopy), and – 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>Treatment regimes</p>	<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 treatment failure 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 for which the medication is to be given in front of the letter and the number of doses per week 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 only given 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>Treatment in children</p>	<p>The drug regimens used for children are the same as for adults except that streptomycin should be avoided and ethambutol is contraindicated.</p> <p>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.</p>
<p>Treatment categories</p>	<p>Treatment categories are essential for prioritization of TB treatment according to public health risk – Category I is the highest priority, Category III the lowest.</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 who 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 is for 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 regime 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 constitutes 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"> – smear-negative pulmonary patients (with limited parenchymal involvement) – 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 of isoniazid and rifampicin every second day. 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 cannot be carried out under direct observation, all patients should be given daily ethambutol and isoniazid in the continuation phase for 6 months.</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. <u>Thiacetazone, an essential drug anti-TB drug which at times is used in combination with isoniazid when financial constraints preclude the used of ethambutol should not be given to HIV-positive TB patients</u> as there is increased risk of severe toxicity and sometimes fatal skin reactions.</p> <p>Controlled clinical trial studies have shown that isoniazid preventive treatment 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 Isoniazid preventive therapy (IPT) has shown to be more effective than other regimens 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 co-infection effectively, TB and HIV programmes should coordinate activities through a TB/HIV co-ordinating body..</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/97.22; 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>Prevention</p>	<p>Detection and treatment of smear -positive (infectious) TB cases is the most effective way 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 internationally 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>
<p>Immunization</p>	<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 – curability of 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 2 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 may spread serious disease to others, especially children.
- All patients should be treated sympathetically and with respect.
- Controlling TB is a community responsibility.

Note: Diagrams should be used as much as possible – a high literacy level should not be assumed. Cured patients are often helpful teachers and supporters of new patients.

21. TYPHOID FEVER

DESCRIPTION

Infectious agent	Bacterium: <i>Salmonella 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° for 3 or more days is considered suspect if the epidemiological context is conducive.</p> <p>Confirmed case: Isolation of <i>S. typhi</i> 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 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	No data available.
Geographical distribution	No data available
Seasonality	No data available.
Alert threshold	Two or more linked cases.
Recent epidemics	No data available.

RISK FACTORS FOR INCREASED TRANSMISSION

Population movement	Yes	Dissemination of multidrug-resistant strains of <i>S. typhi</i>
Overcrowding	Yes	Very important.
Poor access to health services	Yes	<p>Early detection and containment of cases are paramount to reducing transmission.</p> <p>Case–fatality rate is high (10–20%) in the absence of appropriate treatment.</p>
Food shortages	No	
Lack of safe water and poor sanitation	Yes	The most important risk factor.
Others	Yes	Multidrug-resistant strains of <i>S. typhi</i> , 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 and access to adequate sanitation facilities.</p> <p>There is a high risk of epidemics, particularly in complex emergency settings where the above risk factors are common.</p> <p>Rapid diagnosis with determination of antibiotic sensitivity and institution of control measures is key to containing the risk.</p>

PREVENTION AND CONTROL MEASURES

Case management	<p>Early antimicrobial treatment, selected according to the antimicrobial resistance 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.</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 Annex 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. 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, and 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. Probable: not applicable. 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 co-exist. It is not transmitted by contact or other more common means of disease transmission. Once infected, mosquitoes remain so for life.

EPIDEMIOLOGY

Burden	Liberia has been classified by WHO as one of the countries at highest risk of yellow fever. Outbreaks have happened in past years but reports are sketchy and data poor.
Geographical distribution	Endemic throughout country.
Seasonality	In forest areas, where the yellow fever virus is transmitted between mosquitoes and monkeys or chimpanzees, the disease is present throughout the year.
Alert threshold	One suspected case must lead to alert. An outbreak of yellow fever is at least one confirmed case.
Recent epidemics	<p>In previous years the Ministry of Health has reported several outbreaks.</p> <p>In 1995, four counties were affected by an important outbreak. A total of 360 cases and 9 deaths were reported from Grand-Bassa, Margibi, Montserrado (Monrovia) and Bong counties.</p> <p>In 1997, an outbreak was reported from Lofa County with one laboratory-confirmed case.</p> <p>In 1998, 25 cases and 7 deaths from “jaundice with fever” were reported from Nimba county. WHO’s investigation team confirmed yellow fever.</p> <p>In March/April 2000, 3 cases of acute jaundice with fever were admitted to Ganta hospital, Nimba county, with 1 death after admission. The other 2 patients, one 15 years old and one adult, both females, refused treatment and could not be followed up. All had negative malaria smears.</p> <p>In April 2000, a total number of 12 cases of acute jaundice were registered in Sanniquile, Nimba county, but patients refused treatment and left.</p> <p>In August/September 2000, 102 suspected cases, including 3 deaths, were detected in Grand Cape Mount county, bordering Sierra Leone; they originated from two districts.</p> <p>In August 2001, 3 suspected cases (who all died) were reported by WHO from Maryland county. The Pasteur Institute Pasteur in Abidjan confirmed only one case positive for yellow fever IgM.</p>

RISK FACTORS FOR INCREASED TRANSMISSION

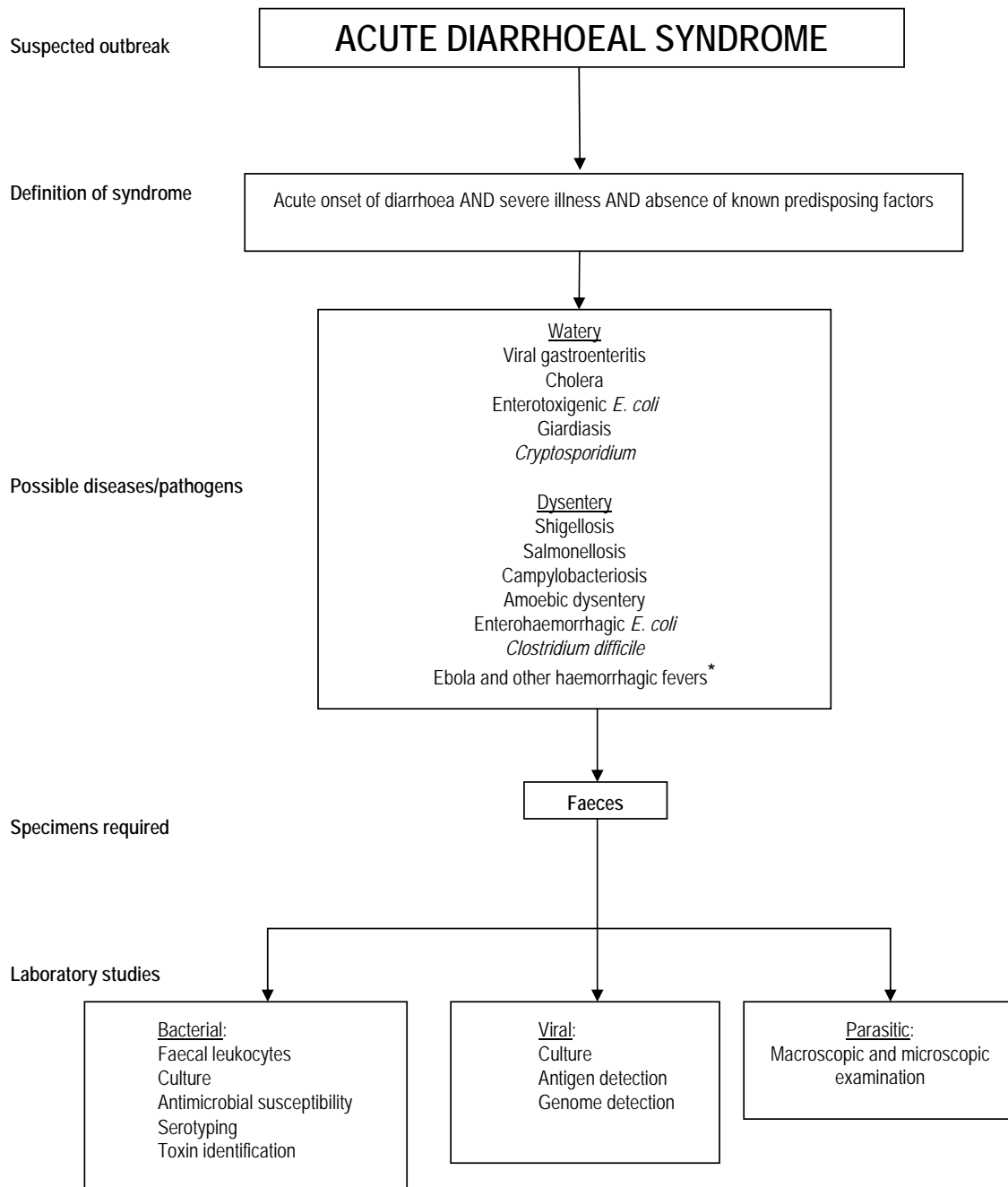
Population movement	Yes	Unvaccinated people moving to areas of endemicity are at risk. Changes in land use increase the risk of breeding of the mosquito vector.
Overcrowding	Yes	Increased population density and increased exposure to mosquito bites in temporary shelters.
Poor access to health services	Yes	Due to collapse of vaccination programmes, surveillance and general health infrastructure to allow rapid detection and response. Increased fatality without proper case management.
Food shortages	No	
Lack of safe water and poor sanitation	No	

<p>Others</p>	<p>Yes</p>	<p>Open water storage provides a favourable habitat for <i>Ae. aegypti</i>. Old tyres, water containers, etc. increase vector breeding. Temporary surface-water bodies (poor drainage leading to pools and open channels of water) may increase vector-breeding opportunities.</p>
<p>Risk assessment conclusions</p>		<p>Yellow fever cases were confirmed in Bong and Nimba counties in February 2004. Inadequate vaccination coverage rates for yellow fever and collapse of mosquito control programmes are the most likely causes.</p> <p>Moreover, population movements as a consequence of war have resulted in large numbers of people living in conditions of poverty and overcrowding, amplifying the risk of transmission. The massive movement of people between Liberia and neighbouring countries that suffer regular outbreaks (such as Côte d'Ivoire and Sierra Leone) is an additional risk factor.</p> <p>Disease surveillance and health infrastructure are poor in Liberia. An outbreak of yellow fever can go undetected because the signs and symptoms overlap with those of many other diseases, making it difficult for health workers to make a definitive diagnosis on this basis alone. Mild cases can go undetected because patients are likely to be treated at home and may not seek care in a health facility.</p> <p><u>Yellow fever virus vaccine (YFV) coverage</u></p> <p>To prevent an epidemic in a country, at least 80% of the population must have immunity to yellow fever. The last yellow fever vaccination campaign in Liberia was carried out in 1995 but coverage was reported to be low, leaving much of the population susceptible.</p> <p>Between 1995 and 1999, mass campaigns were conducted in epidemic-affected counties. Over a period of 6 months, 1 555 888 doses were administered. In Onlu (Margibi county) and in Grand Bassa county, an average of more than 80% of the target population were covered. In the remaining counties, coverage ranged from 75% in Montserrado (Monrovia) to 0% in Grand Ku and Grand Gedeh counties.</p> <p>(Details of Yellow fever coverage report from September 1998, based on population figures from 1984, per county: Grand Bassa: 50.6%; Montserrado 77.8%; Bong 53%; Margibi 97.5%; Upper Lofa 13.2%; Rivercess 4%; Grand Gedeh 0; Sonoe 6%; Maryland 1.5%; Grand Kru 0%; Bomi 0%; Cape Mount 0; Nimba 5%. Catch-up campaign in October 1998: Nimba 89.5%)</p> <p>Following the outbreak in August/September 2000, a mass vaccination campaign was conducted, targeting 150 000 people in Grand Cape Mount county – the coverage achieved is unknown.</p> <p>A further mass campaign in 2002 resulted in YFV coverage of 85% in Montserrado, 48% in Bong, 43% in Nimba and 85% in Rivercess.</p>

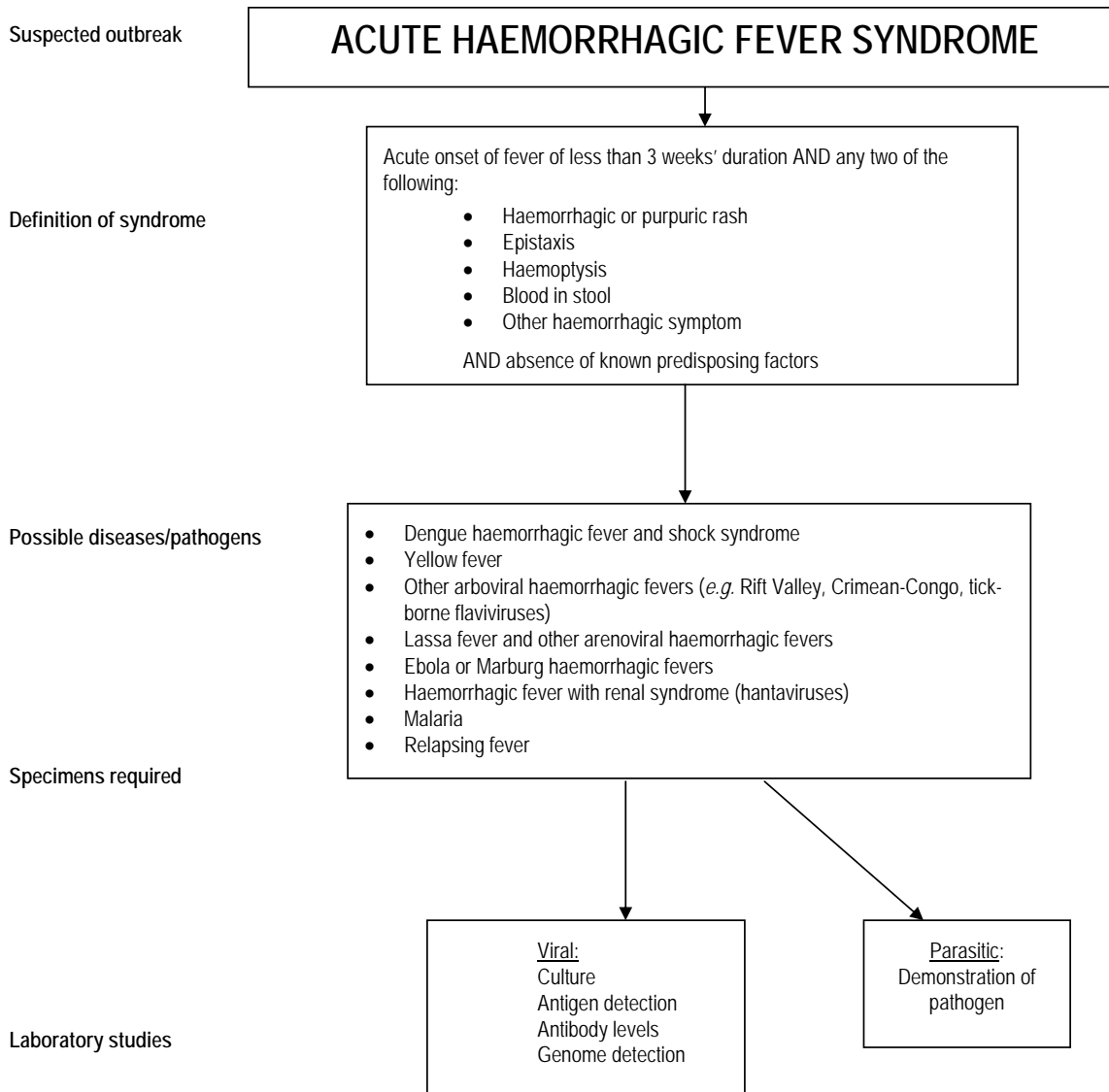
PREVENTION AND CONTROL MEASURES

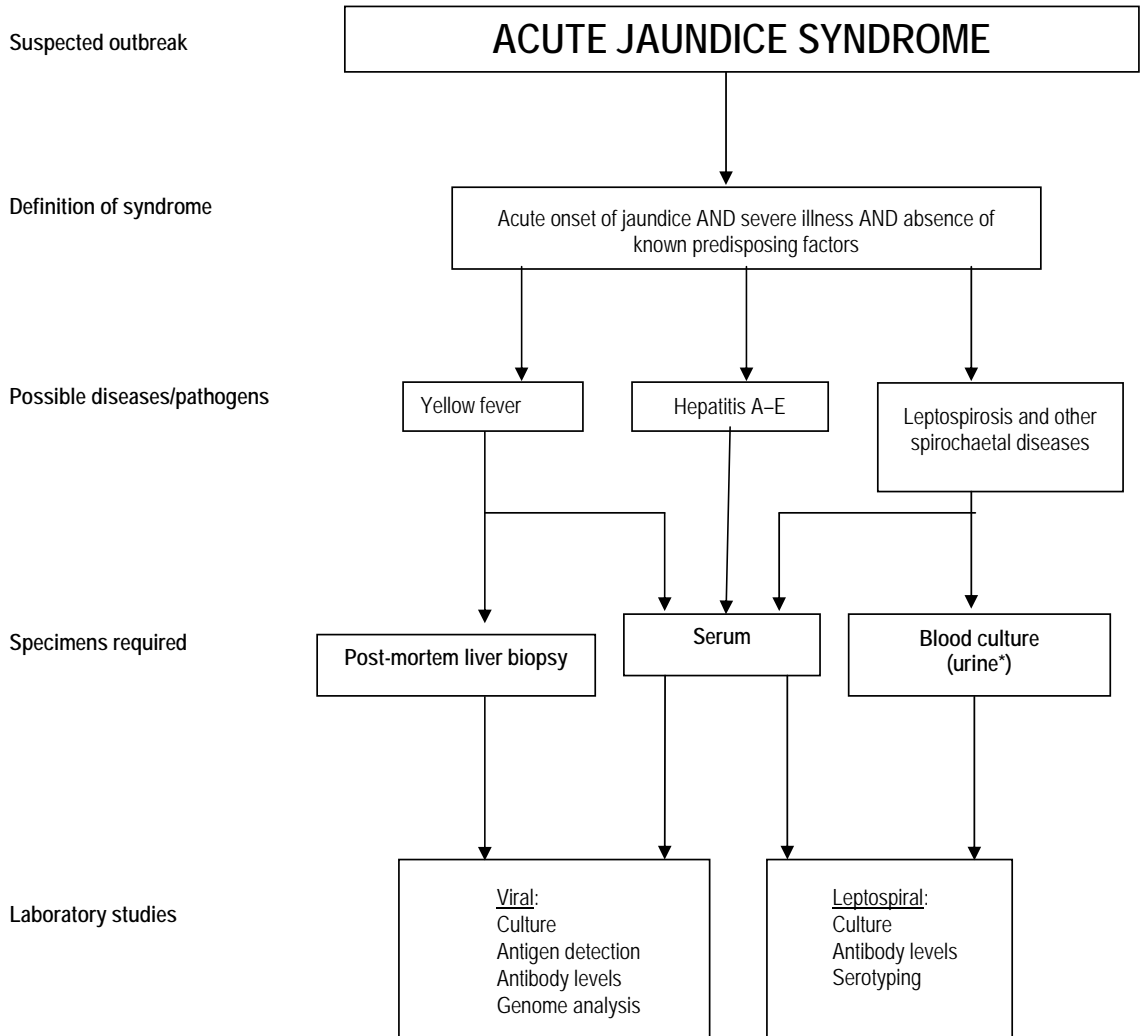
<p>Case management</p>	<p>No specific treatment for yellow fever is available.</p> <p>Dehydration and fever can be corrected with oral rehydration salts.</p> <p>Intensive supportive care may improve the outcome but is rarely available.</p> <p>See: <i>Case Management of Epidemic-Prone Diseases</i> in this Toolkit for Liberia.</p>
<p>Epidemic control</p>	<p>An infected mosquito spreads yellow fever when it bites non-infected humans. When human-to-human transmission is established, the conditions for an epidemic are in place. Depending on the travel patterns of infected humans or infected mosquitoes, the epidemic spreads from village to village and into cities.</p> <p>Under epidemic conditions, the following must be implemented:</p> <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 yellow fever vaccine in routine child immunization programmes and mass preventive campaigns. Yellow fever vaccine 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 for Liberia:</p> <ul style="list-style-type: none"> – vaccinating the population over 9 months in counties where coverage in recent campaigns achieved less than 80%; – if funds are limited, a cheaper 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>Routine mosquito control measures</p> <p>Potential mosquito breeding sites must be eliminated.</p>

ANNEX 1: FLOWCHARTS FOR THE DIAGNOSIS OF COMMUNICABLE DISEASES

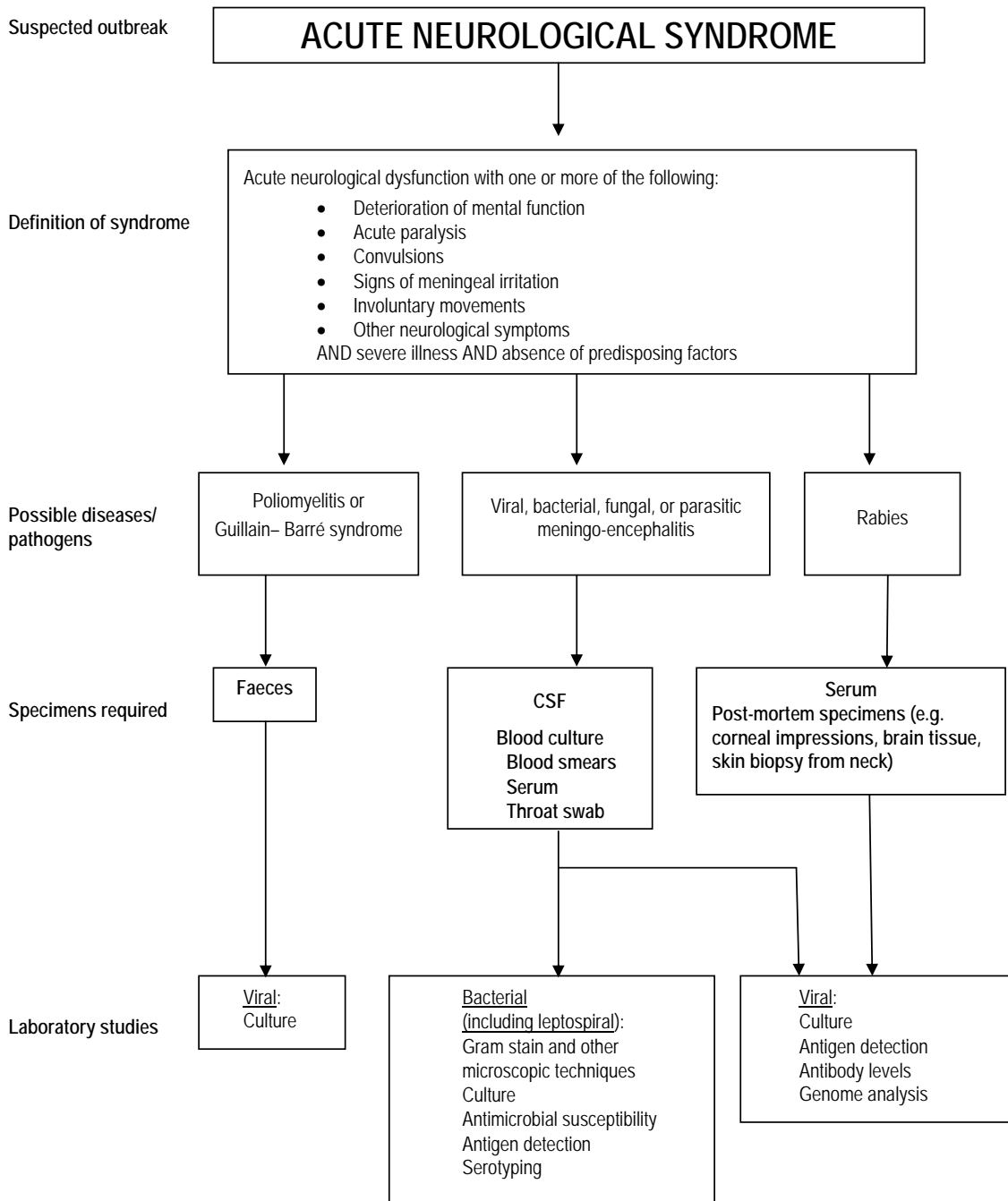


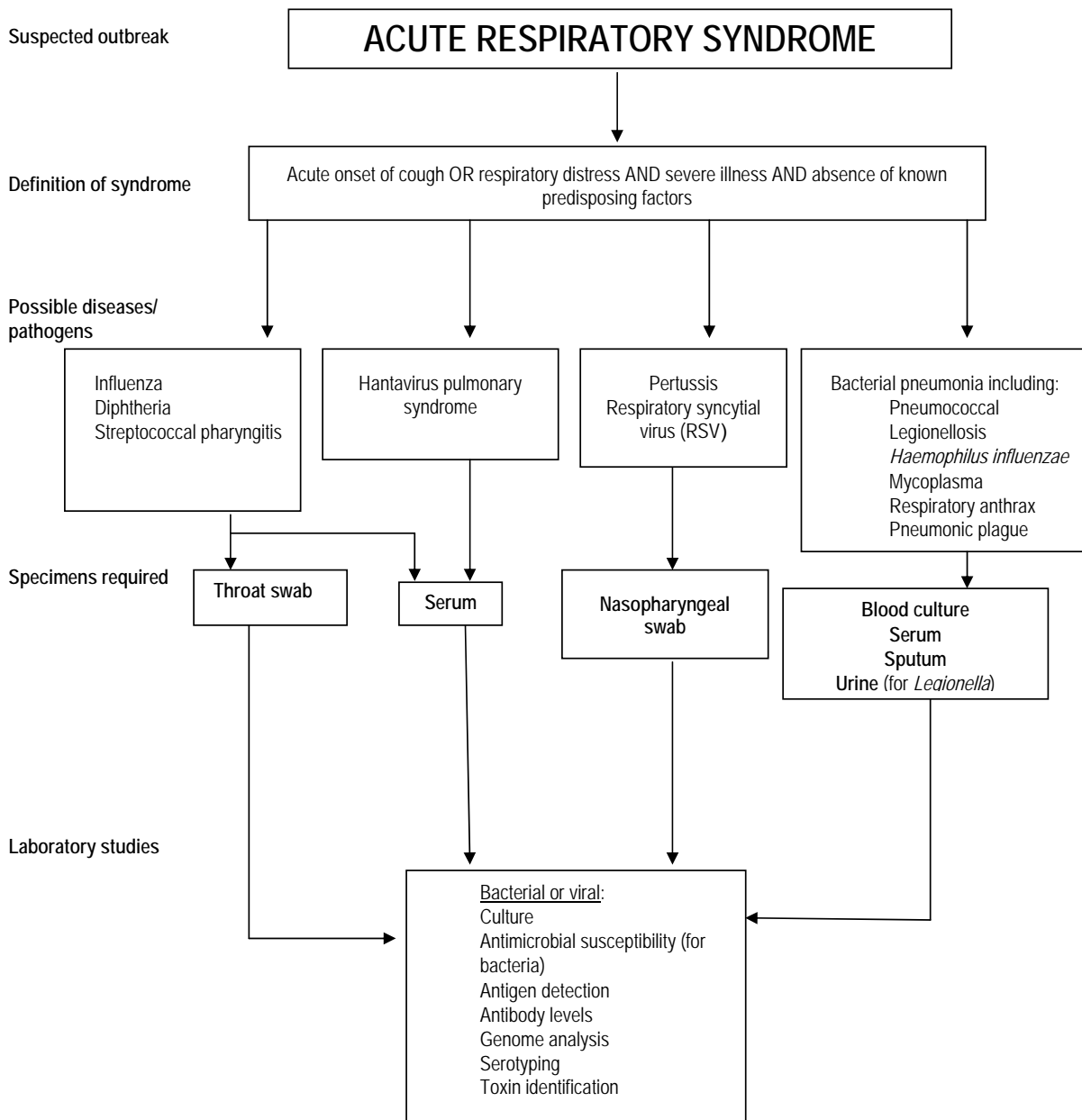
Ebola and other haemorrhagic fevers may initially present as bloody diarrhoea. If such an etiology is suspected, refer to "Acute Haemorrhagic Fever Syndrome" for appropriate specimen collection guidelines (See: Infection control for viral hemorrhagic fevers in the African health care setting, WHO/EMC/ESR/98.2)





* Requires specialized media and handling procedures.





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

ANNEX 2: STEPS IN OUTBREAK MANAGEMENT

<p>PREPARATION</p> <ul style="list-style-type: none"> • 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
<p>DETECTION</p> <p>If a certain number of cases of any of the following diseases/syndromes is diagnosed (i.e. alert threshold is passed):</p> <ul style="list-style-type: none"> • 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 should be modified according to the country's most update epidemiological profile). <p>Inform your health coordinator as soon as possible. The health coordinator should inform the Ministry of Health and WHO.</p>
<p>RESPONSE</p> <p>Confirmation</p> <ul style="list-style-type: none"> • 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 members drawn 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. <p>Investigation</p> <ul style="list-style-type: none"> • 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) <p>Control</p> <ul style="list-style-type: none"> • 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.
<p>EVALUATION</p> <ul style="list-style-type: none"> • Assess timeliness of outbreak detection and response, cost. • Change public health policy if indicated (e.g. preparedness). • Write outbreak report and disseminate.

ANNEX 3: SAFE WATER AND SANITATION

The following are effective methods of obtaining 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 be filtered before chlorination or boiled vigorously rather than chlorinated. Chlorination of turbid water might not make it safe.

See:

- *Cholera fact sheets on Environmental sanitation. Geneva, WHO/EOS/96.4*
- *Cholera and other epidemic diarrhoeal disease control: Technical cards on environmental sanitation. Geneva, WHO 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:

- *Cholera and other epidemic diarrhoeal disease control: fact sheets on environmental sanitation. Geneva, WHO 1996 (WHO/EOS/96.4).*
- Franceys R, Pickford J, Reed R. *A guide to the development of on-site sanitation. Geneva, WHO, 1992.*

ANNEX 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 region that includes Sierra Leone faces substantial challenges in terms of unsafe injection practices and transmission of bloodborne pathogens through injections. In this region, the proportion 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 and ensure 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 needlestick injury.
- Dispose of by open-air incineration and burial of full sharps 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 that are not disposed of properly.

ANNEX 5: KEY CONTACT FOR LIBERIA

Table 1: World Health Organization – Liberia

Office of the WHO Representative	<p>Dr Omar Juma Khatib <i>WHO Representative</i> Sekou Toure Ave, Mamba Point P.O. Box 316 1000 Monrovia 10 Tel: +231 227 378 / +231 226 208 Sat. tel: +871 762 545 625 Mobile: + 377 47 516 801 Fax: +231 226 747 E-mail: omarjk@hotmail.com khatibo.who@undp.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
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 qazis@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 Mark Karam karamm@who.int Dr Ahmed Tayeh tayeha@who.int
HIV/AIDS		Dr Andrew Ball balla@who.int Dr Brian Pazvakavambwa pazvakavambwa@who.int Dr Michel Tailhardes tailhardesm@who.int
Leishmaniasis		Dr Philippe Desjeux desjeuxp@who.int
Leprosy		Dr Denis Daumerie daumeried@who.int Dr Myo Thet Htoon htoonm@who.int

Lymphatic filariasis		Dr Francesco Rio riofo@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 hershb@who.int
Meningococcal disease		Dr William Perea pereaw@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 Mr Chris Everts everts@who.int
Rabies		Dr François-Xavier Meslin meslinf@who.int
Schistosomiasis		Dr Lorenzo Savioli savioli@who.int Dr Dirk Engels engelsd@who.int
Soil-transmitted helminths		Dr Lorenzo Savioli savioli@who.int Dr Dirk Engels engelsd@who.int
Tuberculosis		Dr Salah-Eddine Ottmani ottmanis@who.int Dr Giuliani Gargione gargionig@who.int
Viral haemorrhagic fevers		Dr Cathy Roth rothc@who.int Mr Pierre Formenty formentyp@who.int
Health aspects of biological agents		Dr Ottorino Cosivi cosivio@who.int
Injection safety		Dr Yvan Hutin hutin@who.int
Safe water		Mr Jose Hueb huebj@who.int

ANNEX 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/inf-fs/en/fact264.html
Cholera	Fact Sheet No. 107 Revised March 2000 http://www.who.int/inf-fs/en/fact107.html
Crimean-Congo haemorrhagic fever	Fact Sheet No. 208 December 1998 http://www.who.int/inf-fs/en/fact208.html
Dengue and dengue haemorrhagic fever	Fact Sheet No. 117 Revised November 1998 http://www.who.int/inf-fs/en/fact117.html
Diphtheria	Fact Sheet No. 89 Revised September 2000 http://www.who.int/inf-fs/en/fact089.html
Epidemic dysentery	Fact Sheet No. 108 Revised October 1996 http://www.who.int/inf-fs/en/fact108.html
<i>Escherichia coli</i> O157:H7	Fact sheet No. 103 Revised December 2000 http://www.who.int/inf-fs/en/fact103.html
Food safety and foodborne illness	Fact Sheet No. 237 revised January 2002 http://www.who.int/inf-fs/en/fact237.html Fact Sheet No. 124 January 2002 http://www.who.int/inf-fs/en/fact124.html
Hepatitis B	Fact Sheet WHO/204 Revised October 2000 http://www.who.int/inf-fs/en/fact204.html
Hepatitis C	Fact Sheet No. 164 Revised October 2000 http://www.who.int/inf-fs/en/fact164.html
Influenza	Fact Sheet No. 211 February 1999 http://www.who.int/inf-fs/en/fact211.html
Influenza A(H5N1)	Fact Sheet No. 188 January 1998 http://www.who.int/inf-fs/en/fact188.html
Injection safety: background	Fact Sheet No. 231 Revised April 2002 http://www.who.int/inf-fs/en/fact231.html
Injection safety: facts & figures	Fact Sheet No. 232 October 1999 http://www.who.int/inf-fs/en/fact232.html
Injection Safety: a Glossary	Fact Sheet No. 233 October 1999 http://www.who.int/inf-fs/en/fact233.html
Injection safety: questions & Safety	http://www.who.int/inf-fs/en/fact234.html
Leishmaniasis	Fact Sheet No. 234 May 2000 http://www.who.int/inf-fs/en/fact116.html
Malaria	Fact Sheet No. 94 http://www.who.int/inf-fs/en/fact094.html
Plague	Fact Sheet No. 267 January 2002 http://www.who.int/inf-fs/en/fact267.html

Poliomyelitis	Fact Sheet No. 114 Revised August 2002 http://www.who.int/mediacentre/factsheets/fs114/en/
Rabies	Fact Sheet No. 99 Revised June 2000 http://www.who.int/inf-fs/en/fact099.html
Salmonella	Fact Sheet No. 139 January 1997 http://www.who.int/inf-fs/en/fact139.html
Smallpox	Fact sheet October 2001 http://www.who.int/emc/diseases/smallpox/factsheet.html
Tuberculosis	Fact Sheet no 104 Revised August 2002 http://www.who.int/mediacentre/factsheets/who104/en/
Typhoid fever	Fact sheet No. 149 March 1997 http://www.who.int/inf-fs/en/fact149.html
GUIDELINES/PUBLICATIONS/REPORTS	
First steps for managing an outbreak of acute diarrhoea http://www.who.int/csr/resources/publications/cholera/WHOCDSCSRNC20037.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/denque/whocdsdenic20001c.html	WHO/CDS/(DEN)/IC/2000.1 English only
WHO report on global surveillance of epidemic-prone infectious diseases http://www.who.int/csr/resources/publications/surveillance/WHO_CDS_CSR_ISR_2000_1/en/	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/csr/resources/publications/influenza/WHO_CDS_CSR_EDC_99_1/en/	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 human and animals, 3rd ed.	WHO/EMC/ZDI/98.6
Cholera and other epidemic diarrhoeal diseases control. Technical cards on environmental sanitation, 1997 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	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	English and French
VIDEOS	
Protecting ourselves and our communities from cholera (41 min) http://www.who.int/emc/diseases/cholera/videos.html	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
Roll Back Malaria	http://www.rbm.who.int
Stop TB	http://www.stoptb.org/

ANNEX 7: IMMUNIZATION SCHEDULE FOR LIBERIA

VACCINE	SCHEDULE
BCG	Birth
DTP	6, 10, 14 weeks
OPV	Birth, 6, 10, 14 weeks
Measles	9 months
Vitamin A	9 months
Tetanus Toxoid (TT)	First contact; + 4 weeks; +6 months; +1 year; +1 year (WCBA/pregnant women)
Yellow Fever F (YF)	9 months

WCBA = woman of childbearing age

ANNEX 8: MAP OF LIBERIA



ANNEX 9: BASIC HEALTH INDICATORS FOR LIBERIA

Life expectancy at birth (LE)	41.4 years (both sexes combined, 2000–2005) (Source: UNDP)
Infant mortality rate (per 1000 live births) (IMR)	168 (1990); 111 (2000); 157 (2001) (Source: UNICEF)
Under-5 mortality rate (per 1000 live births) (U5MR)	185 (2000); 235 (2001) (Source: UNICEF)
Maternal mortality ratio (per 100 000 live births) (MMR)	580 (1985–2001) ^a (Source: WHO/UNICEF/Government of Liberia)
Total fertility rate (per woman) (TFR)	6.8 (1990); 6.0 (2000); 6.8 (2000–2005) (Source: UNDP)
Annual population growth rate (%)	3.4 (1990–2000) UNICEF; 4.05 (2000–2005) (Source: UNDP)
Access to safe water source	Overall 32% of the population. Urban: 55%; Rural: 10% (Source: Liberia Demographic Health Survey, 2000)
Access to sanitary excreta disposal	30% (Source: Health Situation Analysis Report 2000, WHO/Ministry of Health Liberia).

^a Most recent year available during the specified period (1995).

ANNEX 10: POPULATION DATA, 2002

Live births	161 000
Female 15–49 years	730 000
Population under 15 years	1 510 (46% of total population, 2001)
Population under 5 years	617 000
Surviving infants	131 000
Total population	3 239 000

IDP/REFUGEE POPULATION		SOURCE
Internally displaced	Total : 500 000 in Liberia 300 000 – in IDP camps and irregular settlements in Monrovia 200 000 – outside Monrovia	UNHCR, September 2003
Refugees	73 000 Liberians in Guinea 53 000 Liberians in Sierra Leone 50 000 Liberians in Côte d'Ivoire 40 000 Liberians in Ghana 38 000 Ivorians in Liberia 13 500 Sierra Leoneans in Liberia	UN OCHA, September 2003 UNHCR, June 2003 U.S. Committee for Refugees, August 2003 UNHCR, September 2003 UNHCR, September 2003 UNHCR, August 2003