World Health Organization

COMMUNICABLE DISEASE EPIDEMIOLOGICAL PROFILE

Côte d'Ivoire

Disease Control in Humanitarian Emergencies (DCE)
Global Alert and Response Department (GAR)

The Communicable Disease Epidemiological Country Profile series was conceived and developed by the WHO team for Disease Control in Humanitarian Emergencies (DCE), to provide up-to-date information on the major communicable disease threats faced by the resident and displaced populations in emergency affected countries.

The information provided aims to assist with the public health strategy, prioritization and coordination of communicable disease control activities between all agencies working in such countries.

Diseases have been included if they fulfil one or more of the following criteria: have a high burden or epidemic potential, are (re) emerging diseases, important but neglected tropical diseases, or diseases subject to global elimination or eradication programmes.

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WHO/HSE/GAR/DCE/2010.3

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Côte d'Ivoire

Design and layout: Rick Jones, Exile: Design & Editorial Services, London (United Kingdom)

Printed by the WHO Document Production Services, Geneva, Switzerland

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Acknowledgements

The production of this communicable disease epidemiological profile was coordinated by the Disease Control Unit in Humanitarian Emergencies (DCE), part of Global Alert and Response (GAR) in the Health Security and Environment cluster (HSE) of the World Health Organization (WHO).

Technical input was received from the Communicable Diseases Working Group on Emergencies (CD-WGE) at WHO headquarters, the Division of Communicable Disease Prevention and Control (DDC) at the WHO Regional Office for Africa, and the Country Office of the WHO Representative for Côte d'Ivoire.

The CD-WGE provides technical and operational support on issues relating to communicable diseases to WHO regional and country offices, ministries of health, other United Nations agencies and nongovernmental and international organizations. The CD-WGE includes the departments of Global Alert and Response (GAR), Food Safety, Zoonoses and Foodborne Diseases (FOS), Public Health and Environment (PHE) in the Health Security and Environment (HSE) cluster; the Special Programme for Research and Training in Tropical Diseases (TDR) in the Information, Evidence and Research (IER) cluster; the Global Malaria Programme (GMP), Stop TB (STB), HIV/AIDS , Control of Neglected Tropical Diseases in the HIV/AIDS,TB, Malaria and Neglected Diseases (HTM) cluster; the departments of Child and Adolescent Health and Development (CAH), Immunization, Vaccines and Biologicals (IVB) in the Family and Community Health (FCH) cluster; Injuries and Violence Prevention (VIP) and Nutrition for Health and Development (NHD) in the Non-communicable Diseases and Mental Health (NMH) cluster; the department of Essential Health Technologies (EHT) in the Health Systems & Services (HSS) cluster; Security and Staff Services (SES) in the General Management (GMG) cluster; the cluster of Health Action in Crises (HAC) and the Polio Eradication Initiative (POL).

DCE gratefully acknowledges the current and previous collaboration and input of the disease-specific focal points of the CD-WGE, the WHO Regional Office for Africa and the Country Office of the WHO Representative for Côte d'Ivoire, which have made the production of this profile possible.

We would also like to thank the Office of Foreign Disaster Assistance (OFDA) of the United States Agency for International Development (USAID) for their support in the development of this document.

Editorial work by Heidi Mattock.

Editorial and production support was provided by the Information Management & Communication team in Global Alert and Response (GAR).

Acronyms and abbreviations

ACT	artemesinin-based combination therapy
ADL	acute adenolymphangitis
AFB	acid-fast bacilli
AFP	acute flaccid paralysis
ALRI	acute lower respiratory infection
ART	antiretroviral treatment
ARV	antiretroviral
BCG	Bacille Calmette–Guérin
BU	Buruli ulcer
CATT	card agglutination trypanosomiasis test
CFR	case-fatality ratio
CSF	cerebrospinal fluid
DALY	disability-adjusted life year
DEC	diethylcarbamazine citrate
DHF	dengue haemorrhagic fever
DOTS	directly observed treatment, short-course
DRC	The Democratic Republic of the Congo
DSS	dengue shock syndrome
DTP3	third dose of diphtheria-tetanus-pertussis vaccine
EIA	enzyme immunoassay
ELISA	enzyme-linked immunosorbent assay
EPI	Expanded Programme on Immunization
FA	fluorescent antibody
GAVI	Global Alliance for Vaccines and Immunization
HAT	human African trypanosomiasis
HEV	hepatitis E virus
Hib	*Haemophilus influenzae* B
HIV/AIDS	human immunodeficiency virus/acquired immunodeficiency syndrome
HPAI	highly pathogenic avian influenza
IASC	Inter-agency Standing Committee
IFA	indirect fluorescent antibody

IDU	injecting dug user
IM	intramuscular
IMCI	Integrated Management of Childhood Illness
IPT	isoniazid preventive therapy
ITN	insecticide treated net
IV	intravenous
LF	lymphatic filariasis
LLIN	long-lasting insecticidal net
MDA	mass drug administration
MDR	multidrug resistance
MDT	multidrug therapy
NIDs	national immunization days
NMCP	national malaria control programme
NSAIDs	non-steroidal anti-inflammatory drugs
NTCP	national tuberculosis control programme
ORS	oral rehydration salts
PCP	*Pneumocystic carinii (jeroveci)* pneumonia
PCR	polymerase chain reaction
PEP	post-exposure prophylaxis
PPE	personal protective equipment
RS	rifampicin and streptomycin
RSV	respiratory syncytial virus
SD	standard deviation
SIA	supplementary immunization activities
SNIDs	subnational immunization days
SSNCP	sleeping sickness national control programme
STH	soil-transmitted helminth
STI	sexually transmitted infection
TB	tuberculosis
TMP–SMX	trimethoprim–sulfamethoxazole
UNAIDS	The Joint United Nations Programme on HIV/AIDS
UNHCR	United Nations High Commission for Refugees
UNICEF	United Nations Children's Fund
VCT	voluntary counselling and testing
WHO	World Health Organization

PART I

Introduction

Purpose

The series *Communicable disease epidemiological profiles* for emergency affected countries were conceived and developed by the World Health Organization (WHO) team for Disease Control in Humanitarian Emergencies (DCE). The aim of these profiles is to provide up-to-date information on the major threats posed by communicable diseases among resident and displaced populations in countries affected by emergencies. Such information is designed to assist with the public health strategy, prioritization and coordination of communicable-disease control activities among all agencies working in such countries.

The purpose of publications in this series is primarily to guide public health actions; although the profile contains clinical information, it is not designed primarily for clinical practice. Clinical decisions should not be based solely on the information contained within this document.

Target audience

Public health managers and professionals working for populations living in Côte d'Ivoire

Document rationale

The diseases presented in this profile have been included on the basis of their high burden or epidemic potential in Côte d'Ivoire, or because they are (re)emerging diseases, important but neglected tropical diseases, or a target for global elimination or eradication programmes. Each chapter describes a specific disease.

The Annexes provide sources of information to guide the formulation of public health actions and include: background information on Côte d'Ivoire (including key national indicators); steps in the management of an outbreak of a communicable disease; flow charts for the diagnosis of communicable diseases; notes on safe water and sanitation; injection safety; disease-specific WHO reference materials; and a WHO contact list.

Document production process

This *Communicable disease epidemiological profile* is the product of collaboration between the Communicable Disease Working Group in Emergencies (CD-WGE), the appropriate WHO region and country offices and DCE. Overall technical coordination is provided by DCE.

Each chapter describing the situation for a specific disease was drafted by DCE then submitted to the appropriate disease WHO Focal Point of the CD-WGE, WHO regional and country offices to ensure technical accuracy and the inclusion of country-specific information. The disease-specific chapters are presented in alphabetical order.

Fig .1 **Map of Côte d'Ivoire showing administrative boundaries**

The quantity and quality of epidemiological data in this particular profile is compromised by the humanitarian crisis in Côte d'Ivoire, which has disrupted health and surveillance systems for many years.

Background to the humanitarian crisis and its impact on health in Côte d'Ivoire

Côte d'Ivoire gained independence from France in August 1960, after 67 years of colonization. Increasing tensions culminated in rebellion during September 2002, dividing the country into the occupied north under the control of the New Forces (*Forces Nouvelles*) and the government-controlled south. French and United Nations (UN) peacekeepers patrol the buffer zone. After a number of failed attempts at peace-brokering, the signing of the Ouagadougou Peace Agreement (OPA) between the Ivorian government and the *Forces Nouvelles* on 4 March 2007 has improved the security environment and the relationship of Côte d'Ivoire with the international community in general.

As of late 2008, an estimated 620 000 people remain internally displaced, mainly to Abidjan (Fig. 1). About 1000 people remain in a camp in the west. In addition, the country hosts some 30 000 Liberian refugees, mainly in the west.

Health-delivery systems have been severely disrupted, particularly in the north and west of the country: 80% of health units in these areas are closed, 85% of the health workers have left. Disease monitoring systems and immunization programmes have been severely interrupted with important consequences, as exemplified by the notification of 17 polio cases in 2004 (see *Poliomyelitis* chapter), outbreaks of yellow fever (13 confirmed cases in May–July 2008; see *Yellow fever* chapter) and meningitis (1020 cases as of 3 August 2008; see *Meningococcal disease*), and re-emergence of diseases such as onchocerciasis (see *Onchoceriasis [river blindness]*).

The consequences of decades of political instability and armed conflict are reflected in the poor rankings of Côte d'Ivoire on the United Nations Develoment Programme (UNDP) Human Development Index (166 out of 177 countries) and the Human Poverty Index (92 out of 108 countries). The latter index in particular reflects severe deprivation in health by measuring the proportion of the population not expected to survive to age 40 years.

Côte d'Ivoire is a considerable distance from achieving the Millennium Development Goals, particularly those concerning infant and maternal mortality, education, provision of water and sanitation and gender equality (on the Gender-related Development Index, the country ranks 151 out of 156 countries). Recent improve-

ments in water supply in urban centres have not been matched in rural areas. Sanitation remains poor: in urban areas and in rural areas open defecation – the riskiest sanitation practice – currently stands at 51%. Communicable diseases account for more than 50% of adult deaths and about 80% of deaths among children of under the age of 5 years.

Diseases for which the prevention and control is of high priority include:

- Diseases with a high burden and with potential for amplification
  - Acute respiratory illness in children
  - Diarrhoeal illnesses
  - Malaria
  - Tuberculosis
  - HIV/AIDS
- Diseases with potential for outbreaks
  - Measles
  - Meningitis
  - Yellow fever
  - Cholera, dysentery
- Diseases subject to global control with potential for programme disruption
  - Polio

Owing to the disruption of immunization programmes and vaccine supply, routine immunization is insufficient to prevent outbreaks of common infectious diseases. Vaccination campaigns should be planned and implemented in accordance with WHO recommendations.

A coordinated approach comprising public health measures and disease prevention, detection, response and control is required for both the priority communicable diseases with outbreak potential and the endemic communicable diseases with potential for amplification.

Further reading

Annex 1: Key national indicators and general information for Côte d'Ivoire; History of the humanitarian crisis

PART II

Country-specific disease information

ACUTE LOWER RESPIRATORY-TRACT INFECTIONS (ALRI)

Description

Clinical description

Acute lower respiratory tract infections (ALRI) include bronchitis, bronchiolitis and pneumonia (broncho- and lobar). Severe pneumonia is fatal in 10–20% of cases if inappropriately treated. Early recognition and prompt treatment are life-saving.

Infectious agent

Bacterial agents account for approximately 30% of ALRI, the most common being *Streptococcus pneumoniae* and *Haemophilus influenzae*. Common *v*iral agents include respiratory syncytial viruses (RSV), adenovirus, parainfluenza virus, rhinovirus, influenza virus and metapneumovirus.

Under-recognized and poorly diagnosed causes of pneumonia include *Staphylococcus aureus, Mycoplasma pneumoniae,* and Gram-negative organisms; the latter occurring particularly in cases of hospital-acquired pneumonia and in immunosuppressed individuals. *Mycobacterium tuberculosis* is an often neglected cause of acute respiratory infections. In addition, HIV-infected patients are also particularly susceptible to *Pneumocystic carinii (jeroveci)* pneumonia (PCP). In Côte d'Ivoire, paragonomiasis (lung fluke) may also cause an acute respiratory illness.

Case classification

Suspected/confirmed

The Integrated Management of Childhood Illness (IMCI) case classification for the purposes of initiating empirical treatment is given below. If wheezing is present, give a trial of rapid-acting inhaled bronchodilator for up to three cycles before classifying as pneumonia.

PNEUMONIA (age 2–59 months)

Cough or difficult breathing,

and fast breathing (50 breaths/minute or more for infants aged 2–11 months, 40 breaths per minute or more for child aged 12–59 months, after trial of bronchodilators if wheeze present),

and no general danger signs (unable to drink or breast feed, vomits everything, convulsions, lethargic or unconscious) **or** chest indrawing or stridor

SEVERE PNEUMONIA/VERY SEVERE DISEASE (age 2–59 months)

Cough or difficult breathing

and any general danger sign (unable to drink or breast feed, vomits everything, convulsions, lethargic or unconscious) **or** chest indrawing (severe pneumonia) or stridor in a calm child.

VERY SEVERE DISEASE (age < 2 months)

Clinically, it is difficult to distinguish between pneumonia and other causes of very severe disease in a young infant. Classify the infant as having very severe disease if any one of the following signs is present: not feeding well **or** convulsions **or** fast breathing (60 breaths per minute or more) **or** severe chest indrawing **or** fever (axillary temperature, 37.5 °C or above) **or** low body temperature (less than 35.5 °C axillary) **or** movement only when stimulated **or** no movement at all

Mode of transmission

Airborne through droplets, additionally through oral contact or contact with hands or items freshly soiled with respiratory secretions.

Incubation period

Incubation varies depending on the infective agent (usually 2–5 days).

Period of communicability

Depends on the infective agent. Usually during the symptomatic phase.

Epidemiology

Disease burden

Worldwide, ALRI are the leading cause of death among children of under the age of 5 years and are estimated to be responsible for more than 2 million deaths in childhood. In developing countries, an estimated 151 million new episodes of pneumonia per year occur in children under the age of 5 years, of which 11–20 million episodes require hospital admission. Furthermore, the human immunodeficiency virus (HIV) epidemic has significantly increased the incidence and geographical distribution of causative agents of ALRI. Despite the high global burden of disease associated with ALRI, there is a lack of clear epidemiological

and clinicopathological data for acute lower respiratory tract infections worldwide. Studies are needed to delineate the causes, incidence rates, patterns of resistance to treatment and effectiveness of management protocols.

An estimated 20% of deaths in children under the age of 5 years are due to pneumonia in Côte d'Ivoire. Only 38% of children under age 5 years with pneumonia are taken to an appropriate health-care provider.

Geographical distribution

ALRI occurs throughout rural and urban areas of Côte d'Ivoire.

Seasonality

In tropical settings, incidence is highest in the rainy season and among children under the age of 5 years.

Alert threshold

An increase in the number of cases above the expected number for that time of the year in a defined area.

Risk factors for increased burden

Population movement

Contact between infected and susceptible individuals can increase transmission of the pathogen. Antibiotic resistant strains can spread to different geographical regions thereby increasing the burden of disease.

Overcrowding

Overcrowding and poor ventilation increases risk of infection.

Poor access to health services

Prompt identification and treatment of cases are the most important control measures. Poor access to effective health services can delay or prevent adequate treatment, without which case-fatality ratios (CFR) can be very high (≥ 20% in emergency situations).

Food shortages

Malnutrition, low birth weight and poor breastfeeding practices are important risk factors for development of the disease and increased severity of illness.

Lack of safe water, poor hygienic practices and poor sanitation

Inadequate safe water, poor personal hygiene, hand washing and ventilation increase the risk of spread of respiratory infection.

Others

Low birth weight and lack of exclusive breastfeeding for the first 4 months of life are additional risk factors.

Prevention and control measures

Case management

The priority is the early recognition and appropriate treatment of cases. Health-care providers trained in IMCI should use the guidelines provided by IMIC (*1,2*).

For the management of HIV-infected children, *Integrated Management of Childhood Illness for high HIV settings* should be used (*3*).

Treatment guidelines are given below for the most common bacterial causes of ALRI. Patients who are slow to respond to treatment should be investigated for other infectious agents. Respiratory illness in HIV-infected patients may be due to a broad range of agents, including common bacterial causes, tuberculosis and PCP.

Supportive measures, such as continued feeding to avoid malnutrition, vitamin A if indicated, antipyretics to reduce high fever, and protection from the cold (especially keeping young infants warm) are part of integrated case management. Prevention of low blood glucose is necessary for severe cases (breastfeeding or sugar-water). For non-severe cases, caregivers should be given careful counselling on home-based care, including adherence to antibiotic treatment.

Signs of malnutrition should be assessed as this increases the risk of death due to pneumonia. Severely malnourished children (bilateral pitting oedema or visible severe wasting or < 70% weight-for-height or < three standard deviations Z score) should be referred to hospital.

Non-severe pneumonia (age 2–59 months)

Five days of antibiotic therapy (oral amoxicillin or co-trimoxazole) should be used, with follow-up in 2 days. Oral amoxicillin should be used twice daily at a dose of 25 mg/kg per dose. co-trimoxazole is recommended as prophylaxis for PCP in all HIV-positive children and in infants born to HIV-infected mothers. Children with wheeze should be given inhaled or oral bronchodilator for 5 days.

Persistent cough for more than 3 weeks should be referred for assessment for tuberculosis.

Severe pneumonia/very severe disease (age 2–59 months)

The first dose of intramuscular (IM) ampicillin and gentamicin should be given, and the child referred for treatment as an inpatient with injectable penicillin or ampicillin for severe pneumonia or injectable penicillin or ampicillin plus gentamicin for very severe disease. Where referral is not possible, continue IM ampicillin (50 mg/kg every 6 hours) and IM gentamicin (7.5 mg/kg, once per day) for 5 days; then, if the child responds well, complete treatment at home or in hospital with oral amoxicillin (25 mg/kg, twice per day) plus IM gentamicin once daily for a further 5 days. Where referral is not possible and injection not available, treat with oral amoxicillin for 5 days at a dose of 45 mg/kg, twice per day.

Very severe disease (age < 2months)

For the first week of life

The first dose of IM ampicillin or penicillin and gentamicin should be given and the patient referred for inpatient treatment. Where referral is not possible, continue IM ampicillin (50 mg/kg) or penicillin (50 000 units/kg per dose) every 12 hours and gentamicin (5 mg/kg per day once per day) for up to 10 days.

For weeks 2–4 of life

The first dose of IM ampicillin or penicillin and gentamicin should be given and the patient referred for inpatient treatment. Where referral is not possible, continue IM ampicillin (50 mg/kg) or penicillin (50 000 units/kg per dose) every 8 hours and gentamicin (7.5 mg/kg per day once per day) for up to 10 days.

Prevention

Efforts must be made to improve early diagnosis and treatment with efficacious antibiotics, particularly through raising community awareness, health education on early danger signs, developing mobile clinics and training health-care workers. Adequate nutrition is important. co-trimoxazole prophylaxis should be used to prevent HIV-related infections in adults and children (*4*). Ensure adequate hygiene, hand-washing, and household ventilation.

Immunization

Immunization against Hib (*Haemophilus influenzae* b), measles, pertussis and pneumococcal conjugate reduces the impact of ALRI. Pneumococcal conjugate

is not yet part of national immunization guidelines in Côte d'Ivoire; there are plans to introduce Hib.

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4. *Guidelines on co-trimoxazole prophylaxis for HIV-related infections among children, adolescents and adults. Recommendations for a public health approach.* Geneva, World Health Organization, 2006 (http://www.who.int/hiv/pub/guidelines/ctx/en/index.html, accessed June 2009).

Further reading

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CD-WGE technical focal point: Department of Child and Adolescent Health and Development (CAH)

AFRICAN TRYPANOSOMIASIS (SLEEPING SICKNESS)

Description

Clinical description

A protozoal infection that leads to body wasting, somnolence, coma and death. The disease is always fatal without treatment.

- First stage (haemolymphatic involvement):
  - A painful chancre (papular or nodular) at the primary site of a tsetse fly bite (rare in chronic illness).
  - Possibly fever, intense headache, insomnia, painless lymphadenopathy, anaemia, local oedema and rash.
- Second stage (neurological involvement):
  - Parasites cross the blood–brain barrier and attack the central nervous system.
  - Cachexia, somnolence and signs of central nervous system involvement.

The disease may last for several months or even years.

Infectious agent

Protozoan: *Trypanosoma brucei gambiense* (chronic illness) and *T. b. rhodesiense* (acute illness). The latter occurs in eastern and southern Africa; as it has not been recorded in Côte d'Ivoire, it will not be discussed in detail here.

Case definition

Suspected case:* any case, **without direct demonstration of the parasite** compatible with the clinical description **and/or** with positive serology.

Confirmed case: a case with direct demonstration of the parasite, whether or not compatible with the clinical description.

> **First stage:** parasite seen in blood and/or lymph nodes, with cerebrospinal fluid (CSF) containing no detectable trypanosomes and a leukocyte count ≤ 5/ml.
>
> **Second stage:** parasite seen in blood and/or lymph nodes, with CSF containing trypanosomes and/or a leukocyte count > 5/ml.

** In the first stage or early in the second stage of the disease, there are often no clinical signs or symptoms classically associated with the disease. Suspicion is then based on the local risk of contracting the disease and on the local historical background of the disease.*

Laboratory

- Serological (for screening):
- Card agglutination trypanosomiasis test (CATT): for *T.b. gambiense* only. A negative CATT result does not exclude trypanosomiasis as the test is not 100% sensitive; a positive result must be confirmed by microscopy as CATT is not 100% specific.
- Parasitological (for diagnosis): detection (by microscopy) of trypanosomes in blood, lymph node aspirates or CSF.

Mode of transmission

The disease is transmitted primarily through the bite of an infected tsetse flie (*Glossina* spp). Transmission is also possible through contamination during an infected blood transfusion or through the placenta (congenital).

Incubation period

T. b. gambiense infection has a long incubation period that can last 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 (5–21 days after the infective bite). Parasitaemia occurs in waves of varying intensity in untreated cases during all stages of the disease. Once infected, the tsetse fly remains infective for life (1–6 months).

Reservoirs

Humans are the major reservoir of *T. b. gambiense* infection. The role of domestic and wild animals is not clear.

Epidemiology

Disease burden

T. b. gambiense causes the chronic form of African trypanosomiasis, a neglected tropical disease. *T. b. gambiense* is endemic in Côte d'Ivoire and was first reported

in 1934. Large-scale control programmes in the region nearly eliminated the disease in the 1960s, but it lingered on in the forest zone, essentially in Guinea (Boké, Labé, Kissidougou) and Côte d'Ivoire (Man, Danané and Daloa in the Centre West, and Abengourou in the South East), where it receded slowly. The number of cases of the disease rose steadily between the 1970s and 1990s. Over the last 10 years, the roll out of the Sleeping Sickness National Control Programme (SSNCP) has resulted in a marked decrease in the numbers of new cases: less than 100 cases were reported per year between 2000 and 2007 (Table 1).

Table 1. **Annual detection of cases of African trypanosomiasis, Côte d'Ivoire, 2000–2007**

Year	Annual No. of cases screened	Annual No. of cases reported
2007[a]	—	13
2006	—	29
2005	—	40
2004	10 878	72
2003	14 019	51
2002	11 401	92
2001	8 071	84
2000	23 913	169

Source: *Weekly epidemiological record* 2006, 81:69–80 (http://www.who.int/wer/2006/wer8108.pdf; accessed August 2009).

[a] World Health Organization, unpublished data.

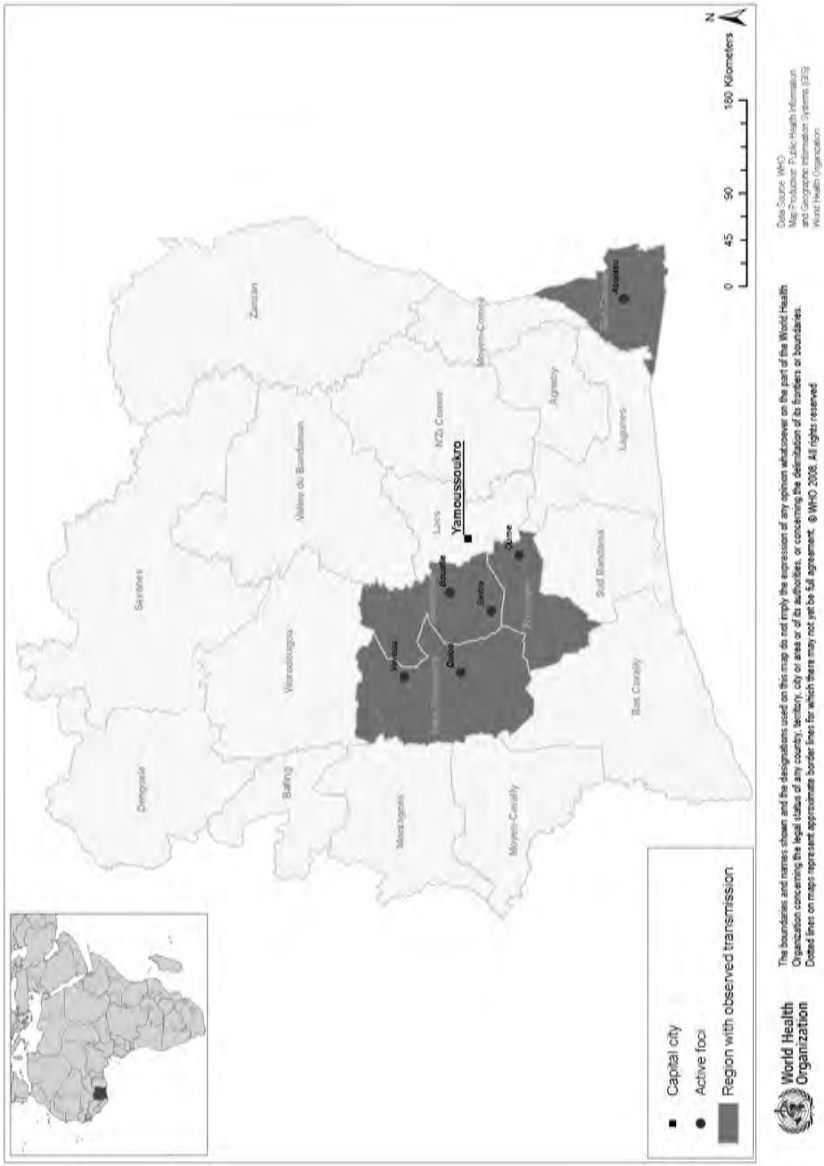
Geographical distribution

The disease is confined to tropical Africa between 15° North and 20° South (corresponding to the distribution of the tsetse fly). *T. b. gambiense* is considered to be endemic in 24 sub-Saharan countries, although some countries have not reported any cases in recent years. Sleeping sickness (including *T. b. rhodesiense*) threatens more than 60 million people, of which only 3–4 million are under surveillance, with regular examination or access to a health centre that can provide screening.

African trypanosomiasis occurs in circumscribed zones, and observed prevalence rates vary greatly from one geographical area to another, even between villages within the same area. In west Africa, endemic African trypanosomiasis (*T. b. gambiense*) has shifted from north to south over the last century. Most of the

northern foci that produced the largest numbers of cases of African trypanosomiasis (Senegal, the Niger basin in Mali, the Volta foci in Burkina Faso, Niger) are no longer active. Guinea and Côte d'Ivoire are the most affected countries at present, with foci in coastal areas (littoral Guinea), or within the forest or forest–savannah

Fig. 2 **Geographical distribution of African trypanosomiasis by region, Côte d'Ivoire, 2008**



transition zones (centre-west Ivorian foci). The disease appears to be limited to areas with > 1200 mm annual rainfall. Border zones favour development of the disease.

At present, active foci of transmission in Côte d'Ivoire have been identified in the centre-west of the country (Oume in Fromager, Daloa and Vavoua in Haut Sassandra, and Bouafle and Sinfra in Marahoué) and the south-east (Aboisso in Sud Camoé) (Fig. 2). The SSNCP, an administrative structure without the logistic capacity to perform surveys, is technically assisted by two national structures (the *Projet de recherché cliniques sur la trypanosomiase,* based in Daloa, and the *Institut Pierre Richet,* based in Abidjan).

Seasonality

The disease has no clear seasonal pattern.

Outbreaks

Outbreaks occur when human–fly contact is intensified, when reservoir hosts introduce virulent strains into a tsetse-infested area or when populations are displaced into endemic areas.

Risk factors for increased burden

Population movement

Displacement of human populations into endemic areas increases the risk of transmission. Movement of infected flies or reservoir hosts may introduce virulent trypanosome strains into a tsetse-infested area.

Overcrowding

Tsetse habitats are often disturbed by an increase in population density thereby reducing fly density and reducing risk of transmission.

Poor access to health services

Systematic population screening is necessary, particularly for *T. b. gambiense* infection. This runs a chronic course and the symptoms evolve slowly and insidiously and may go unnoticed. Poor access to well-trained and equipped health-care personnel further delays diagnosis.

Food shortages

Indirectly, foraging, fishing and hunting in the forest and near rivers increases human–fly contact and consequently the risk of the disease.

Lack of safe water, poor hygienic practices and poor sanitation

The search for water may lead people into tsetse habitats, increasing the risk of the disease. The tsetse fly is not attracted by dirty water.

Prevention and control measures

Case management

Early screening and diagnosis are essential, as treatment is easier during the first 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).

Diagnosis and treatment require trained personnel; self-treatment is not possible. All confirmed cases must follow a lumbar puncture to determine the stage of the disease and should be treated as soon as possible.

Most available drugs have been in production for many years, are difficult to administer in poor conditions, and frequently unsuccessful in curing the disease.

First stage:

- **Pentamidine** – 4 mg/kg per day intramuscular (IM) for 7 consecutive days on an outpatient basis.

Second stage:

- **Eflornithine** – hospitalization with 100 mg/kg slow intravenous (IV) infusion over 2 hours given every 6 hours for 14 days.
- **Melarsoprol** – hospitalization with injections of 2.2 mg/kg per day administered IV for 10 consecutive days. Melarsoprol causes reactive encephalopathy in 5–10% of patients, with a fatal outcome in about half the cases. Increasing rates of resistance to melarsoprol (as high as 25%) have been reported in some areas in *T. b. gambiense* foci in southern Sudan, Kasai and Equateur provinces in the Democratic Republic of the Congo (DRC) and foci in northern Uganda and northern Angola. The drug must be administered in hospital settings and in intensive-care units if possible.
- In case of treatment failure with one drug, use the other.

Since 2001, a public–private partnership signed by WHO has made all drugs for the treatment of African trypanosomiasis widely available. The drugs are donated to WHO and requests for supplies are made to WHO by governments of disease-

endemic countries and organizations working in association with these governments. All the drugs are provided free of charge: recipient countries pay only the transport costs and customs charges. Drug donations to WHO are sufficient to meet global needs until 2011.

Prevention

Routine preventive measures through public education on the following topics should be encouraged:

- Avoidance of known foci of sleeping sickness and/or tsetse infestation.
- Wearing suitable clothing (including long sleeves and long trousers) in endemic areas.
- Routine use of insect repellents and mosquito nets.

Case detection and containment of the human reservoirs through periodical population screening and chemotherapy of cases remain the cornerstones of disease control for *T. b. gambiense* sleeping sickness. Active periodical screening (active case-finding) of the population of endemic foci by mobile screening teams is the best option, since infected subjects can remain asymptomatic and contagious for months or years before developing overt symptoms. Screening usually comprises CATTs of the entire population visited by teams.

Tsetse-fly vector-control programmes with the application of residual insecticides:

- Through community use of insecticide-impregnated traps and screens; or
- Through aerial spraying.

Destruction of tsetse habitats by selective clearing of vegetation: clearing bushes and tall grasses around villages is useful when peridomestic transmission occurs. Indiscriminate destruction of vegetation is NOT recommended.

Immunization

None available.

Further reading

Human African trypanosomiasis. Vector control. Geneva, World Health Organization, Internet document, updated 2009 (http://www.who.int/trypanosomiasis_african/vector_control/en/index.html, accessed June 2009).

CD-WGE technical focal point: Department of Control of Neglected Tropical Diseases (NTD)

BACILLARY DYSENTERY (SHIGELLOSIS)

Description

Clinical description

Bacillary dysentery (shigellosis) is an acute bacterial infection involving the large and distal small intestines and is characterized by small-volume loose stools. Typically, the stools contain blood and mucous diarrhoea (dysentery), and are associated with fever, nausea, vomiting, abdominal cramps and rectal pain (tenesmus). Asymptomatic and mild infections occur, many cases presenting as watery diarrhoea. Uncomplicated disease is usually self-limited and resolves in 4 to 7 days. Complications include intestinal perforation, toxic megacolon, rectal prolapse, haemolytic uraemic syndrome and convulsions (in young children).

Disease severity and case fatality vary with the host (age, nutritional status) and the bacterial serotype. The case-fatality ratio (CFR) is estimated to be < 1% amongst those whose illness is not so severe as to require treatment in hospital. However CFRs can reach 15%. More severe disease and greater risk of death is likely to be seen in infants and adults aged > 50 years, children who have not been breastfed, children recovering from measles, the malnourished or any patient who develops dehydration, unconsciousness, hypo- or hyperthermia or presents with a history of convulsions.

Infectious agent

Bacterium: genus *Shigella* includes four species, *S. dysenteriae, S. flexneri, S. boydii* and *S. sonnei*, also designated as serogroups A, B, C and D respectively. Each serogroup is further subdivided into serotypes.

S. sonnei and *S. boydii* usually cause relatively mild illness.

Although *S. flexneri* is the chief cause of endemic shigellosis in developing countries, *S. dysenteriae* serotype 1 (Sd1) causes the most severe disease and is usually responsible for epidemics that are often large, even regional.

Case definition

Suspected case: Diarrhoea with visible blood in the stools.

Confirmed case: A suspected case with isolation of *Shigella* from stools.

Mode of transmission

Faecal–oral route, particularly contaminated water and food. The infectious dose of microbes in humans is very low (10–100 organisms). The organism can also be transmitted by flies. Food stalls are a common source of contaminated meals.

Incubation period

The incubation period is usually 1–3 days, and may be up to 1 week for Sd1.

Period of communicability

During acute infection and up to 4 weeks after illness (without treatment); 2–3 days with appropriate treatment. Asymptomatic carriers exist.

Reservoirs

Humans are the only significant reservoir, although outbreaks have occurred among primates.

Epidemiology

Disease burden

Globally, shigellosis is estimated to cause 80 million cases of bloody diarrhoea and 700 000 deaths per year. About 99% of infections occur in developing countries and about 70% of cases and 60% of deaths occur in children under the age of 5 years. Illness in infants aged less than 6 months is unusual. In endemic areas, the disease is more severe in young children than in adults, among whom many infections may be asymptomatic. Unlike in east Africa, outbreaks of shigellosis have not been reported in Côte d'Ivoire. Only sporadic cases have been documented, particularly in the rainy season. In west Africa, shigellosis is less common than salmonellosis or vibriosis.

Geographical distribution

Worldwide: Shigellosis is endemic in temperate and in tropical climates. Multidrug-resistant *Shigella* with considerable geographical variations has appeared worldwide in relation to the widespread use of antimicrobial agents. In Côte d'Ivoire, diarrhoeal illnesses occur throughout the country, but persons living in rural areas and in the western regions are particularly vulnerable.

Seasonality

No data available.

Alert threshold

In the absence of a clear epidemic threshold, an epidemic should be suspected if:

- There is an unusual and sudden rise in the number of new cases or deaths due to bloody diarrhoea reported weekly;
- There is an increase in the proportion of bloody diarrhoea among diarrhoeal cases; or
- There are five or more linked cases of bloody diarrhoea.

Any of the above scenarios should lead to investigation of the disease agent by laboratory testing.

Outbreaks

Sd1 has the potential to cause epidemics with a high rate of morbidity and mortality. Susceptibility is general. The rate of secondary attacks in households can be as high as 40%.

Risk factor for increased burden

Population movement

Complex emergencies and natural disasters such as flooding, resulting in large population movements and overcrowded refugee camps, are classic settings for explosive outbreaks.

Overcrowding

Overcrowding increases the risk of infection. The risk of epidemics of Sd1 is high in camp settings (up to one third of the population at risk may be affected).

Poor access to health services

Early detection and containment of cases are vital in reducing transmission. Lack of reporting mechanisms for outbreaks and poor surveillance and monitoring are further obstacles to the effective prevention and control of this disease.

Food shortages

Malnourished people of all ages are susceptible to severe disease and death. Breastfeeding of infants and young children is protective.

Lack of safe water, poor hygienic practices and poor sanitation

Lack of safe water, poor sanitation, poor hygiene, lack of soap are all important risk factors, and can lead to contamination of food items, especially from food stalls.

Prevention and control measures

Case management

Early and appropriate therapy is very important; treatment with an effective antimicrobial can reduce the severity and duration of shigellosis. Selection depends on resistance patterns of the bacteria and on drug availability. See Table 2 for treatment recommendations.

The problem of rapid acquisition of antimicrobial resistance in treating Sd1 in Africa is a cause of concern. It is therefore important to confirm the susceptibility of *S. dysenteriae* to antibiotics in the early stages of an outbreak. Resistance patterns may vary during the course of an outbreak and regular stool sampling is required. Ciprofloxacin is the current first-line antibiotic of choice recommended for treatment of Sd1.

Data on antibiotic resistance in Côte d'Ivoire are not readily available.

Table 2. **Recommendations for treatment of *Shigella dysenteriae* type 1 (Sd1)**

Patient group	Treatment [a]	Dose	Dosing frequency	Duration of treatment
Adults	Ciprofloxacin	500 mg	Twice per day	3 days
Children	Ciprofloxacin	250 mg/15 kg	Twice per day	3 days
Children aged less than 6 months	Add zinc	10 mg	Daily	2 weeks
Children aged 6 months to 12 years	Add zinc	20 mg	Daily	2 weeks

Source: *First steps for managing an outbreak of acute diarrhoea*. Geneva, World health Organization, 2004 (*http://www.who.int/topics/cholera/publications/first_steps/en/index.html*, accessed June 2009).

[a] Rapidly evolving antimicrobial resistance is a significant problem; Sd1 is usually resistant to ampicillin and trimethoprim–sulfamethoxazole (TMP–SMX).

Supportive treatment using oral rehydration salts (ORS), continued feeding (frequent small meals) and antipyretics to reduce high fever. Continue breastfeeding infants and young children who are breastfed.

Sd1 is often more severe or fatal in young children, the elderly and the malnourished: prompt treatment with antibiotics is essential. If in short supply, antibiotics should be reserved for high-risk groups.

Prevention

The prevention of shigellosis relies mainly on adequate water, sanitation, and hygiene in the community (see *Diarrhoeal diseases [others):* Prevention; and Annex 4: *Safe water and sanitation*).

Immunization

Not available. Several vaccine candidates are under development.

Epidemic control

Inform the health authorities if one or more suspected cases are identified. Early detection and notification of epidemic dysentery, especially among adults, allow timely mobilization of resources needed for appropriate case management and control.

Confirm the outbreak, following WHO guidelines (*1*).

Rectal swabs from suspected cases should be collected and immediately transported in an appropriate medium (e.g. Cary-Blair) maintaining cold chain (0–4 °C), to a laboratory for culture to confirm the diagnosis of Sd1. Ideally, the specimen should be analysed within 2 hours. The viability of bacteria in this medium when refrigerated for 1–3 days is very variable and the findings of laboratory analyses carried out on such samples are unreliable. It is therefore recommended that 10–20 samples are used to confirm the outbreak, the pathogen strain and antibiotic susceptibility. Fresh stool samples can be sent for analysis if Cary-Blair medium is not available, but the sample must reach the laboratory and be processed within 6 hours. Once the outbreak is confirmed, it is not necessary to obtain laboratory confirmation for every patient.

Testing of Sd1 isolates for antimicrobial susceptibility should be done at regular intervals to determine whether treatment guidelines remain appropriate. International referral laboratories are available to assist in identification of the organism and confirmation of the antimicrobial resistance pattern.

Do not wait for laboratory results before starting treatment/control activities

Patients with known *Shigella* infections should not be employed to handle food or to provide child or patient care. Patients must be informed of the importance and effectiveness of hand-washing with soap and water after defecation as a means of curtailing transmission.

References

1. *Cholera outbreak: assessing the outbreak response and improving preparedness.* Geneva, World Health Organization, 2004 (WHO/CDS/CPE/ZFK/2004.4; http://www.who.int/topics/cholera/publications/cholera_outbreak/en/index.html, accessed July 2009)

Further reading

First steps for managing an outbreak of acute diarrhoea. Geneva, World Health Organization, 2003 (WHO/CDS/CSR/NCS/2003.7 Rev 1) (*http://www.who.int/cholera/publications/first_steps/en/index.html*, accessed June 2009).

Acute diarrhoeal diseases in complex emergencies: critical steps. Geneva, World Health Organization, 2004 (WHO/CDS/CPE/ZKF/2004) (http://www.who.int/topics/cholera/publications/critical_steps/en/index.html, accessed June 2009).

Guidelines for the control of shigellosis, including epidemics due to Sd1. Geneva, World Health Organization, 2005 (*http://www.who.int/topics/cholera/publications/shigellosis/*, accessed June 2009).

The treatment of diarrhoea. A manual for physicians and other senior health workers. Geneva, World Health Organization, 2005 (http://*www.who.int/entity/child_adolescent_health/documents/9241593180/en/*, accessed June 2009).

Interagency diarrhoeal disease kits - information note. Geneva, World Health Organization, February 2006 (http://www.who.int/topics/cholera/materials/en/, accessed June 2009).

Emerging issues in water and infectious disease. [Internet document] (http://www.who.int/water_sanitation_health/emerging/en/, accessed June 2009).

CD-WGE technical focal point: Department of Public Health and Environment (PHE)

BURULI ULCER (*Mycobacterium ulcerans*)

Description

Clinical description

Buruli ulcer classically presents as a chronic painless skin ulcer with undermined edges and a yellow-white necrotic base, or as a large area of induration or diffuse swelling of the legs and arms. Half of those affected are children living near wetlands. The disease progresses without pain or fever. Without treatment, massive ulcers result, sometimes with bone involvement, resulting in gross deformities. When lesions heal, scarring may cause restricted movement of limbs and other permanent disabilities in about a quarter of patients.

Infectious agent

Bacterium: *Mycobacterium ulcerans*

Case definition

Suspected case:

A person presenting in an endemic area with a skin lesion with the following features:

Active case: Non-ulcerative: papules, nodules, plaques, and oedema;

Ulcerative: skin ulcer with undermined edges, cotton-wool-like appearance, and thickening and darkening of the skin surrounding the lesion.

Inactive case: Evidence of previous infection with a depressed stellate scar.

Confirmed case:

A suspected case confirmed by polymerase chain reaction (PCR) and/or one of the other tests available:

- PCR;
- Demonstration of acid-fast bacilli in a smear stained with Ziel-Nielsen;
- Histopathology;
- Culture.

Mode of transmission

An environmental pathogen, *M. ulcerans* has been detected in soil and water by PCR. Recently, it has also been cultivated from aquatic insects. Transmission is probably by exposure to an infected environment; some patients report antecedent trauma. Research is underway to explore the possibility of transmission via insects.

Incubation period

Usually 2–3 months, but the bacterium may remain latent for several years.

Period of communicability

Human-to-human transmission is extremely rare.

Reservoirs

Uncertain. Water-dwelling insects, snails and fish are naturally infected and may serve as natural hosts for *M. ulcerans*.

Epidemiology

Disease burden

An emerging disease, Buruli ulcer is the third most common illness caused by mycobacteria occurring in immunocompetent individuals worldwide. Buruli ulcer is endemic in Côte d'Ivoire and about 2000 cases are reported each year; Côte d'Ivoire thus has the highest prevalence of disease caused by *M. ulcerans* in the world. Although the associated mortality is low, the destruction of skin and soft tissue leads to serious deformity and functional disabilities with resultant social and economic problems for the patient. The first case was reported in 1978 and *M. ulcerans* has since become increasingly recognized as a health problem (Table 3). In 1997, a nationwide survey identified 10 382 cases.

Table 3. **Annual number of reported cases of Buruli ulcer in Côte d'Ivoire**

Year	Annual number of reported cases
2007*	2191
2006[a]	1872
2005	1741
2004	1170
2003	1235
2002	No reported cases
2001	1712
2000	1574
1999	1351

Source: Global Buruli Ulcer Initiative (http://www.who.int/buruli/information/Buruli%20ulcer_WER_2008.pdf; http://www.afro.who.int/buruli/index.html).

[a] Annual meeting on Buruli Ulcer, Geneva, World Health Organization, 2008 (unpublished data).

Geographical distribution

The disease has been reported in more than 30 countries worldwide and is largely a tropical and subtropical disease, although it has also been seen in temperate Australia. In Africa, Buruli ulcer is endemic, particularly between latitudes 10° north and south, including in Côte d'Ivoire and neighbouring countries. Potential high-risk areas are found near irrigated rice fields, banana plantations, and dams for irrigation and acquaculture.

Seasonality

No seasonal variations have been observed. Increased rates are seen with flooding.

Outbreaks

None formally recorded, but may occur in settings of environmental change that promote flooding (e.g. dam construction and deforestation).

Risk factors for increased burden

Population movement

Movement into rural wetlands in endemic countries may increase exposure to infection.

Overcrowding

Not a risk factor.

Poor access to health services

Delayed presentation of chronic ulcer leads to increased morbidity and disability.

Food shortages

Not a risk factor.

Lack of safe water, poor hygienic practices and poor sanitation

Secondary bacterial infection is associated with poor hygiene and poor wound care.

Prevention and control measures

Case management

Early diagnosis and treatment is essential; community education and training peripheral health workers is an important part of any strategy to reduce the impact of Buruli ulcer.

Diagnosis can be made clinically by experienced field-workers in endemic areas; confirmatory laboratory diagnosis should be made whereever possible.

Current recommendations for treatment are as follows:

- A combination of rifampicin and streptomycin/amikacin for 8 weeks as a first-line treatment for all forms of the active disease. Nodules or uncomplicated cases can be treated without hospitalization.
- Surgery to remove necrotic tissue, cover skin defects and correct deformities.
- Interventions to minimize or prevent disabilities.

Cumulative experience of treating about 300 patients in Benin, Cameroon and Ghana has shown that treatment with rifampicin and streptomycin (RS) for 8 weeks according to WHO guidelines leads to complete healing of nearly 50% of skin lesions. Some patients can be treated on an ambulatory basis. Recurrence after antibiotic treatment is less than 2% compared with 16–30% with surgical treatment alone. These encouraging developments are changing the strategy for the control and treatment of Buruli ulcer which, until 2004, focused on surgical treatment.

Epidemic control

Uncommon, but measures include early surveillance reporting, wound care and education.

Prevention

Wear clothing that covers the extremities. Educate the population so as to encourage earlier presentation, diagnosis and treatment.

Immunization

None currently available. Bacille Calmette–Guérin (BCG) vaccination appears to offer some short-term protection from the disease. Although the protection is limited, ensuring complete coverage of BCG vaccination in affected rural areas may be useful.

Further reading

Provisional guidance on the role of specific antibiotics in the management of Mycobacterium ulcerans disease (Buruli ulcer). Geneva, World Health Organization, 2004 (*http://whqlibdoc.who.int/hq/2004/WHO_CDS_CPE_GBUI_2004.10.pdf*, accessed June 2009).

CD-WGE technical focal point: Department of Control of Neglected Tropical Diseases (NTD)

CHOLERA

Description

Clinical description

Cholera is an acute bacterial infection of the intestine, characterized in its severe form by sudden onset of painless watery diarrhoea ("rice-water" stool) and vomiting, which can result in rapid severe dehydration. Both children and adults can be infected.

Infectious agent

Bacterium: *Vibrio cholerae* O1 (classical and El Tor) and O139.

Case definition

Suspected case:

A **cholera outbreak** should be suspected if:

- A person aged more than 5 years develops severe dehydration or dies from acute watery diarrhoea (clinical case); **or**
- 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.

Confirmed case:

Isolation of *V. cholerae* O1 or O139 from stools in any patient with diarrhoea. A new rapid diagnostic test is currently being evaluated.

Mode of transmission

Mainly faecal–oral route

- Drinking contaminated water, including accidental ingestion of contaminated surface water.
- Eating food (fruits and vegetables) contaminated by water, soil, or contaminated during preparation (e.g. rice, millet, food from street vendors) or contaminated seafood.
- Person to person:

- When taking care of patients with cholera;
- Via direct contact with the bodies of deceased patients with cholera (e.g. washing the body for funeral ceremonies).

- Indirect contamination (hands) with poor hygiene practices and lack of soap. Wound infections can arise from environmental exposure, especially by brackish water from occupational accidents among fishermen.

Incubation period

From a few hours to 5 days; usually 2–3 days.

Period of communicability

During the symptomatic phase until 2–3 days after recovery; very rarely, for months. Asymptomatic carriers are common.

Reservoirs

The main reservoir is humans, though recent environmental reservoirs have been found in association with copepods or other zooplankton in brackish water or estuaries.

Epidemiology

Disease burden

In 2007, WHO recorded 177 963 cases globally (4031 deaths; case-fatality ratio, CFR, 2.3%), 93% of which were in Africa (166 583 cases; 3994 deaths; CFR, 2.4%). Of these, 18 354 cases were reported from west Africa (458 deaths; CFR, 2.5%). West Africa had a large surge in cholera cases in 2005 with 76 881 cases being reported, decreasing to 17 419 in 2006. In Côte d'Ivoire, an increase in the number of cases was not reported until 2006 (Table 4). Globally, the true number of cholera cases is much higher than reported owing to under-reporting, inconsistencies in the case definition, and lack of a standardized reporting techniques.

Table 4. **Morbidity, mortality and case-fatality ratio due to cholera, Côte d'Ivoire, 2001–2007**

Year	Total No. of cases (including imported cases)	Deaths	Case-fatality ratio (CFR)
2007	8	1	12.5
2006	414	16	3.6
2005	39	6	15.4
2004	105	9	8.6
2003	1034	50	4.8
2002	4188	143	3.4
2001	5912	305	5.2

Source: Cholera, 2007. *Weekly Epidemiological Record* 2008, 83:261–284 (http://www.who.int/wer/2008/wer8331/en/index.htm, accessed July 2009).

Geographical distribution

Cholera is endemic throughout Côte d'Ivoire.

Seasonality

Generally, cases are distributed throughout the wet season starting in May–June. Climate fluctuations related to warming of oceans, such as *El Niño*, can be associated with an increase in the incidence of cholera.

Alert threshold

Any suspected case must be investigated.

Outbreaks

Outbreaks can occur where water supply, sanitation, food safety, and hygiene are inadequate.

The greatest risk occurs in over-populated communities and refugee settings characterized by poor sanitation and unsafe drinking-water, with a greater risk of person-to person transmission. Owing to the very short incubation period (2 hours to 5 days), the number of cases can rise extremely quickly.

Risk factors for increased burden

Population movement

Important for transmission of the infectious agent.

Overcrowding

Overcrowding increases the risk of contact with infected vomitus, excreta and contaminated food or water.

Poor access to health services

Lack of health care and proper case management increase the risk of case fatality. Early detection and containment of cases (isolation facilities) are vital in reducing transmission. Lack of reporting mechanisms for outbreaks, poor surveillance and monitoring are obstacles to effective prevention and control of disease.

Food shortages

Malnutrition increases the risk of severe diarrhoeal illness with marked fluid and electrolyte disturbances. This in turn, contributes to further malnutrition. Prolonged diarrhoea in malnourished patients may result in a large increase in fluid, electrolyte and nursing needs.

Lack of safe water, poor hygienic practices and poor sanitation

The most important risk factors. Cultural practices may also be a risk factor, e.g. funeral practices (which include meals prepared by those who have prepared the body without stringent hygiene) can be particularly hazardous.

Prevention and control measures

Case management

About 20% of those who are infected develop acute, watery diarrhoea; 10–20% of these individuals develop severe watery diarrhoea with vomiting. If these patients are not promptly and adequately treated, the loss of such large amounts of fluid and salts can lead to severe dehydration and death within hours. The CFR in untreated cases may reach 30–50%. Treatment is straightforward (basically, rehydration) and, if applied appropriately, should keep CFRs below 1%.The mainstay of the case management of cholera is the treatment of dehydration using oral rehydration salts (ORS) or intravenous (IV) fluids, such as Ringer's lactate (*1*). Large volumes

of ORS can be required. IV rehydration should be used for severe cases only. See also Annex 4: *Safe water and sanitation*).

Use of antibiotics (doxycycline/tetracycline) is not essential for the treatment of disease, but may be used in severe cases to reduce the volume of diarrhoea (and of the rehydration solutions required), shorten its duration and the period during which *V. cholera* is excreted. The pattern of susceptibility to antimicrobials should be assessed in order to select the most appropriate antibiotic. Increasing resistance of *Vibrio* species to cotrimoxozole has been reported from Côte d'Ivoire.

Strict hand-washing practices and proper disinfection of articles and linen used by cholera-infected patients is important to prevent the spread of cholera, as is adequate hygiene during funeral preparations.

Prevention

Provision and use of safe water, adequate sanitation and health education for proper hygiene and food safety are important prevention measures (see *Diarrhoeal dieases [others]*: Prevention; and Annex 4: *Safe water and sanitation*).

Immunization

Implementation of the normal prevention and control measures, including the improvement of water and sanitation, remains the foundation of outbreak prevention and response. The use of oral cholera vaccine (OCV) is considered to be an additional public health tool to the normal recommended cholera prevention and control measures, especially when given pre-emptively if the population at risk can be accurately identified. Currently there is only one WHO pre-qualified oral cholera vaccine: killed whole-cell *V.cholera* O1 recombinant B-subunit of cholera toxoid (WC/rBS) is available for use in public health.

The relevance of OCVs should be assessed by using the WHO decision-making tool on a case-by-case basis (*2*). Old parenteral cholera vaccine should not be used and has never been recommended by WHO.

For more specific information on cholera vaccines and their use, see *Further reading* below or contact the Global Task Force on Cholera Control at WHO/HQ: cholera@who.int.

Epidemic control

As the risk of cholera outbreaks is high in overcrowded settings, preparedness is the key factor for successfully reducing associated mortality. Disease surveillance

systems must be strengthened in order to be sufficiently sensitive to detect major outbreaks. Ideally, cholera treatment units should be prepared before the emergence of an outbreak in high-risk settings.

Inform the health authorities immediately if one or more suspected cases are identified. Confirm the outbreak, following WHO guidelines (*3*).

Stool samples must be taken with a rectal swab and transported in Cary-Blair medium. If a transport medium is not available, a cotton-tipped rectal swab can be soaked in the liquid stool, placed in a sterile plastic bag, tightly sealed and sent to the laboratory. It is recommended that at least 10 cases are used to confirm the outbreak and identify antibiotic susceptibility. Once confirmed, it is not necessary to obtain laboratory confirmation for every patient.

Do not wait for laboratory results before starting case-management and control activities (*4, 5*).

- Ensure prompt case management and confirm the diagnosis.
- Isolate severe cases in cholera treatment centres.
- Provide adequate community involvement, health education and active case finding.
- Set up ORS corners[1] to increase the population's access to oral rehydration.
- Ensure access to safe water and proper sanitation.
- Ensure hand-washing with soap.
- Ensure safe food handling.
- Ensure adequate disinfection and hygiene during funerals.
- Find and treat the source of infection as soon as possible.

Inter-agency diarrhoeal-disease kits may be obtained for preparedness or response (*6*). These kits comprise four separate modules containing the drugs, ORS, documents and other material necessary for management and control of 100 severe cases of cholera in a cholera treatment centre or 400 mild or moderate cases of cholera in an oral rehydration unit. Complete kits are advised for preparedness, but each module may be ordered separately according to the availability of components locally.

All components of the diarrhoeal-diseases kit have a minimum of 75% remaining shelf-life. No cold chain is required. These kits or modules may be purchased

1. Area for patient observation and dispensing of oral rehydration salts (ORS), used to rehydrate patients.

by non-WHO agencies through The Medical Export Group BV (MEG), Gorinchem, The Netherlands (info@meg.nl and www.meg.nl), or by WHO through a usual requisition (product "kit and modules" are available in the WHO catalogues on the intranet under "kits").

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6. *Interagency diarrhoeal disease kits - information note*. Geneva, World Health Organization, February 2006 (http://www.who.int/topics/cholera/materials/en/, accessed June 2009).

CD-WGE technical focal point: Department of Public Health and Environment (PHE)

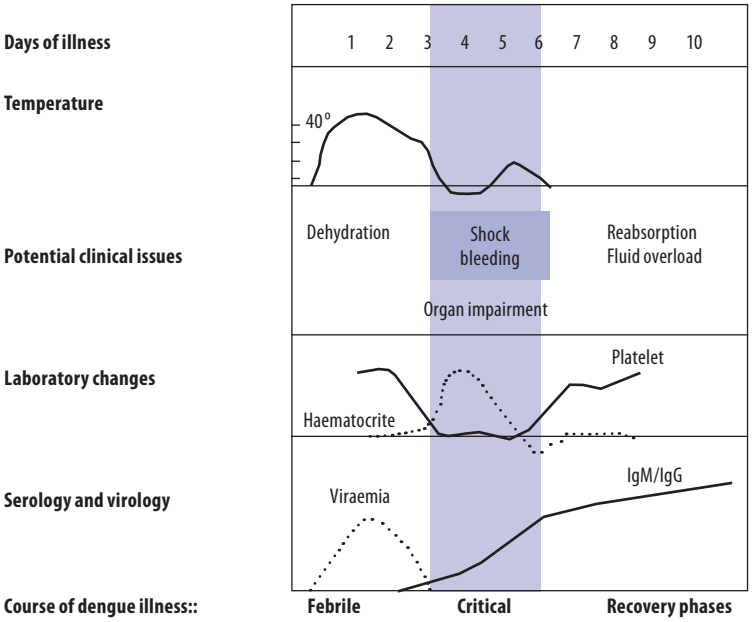
DENGUE

Description

Clinical description

Dengue infection is a systemic and dynamic disease. It has a wide clinical spectrum that includes severe and non-severe forms of clinical manifestations. After the incubation period, the illness begins abruptly and is followed by three phases: febrile, critical and recovery phase (see Fig. 3). A revised clinical classification according to levels of severity (dengue fever +/- warning signs and severe dengue) is proposed to replace dengue fever, dengue haemorrhagic fever (DHF) and dengue shock syndrome (DSS) (see Fig. 4).

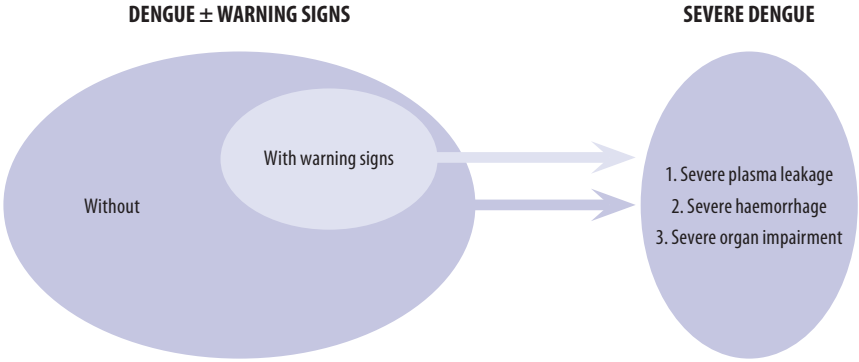
Fig. 3 **Time-course of dengue illness**



Source: *Dengue: Guidelines for diagnosis, treatment, prevention and control. New edition 2009.* Geneva, World Health Organization, 2009 (http://whqlibdoc.who.int/publications/2009/9789241547871_eng.pdf, accessed November 2009).

Haematocrite the same as erythrocyte volume fraction, EVF,

Fig. 4 **Suggested dengue case classification and levels of severity**



CRITERIA FOR DENGUE ± WARNING SIGNS

Probable dengue
live in /travel to dengue endemic area.
Fever and 2 of the following criteria:

- Nausea, vomiting
- Rash
- Aches and pains
- Tourniquet test positive
- Leukopenia
- Any warning sign

Laboratory-confirmed dengue
(important when no sign of plasma leakage)

Warning signs*

- Abdominal pain or tenderness
- Persistent vomiting
- Clinical fluid accumulation
- Mucosal bleed
- Lethargy, restlessness
- Liver enlargment >2 cm
- Laboratory: increase in HCT concurrent with rapid decrease in platelet count

*(requiring strict observation and medical intervention)

CRITERIA FOR SEVERE DENGUE

Severe plasma leakage
leading to:

- Shock (DSS)
- Fluid accumulation with respiratory distress

Severe bleeding
as evaluated by clinician

Severe organ involvement

- Liver: AST or ALT >=1000
- CNS: Impaired consciousness
- Heart and other organs

Source: *Dengue: Guidelines for diagnosis, treatment, prevention and control. New edition 2009.* Geneva, World Health Organization, 2009 (http://whqlibdoc.who.int/publications/2009/9789241547871_eng.pdf, accessed November 2009).

AST, aspartate aminotransferase; ALT, alanine aminotransferase; CNS, central nervous system; DSS, dengue shock syndrome

Febrile phase

This acute febrile phase usually lasts 2–7 days with at least two of the following: rash; anorexia and nausea; aches and pains; leukopenia; positive result for the tourniquet test; any warning sign (abdominal pain or tenderness, persistent vomiting, clinical fluid accumulation, mucosal bleed, lethargy/restlessness, liver enlargement of > 2 cm, increase in erythrocyte volume fraction concurrent with rapid decrease in platelet count). Dehydration is sometimes present; children with high fever may present with febrile convulsions.

Critical phase

Around day 3–7 of illness the temperature drops to 37.5–38 °C or less and remains below this level ("defervescence"). Around time of defervescence, patients can either improve or deteriorate. Those who improve after defervescence have **dengue without warning signs**. Those who deteriorate will manifest warning signs: **dengue with warning signs**. Some of these patients may further deteriorate to severe dengue with severe plasma leakage leading to shock (DSS) with or without respiratory distress, severe bleeding and/or severe organ impairment.

Recovery phase

Gradual recovery takes place in the next 48–72 hours.

Infectious agent

The dengue virus complex comprises four antigenically related but distinct viruses designated dengue virus serotypes 1 to 4. Serotype 2 is most common in west Africa. Recovery from infection by one of the viruses confers lifelong immunity only against that particular virus, and partial and transient immunity to the others. Sequential infection increases the risk of developing DHF.

Case definition

Suspected case: A case compatible with the clinical description.

Probable case:

A case compatible with the clinical description plus:

Supportive serology (reciprocal haemagglutination-inhibition antibody titre ≥ 1280, comparable IgG enzyme immunoassay (EIA) titre or positive IgM antibody test in late acute or convalescent-phase serum specimen);

Occurrence at same location and time as other confirmed cases of dengue fever.

Confirmed case:

A case compatible with the clinical description, confirmed by a laboratory.

Laboratory criteria: at least one of the following:

- Isolation of the dengue virus from serum, plasma, leukocytes, or autopsy samples;
- Demonstration of a ≥ 4-fold change in reciprocal IgG or IgM antibody titres to ≥ 1 dengue virus antigens in paired serum samples;
- Demonstration of dengue virus antigen in autopsy tissue by immunohistochemistry or immunofluorescence or in serum samples by EIA; and/or
- Detection of viral genomic sequences in autopsy tissue, serum or CSF samples by polymerase chain reaction (PCR).

Mode of transmission

Dengue has a human–mosquito–human transmission cycle involving female mosquitoes of the genus *Aedes* (*Stegiomyia*), principally *Aedes aegypti. Ae. furcifer-taylori* complex mosquitoes are involved in enzoonotic monkey–mosquito transmission in west Africa.

Incubation period

Commonly 4–7 days (range, 3–14 days).

Period of communicability

No direct person-to-person spread. Patients are infective for mosquitoes from shortly before the onset of fever to the end of the febrile period (usually 3–5 days). The mosquito becomes infective 8–12 days after the viraemic blood-meal and remains so for life.

Epidemiology

Disease burden

Initially thought to be a sporadic disease with occasional epidemics, the numbers of cases of dengue have risen dramatically since the 1970s and dengue now occurs in more than 100 countries, particularly in South-east Asia, the Americas, the Western Pacific and Africa. Globally, there are an estimated 50 million cases of dengue fever each year, with 2.5 billion persons living in at-risk regions.

Dengue is a poorly recognized illness and is probably under-reported in Côte d'Ivoire. Other than during epidemics of yellow fever, distinguishing between the various arbovirus diseases (dengue, yellow fever etc) and other endemic febrile illnesses (malaria, typhoid etc) is difficult.

Epidemics of dengue have been reported more frequently in east than in west Africa. All four serotypes have caused outbreaks in east Africa and have been detected in various parts of west Africa, including Nigeria, Senegal and Burkina Faso. A few sporadic cases have been reported from Côte d'Ivoire, but most literature on west African dengue focuses on entolomological studies. Dengue surveillance data for Côte d'Ivoire is not currently available. Enhanced global surveillance with participation in DengueNet is encouraged (*1*). An epidemic of dengue occurred in west Africa including Côte d'Ivoire in 1982. No further outbreaks have been recognized in Côte d'Ivoire thus far.

Geographical distribution

Geographical distribution is uncertain. It is expected that urban areas with *Aedes aegypti* mosquitoes would bear the brunt of the burden of disease.

Reservoirs

The viruses are maintained in a human–*Aedes aegypti* mosquito cycle in tropical urban centres. A monkey–mosquito cycle has been demonstrated to occur in forests of South-east Asia and western Africa, but there is no current evidence that this is an important reservoir for transmission to humans.

Seasonality

In hyperendemic areas, dengue occurs in multiannual cycles approximately every 5–6 months owing to a combination of seasonal vector variation (more in the wet season) and a short-lived period of cross immunity between serotypes following each infection.

Alert threshold

Insufficient data to calculate an alert threshold for Côte d'Ivoire.

Epidemics

Large epidemics have occurred globally. An unprecedented pandemic occurred in 1998 when 56 countries reported 1.2 million cases of dengue and DHF.

Risk factors for increased burden

Population movement

Movement of viraemic populations into non-endemic areas where the vector is present will increase dengue transmission. Movement into endemic areas, establishment of new human settlements and creation of temporary shelters (where drinking-water is obtained from outside sources or from rain-water harvesting and storage in household containers) will increase transmission.

Overcrowding

In emergency situations, close proximity of human habitation to water-storage containers and accumulation of water will promote increased breeding sites for *A. aegypti* and increased contact between humans and the vector.

Poor access to health services

Health centres are essential as an alert network and for early diagnosis and treatment of suspected cases. Without proper treatment, case-fatality ratios (CFRs) for DHF can exceed 20%. Breakdown of vector-control activities increases risk of the disease.

Food shortages

Not relevant.

Lack of safe water, poor hygienic practices and poor sanitation

The accumulation of water in discarded vessels and debris may become vector-breeding sites.

Prevention and control measures

Case management

Treatment of dengue is limited to supportive care, including rehydration and antipyretics. There are no effective antiviral treatments for dengue (*2*).

Dengue fever in an ambulatory patient who is stable and able to tolerate oral fluids should be managed at home with fever control (paracetamol and tepid sponging; non-steroidal anti-inflammatory drugs – NSAIDs – such as aspirin are contraindicated), destruction of local breeding sites, and daytime sleeping under an insecticide treated net. Patients should be reviewed daily, and instructed to return urgently in the presence of warning signs.

Dengue fever with warning signs, or in the presence of other factors such as pregnancy, extremes of age, or co-existing illness, should be managed as inpatient with careful intravenous rehydration with isotonic solution as required. Care must be taken to watch for and avoid over-hydration.

Severe dengue fever (*3*) should be referred as a medical emergency for intensive inpatient treatment. Blood transfusion may be required if severe bleeding.

Prevent access of day-biting mosquitoes to patients with screens or insecticide-treated nets, and spray the facility with residual insecticide, to reduce onward transmission to the susceptible population.

Prevention

The most effective, economical and safe method is integrated vector control, including reduction of mosquito breeding sites, public education, biological and chemical control, vector and disease surveillance, provision of potable water, effective sanitation and solid waste management (*4*).

Biological and chemical control measures target either the larvae or the adults. In urban areas, *Aedes* mosquitoes breed on water collections in artificial containers such as plastic cups, used tyres, broken bottles, flower pots, etc. Continued and sustained artificial container reduction or covering or periodic draining of artificial containers is the most effective way of reducing the larvae and thereby the *Aedes* mosquito load in the community. Larvicides are also highly effective but should be long-lasting and preferably be safe for use in drinking-water (e.g. temephos). Insect growth-regulators are also available and are both safe and long-lasting e.g. pyriproxyfen. Biological control includes *Bacillus thuringiensis* H-14 (BTI), the crustacean *Mesocyclops* and several species of larviforous fish. For reducing the adult mosquito load, space spraying with insecticide from trucks or aircraft can be effective, although only transiently.

Where insecticides or larvicides are used, regular monitoring of vector susceptibility is necessary to ensure the appropriate choice of chemical.

All mosquito-control efforts should be accompanied by active monitoring and surveillance of the natural mosquito population in order to determine the impact of the programme.

Aedes are chiefly day-biting, with increases in biting rates after sunrise and before sunset. Prevention of mosquito bites can be achieved using long-sleeved clothing, repellents based on *N,N*-diethyl-meta-toluamide (DEET), insecticide-treated mosquito nets, fly-wire screening of windows and mosquito coils or vapour mats. None of these are sufficiently effective when used in isolation.

Piped water supplies, well-maintained drainage and efficient systems for removal of domestic waste also have a large impact.

Immunization

There is no commercially available vaccine for dengue. Several vaccine candidates are in development.

Epidemic control

The mainstay of epidemic control is vector control (*5*). Eliminate larval habitats of *Aedes* mosquitoes in urban or peri-urban areas. All stored water containers should be kept covered at all times. Empty water from coolers, tanks, barrels, drums, and buckets. There should be no water in coolers when not in use. Remove water every other day from sites where water unavoidably accumulates and cannot be covered e.g. refrigerator drip pans. Apply larvicide to other potential larval habitats.

Space spraying of insecticide from trucks or aeroplanes to kill adult mosquitoes is considered in some settings, with appropriate entomological advice and monitoring.

A concurrent public information campaign is important.

Coordinated multidisciplinary care is required with facilities set aside for a large incoming case load. Triage will be required and a case definition, clinical protocols, information and education for staff, and provision of sufficient supplies. Hospital beds should be screened with insecticide-treated nets and/or spray the hospital with indoor residual insecticide.

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CD-WGE technical focal point: Department of Global Alert and Response (GAR) + Special Programme for Research and Training in Tropical Diseases (TDR) + Department of Control of Neglected Tropical Diseases (NTD)

DIARRHOEAL DISEASES (OTHERS)

Description

Clinical description

Acute diarrhoeal illness is a symptom of infection by many different viral, bacterial and parasitic infectious agents and often presents with other clinical symptoms such as nausea, vomiting and fever. Diarrhoeal diseases can clinically manifest as:

- Acute watery diarrhoea;
- Acute bloody diarrhoea (dysentery); and
- Persistent diarrhoea, lasting 14 days or longer.

In Côte d'Ivoire, a high case-fatality ratio (CFR) exists regardless of cause. Infants of under the age of 5 years are particularly vulnerable; malnutrition and human immunodeficiency virus (HIV) infection are additional contributing factors.

Infectious agents

Bacteria: the bacteria that cause the most severe outbreaks are *Shigella dysenteriae* type 1 (see *Bacillary dysentery*) and *Vibrio cholerae* (see *Cholera*). *Salmonella* spp (see *Typhoid fever* for *S. typhi* and *paratyphi*) and a small number of strains of *Escherichia coli* may also cause disease.

Protozoa: such as *Entamoeba histolytica*, *Giardia lamblia* and *Cryptosporidium parvum*.

Viruses: In principle, viruses remain the major cause of acute diarrhoea in children (> 80%) and rotavirus predominates within this group. Norwalk virus is also important.

Case definition

Acute (watery) diarrhoea (in childhood)

Clinical case definition

Acute watery diarrhoea (passage of three or more loose or watery stools in the preceding 24 hours) with or without dehydration.

Laboratory criteria for diagnosis

Laboratory culture of stools may be used to confirm possible outbreaks of specific agents, but is not necessary for case definition.

Acute (bloody) diarrhoea

Clinical case definition

Acute diarrhoea with visible blood in the stool.

Laboratory criteria for diagnosis

Laboratory culture of stools may be used to confirm possible outbreaks of specific diarrhoea, such as *S. dysenteriae type 1*, but is not necessary for case definition.

Mode of transmission

All diarrhoeal diseases are transmitted via the faecal–oral route, particularly through contaminated water and food. Some infections result in a long carrier phase during which the patient may continue to excrete the organism despite being asymptomatic, leading to further spread of disease.

Incubation period

Salmonella generally requires an 8–48-hour incubation period, whereas the incubation period for *E. coli* 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 is 2–4 weeks for *E. histolytica*, 7–10 days for *G. lamblia* and 7 days for *C. parvum*. The incubation period for rotavirus is about 48 hours, it mainly affects children of under the age of 5 years and symptoms may last for up to 1 week. Incubation period of *Campylobacter* is usually 2–5 days (range, 1–10 days).

Period of communicability

During the acute stage of the disease and for the duration of faecal excretion. Temporary carrier status for *Salmonella* can persist for several months.

Reservoirs

Humans

Additionally, cattle (*C. parvum, Salmonella spp*, some *E. coli*), and domestic animals (*C. parvum, Salmonella spp*).

Epidemiology

Disease burden

Diarrhoeal diseases account for a high proportion of childhood and adult illness and death in Côte d'Ivoire. Compounded by a high rate of malnutrition and limited health care (including basic rehydration facilities), the burden of morbidity and mortality associated with diarrhoeal diseases is unduly high: 15% of all deaths of children under age 5 years in 2000–2003, 5% of deaths of neonates in 2000, and 16% of deaths of people of all ages in 2002 were due to diarrhoeal diseases in Côte d'Ivoire (*1*).

In Côte d'Ivoire, 81% of the population (urban, 98%; rural, 66%) has access to a supply of improved water (*2*), while 24% of the population has adequate sanitation facilities (urban, 38%; rural, 12%; 51% practise open defecation).

The increasing prevalence of HIV infection has led to a rise in the frequency of non-typhi *Salmonella* infection in Côte d'Ivoire and other parts of Africa; non-typhi *Salmonella* accounts for more than 60% of documented bacteraemias in some studies.

Geographical distribution

Diarrhoeal diseases occur throughout the country, but persons living in rural areas and in the western regions are particularly vulnerable.

Seasonality

Year-round exposure and risk.

Alert threshold

An increase in the number of cases reported that is greater than the expected number compared with the same period in previous years in a defined area.

Epidemics

Shigellosis is less common than typhoid and cholera in west Africa. A few cases of *EHEC* 015:H7 were reported in Côte d'Ivoire following the large outbreak in southern Africa in 1992, which involved humans and cattle. This illness is very similar to shigellosis and should be considered in the investigation of an outbreak of bloody diarrhoea.

Risk factors for increased burden

Population movement

Complex emergencies and natural disasters such as flooding resulting in large population movements and overcrowded refugee camps are classic settings for explosive outbreaks.

In camp settings, diarrhoeal diseases may account for between 25% and 40% of deaths in the acute phase of an emergency. More than 80% of deaths are among children aged less than 2 years.

Overcrowding

Overcrowding facilitates transmission.

Poor access to health services

Lack of health care and proper case management increases case fatality. Early detection and containment of cases is vital in reducing transmission. Lack of reporting mechanisms for outbreaks and poor surveillance and monitoring are further obstacles to effective prevention and control of disease.

Food shortages

Malnutrition increases the susceptibility to and severity of diarrhoeal disease. This in turn, contributes to further malnutrition.

Lack of safe water, poor hygienic practices and poor sanitation

Lack of safe water for drinking and food preparation, poor personal hygiene and food hygiene practices, and poor sanitation facilities are the most important risk factors for diarrhoeal epidemics.

Common sources of infection in emergency situations are:

- Contaminated water sources (e.g. by 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 or serving pots.

Prevention and control measures

Case management

Reduction of case fatality due to diarrhoeal disease is primarily related to effective management of dehydration, particularly in children.

- Prevention of dehydration – give recommended home fluid and oral rehydration salts (ORS).
- Treatment of dehydration – with ORS for mild to moderate dehydration, or with intravenous (IV) fluids (Ringer's lactate) for severe dehydration, is the mainstay of the management of diarrhoeal illness.

Use of antibiotics depends on the infectious agent involved (*3*).

Resume feeding with a normal diet when vomiting has stopped. It is important to separate those who are eating from those who are not. Food should be cooked on site. Continue breastfeeding infants and young children.

Zinc supplementation for children can decrease the severity and duration of illness: 20 mg of zinc per day for 14 days for children up to the age of 12 years; 10 mg per day for 14 days for infants under the age of 6 months.

Prevention

Prevention of diarrhoeal diseases depends on the provision and use of safe water, adequate sanitation and health education for proper hygiene and food safety.

Safe drinking-water:

- Provision of an adequate and safe supply, collection and storage system (*4*). The minimum emergency requirement is 15 litres/person per day;
- Provision of information on the importance of clean water and appropriate household storage of water.

Safe disposal of human excreta:

- Provision of adequate facilities for the disposal of human waste (*5*). The minimum emergency standard is one latrine for every 20 people.
- Provision of information on the importance of human-waste disposal, use of sanitation covers and correct maintenance of sanitation facilities.

Hand-washing with soap:

- Provision of soap in sufficient quantities for hand-washing, bathing and laundry needs.
- Health education on the relationship between disease spread and lack of or poor hand-washing before eating, after defecation, before food preparation and after cleaning/changing children's nappies (*6*).

Food safety:

- Provision of adequate food storage facilities (for both uncooked and cooked food), cooking utensils, adequate quantities of water and fuel to allow for cooking and re-heating.
- Health education on the importance of food safety and safe food-handling ("wash it, peel it, or leave it") (*7*).

Breastfeeding:

- Provision of information on the protective qualities of breastfeeding and the importance of breastfeeding ill children (*8*).
- Practical support for breastfeeding ill children.

Immunization

Rotavirus vaccine has been introduced in some developed countries (not in Côte d'Ivoire).

Epidemic control

Inform the health authorities immediately if there is an increase in the number of cases reported above the expected number is identified.

Confirm the outbreak, ensure proper case-management following WHO guidance given in Annexes 2 and 3.

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CD-WGE technical focal point: Department of Public Health and Environment (PHE)

DIPHTHERIA

Description

Clinical description

Diphtheria is an acute toxin-mediated disease caused by toxigenic strains of *Corynebacterium diphtheriae*. The disease affects the mucous membranes of the respiratory tract (respiratory diphtheria), the skin (cutaneous diphtheria), and occasionally mucous membranes at other sites (eyes, ears, or vagina). Cutaneous and nasal diphtheria are localized infections that are rarely associated with systemic toxicity.

Symptoms of respiratory diphtheria have a gradual onset and include mild fever (rarely > 38 °C), malaise, sore throat, difficulty in swallowing, loss of appetite and (with laryngeal involvement) hoarseness. Within 2–3 days, a firmly adherent, grey membrane forms over the mucous membrane of the tonsils, pharynx, or both. In severe cases, cervical lymphadenopathy and soft-tissue swelling in the neck give rise to a "bull-neck" appearance. Extensive membrane formation may result in life-threatening or fatal airway obstruction. Diphtheria toxin can cause serious, life-threatening systemic complications, including myocarditis and neuritis. In most cases, transmission of the infectious agent, *Corynebacterium diphtheriae,* to susceptible individuals results in transient pharyngeal carriage rather than disease. Respiratory diphtheria is a medical emergency with a case-fatality ratio (CFR) of 5–10%, even with treatment.

Infectious agent

Bacterium: *Corynebacterium diphtheriae*.

Case definition

Suspected case: Not applicable.

Probable case: Laryngitis **or** pharyngitis **or** tonsillitis **and** an adherent membrane of the tonsils, pharynx and/or nose.

Confirmed case: A probable case that is laboratory-confirmed or linked epidemiologically to a laboratory-confirmed case.

Carrier: presence of C. *diphtheriae* in the nasopharynx, without symptoms.

Note: People with cultures that are positive for C. *diphtheriae* and who do not meet the clinical description (i.e. asymptomatic carriers) should not be reported as probable or confirmed cases of diphtheria.

Laboratory confirmation

Isolation of a toxigenic strain of C. *diphtheriae* from a clinical specimen, or four-fold or greater rise in serum antibody levels (but only if both samples of serum were obtained before the administration of diphtheria toxoid or antitoxin).

Mode of transmission

Transmission is by contact (usually direct, rarely indirect) with the respiratory droplets of a case or carrier, discharges from skin lesions, or contaminated objects (uncommon). In rare cases, the disease may be transmitted through foodstuffs (raw milk has served as a vehicle).

Incubation period

Usually 2–5 days, occasionally longer.

Period of communicability

Until *C. diphtheriae* bacteria have disappeared from discharges and lesions; usually 2 weeks or less and seldom more than 4 weeks. The rare chronic carrier can shed *C. diphtheriae* for 6 months or more. The disease is usually not contagious 48 hours after treatment with antibiotics is instituted.

Reservoirs

Humans.

Epidemiology

Disease burden

Estimates of the disease burden in Africa are unreliable due to under-diagnosis and under-reporting. WHO in 2002 estimated that 2000 deaths were attributable to diphtheria in Africa. Carrier rates in Africa have been estimated to be as high as 9.3% in children in the general population.

No cases of diphtheria in Côte d'Ivoire were reported to WHO between 2001 and April 2009. Low vaccination coverage (less than 80%), increases the risk of outbreaks. Coverage of DTP3 (the third dose of diphtheria-tetanus-pertussis vaccine)

was 76% in 2007 (72% in 2000, dropping to 50–57% during 2001–2005, increasing again to 77% in 2006) (*1*).

Geographical distribution

Occurs throughout west Africa.

Seasonality

No data available. In temperate climates, the frequency of cases of diphtheria increases in the colder months. In warm climates, transmission occurs throughout the year. Seasonality in Côte d'Ivoire is not known.

Alert threshold

Any suspected or probable case must be investigated.

Epidemics

No recent outbreaks or epidemics have been reported from Côte d'Ivoire.

RISK FACTORS FOR INCREASED BURDEN

Population movement

Large movements of non-immunized populations into endemic regions carry a risk of outbreaks. Carriers of the bacterium may spread the disease to susceptible individuals.

Overcrowding

Overcrowding of susceptible groups (particularly infants and children) facilitates transmission and promotes outbreaks.

Poor access to health services

Poor access to immunization services will lead to low vaccination coverage (< 80%) for diseases that are included in the routine Expanded Programme on Immunization (EPI), thereby increasing the proportion of susceptible persons and the risk of outbreaks. Early detection and containment of cases reduce transmission.

Food shortages

Not relevant.

Lack of safe water, poor hygienic practices and poor sanitation

All are risk factors especially for toxigenic cutaneous diphtheria.

Prevention and control measures

Case management

Early diagnosis and proper case management with rapid investigation of contacts are essential.

Diphtheria antitoxin and antibiotics are the cornerstone of therapy for diphtheria.

The antitoxin neutralizes the diphtheria toxin only before its entry into cells. It is therefore critical that diphtheria antitoxin be administered as soon as a presumptive diagnosis has been made.

Antibiotic therapy, by killing the organism, has three benefits:

- Termination of toxin production;
- Improvement of local infection;
- Prevention of spread of the organism to uninfected people.

Do not wait for laboratory results before starting treatment/control activities.

Patients

Standard treatment includes diphtheria antitoxin and antibiotics.

- Diphtheria antitoxin

Diphtheria antitoxin (20 000–100 000 units) is given preferably intravenously (IV), or intramuscularly (IM), immediately after throat swabs have been taken and sensitivity-testing performed (the dose administered depends on the extent of disease and guidelines should be followed (*2*).

IV administration is preferred, particularly for delayed treatment, and for extensive or severe disease (see United Kingdom guidelines or the CDC Diptheria Anti Toxin (DAT) protocol).

Depending on the manufacturer, DAT comes as 1000 IU/ml, 2000 IU/ml, and 10 000 IU/ml preparations. Thus IM administration will usually require many injections; moreover, antiserum availability in the circulation is markedly delayed.

plus

- Antibiotics
  - Procaine penicillin IM (25 000–50 000 units/kg per day for children; 1.2 million units per day IM for adults in two divided doses) **or** parenteral erythromycin (40–50 mg/kg per day to a maximum of 2 g per day) until the patient can swallow; **then**
  - An age related dose of oral penicillin V (125–250 mg, four times per day) or oral erythromycin (40–50 mg/kg per day to a maximum of 2 g per day) in four divided doses.

Antibiotic treatment should be continued for a total of 14 days.

Isolation

Pharyngeal diphtheria – strict isolation is necessary.

Cutaneous diphtheria – strict isolation is not necessary. However, barrier precautions must be observed in order to prevent contact with cutaneous lesions.

Note: Clinical diphtheria does not necessarily confer natural immunity, and patients should therefore be vaccinated before discharge from a health facility.

Close contacts

Surveillance for 7 days for all people having close contact with a patient, regardless of vaccination status, and throat culture. Close contacts include household members and other individuals with a history of direct contact with a case, as well as health-care staff exposed to oral or respiratory secretions of a case.

All contacts should receive a dose of age-appropriate diphtheria toxoid-containing vaccine, unless a dose has been received within the previous 12 months.

All close contacts must receive a single dose of benzathine penicillin G IM (600 000 units for children aged less than 6 years; 1.2 million units for persons aged 6 years or more). Alternatively, a 7-day course of oral erythromycin can be given. If the nasal or throat culture is positive for *toxigenic C. diphtheriae,* give antibiotics as described under the "Patients" section above.

Carriers

A "carrier" is someone cultured in the context of a community survey, but not necessarily a close contact of an identified case which should be treated as per the "Close contacts" section above.

All carriers must receive:

- A single dose of benzathine penicillin G IM (600 000 units for children aged less than 6 years; 1.2 million units for persons aged 6 years or more). Alternatively, a 7-day course of oral erythromycin can be given. If the culture is positive for *C. C. diphtheriae*, give antibiotics as described under "Patients".
- An age-appropriate dose of diphtheria toxoid-containing vaccine, unless a dose has been received within the previous 12 months.

Prevention

Three measures:

- Ensuring high population immunity through vaccination (primary prevention);
- Rapid investigation and treatment of contacts (secondary prevention of spread);
- Early diagnosis and proper case management (tertiary prevention of complications and deaths).

Immunization

In non-epidemic situations, the approach to a previously unvaccinated population is to provide a full three-dose primary series (the first two doses are separated by 1–2 months and the third dose is given 6–12 months after the second) using:

- For children aged < 7 years: DTP (diphtheria-tetanus-pertussis vaccine);
- For children aged ≥ 7 years and adults: Td (combination of diphtheria and tetanus toxoid with reduced diphtheria content).

The current routine national immunization schedule in Cote d' Ivoire includes three doses of DTP-containing vaccines given at age 6 weeks, 10 weeks and 14 weeks. A subsequent booster dose is not part of the current national schedule.

For immunization in epidemic situations, see under "Epidemic control".

Epidemic control

- Inform the health authorities if one or more suspected cases are identified.
- Confirm the suspected outbreak, following WHO guidelines (see Annex 2).
- Investigate any probable case; check whether it fulfils the case definition, record date of onset, age and vaccination status.

- 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 C. *diphtheriae*.
- 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 proved not to be carriers.
- Implement outbreak-response measures; give priority to case management and immunization of population in areas not yet affected but to which the outbreak is likely to spread.
- **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 after 1–2 months to provide at least two doses of diphtheria toxoid-containing vaccine (preferably a combination of age appropriate (DT or Td) diphtheria and tetanus toxoids with reduced diphtheria content. If Td is unavailable, diphtheria-tetanus (DT) can be used for epidemic control). A third dose should be provided after 6–12 months for persons without a known history of previous primary immunization.

References

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Epidemiology and Prevention of Vaccine-Preventable Diseases - Diphtheria Chapter. (http://www.cdc.gov/vaccines/vpd-vac/default.htm; accessed August 2009).

CD-WGE technical focal point: Department of Immunization, Vaccines and Biologicals (IVB)

DRACUNCULIASIS (GUINEA-WORM DISEASE)

Description

Clinical description

Dracunculiasis (Guinea-worm disease) is a painful infection affecting subcutaneous and deeper tissues, which is contracted by drinking-water contaminated with Guinea-worm (a large roundworm) larvae. Approximately 1 year after the initial infection with larvae, the gravid female worm induces a painful blister as it attempts to emerge through the skin (more than 90% of blisters occur in the lower limbs). Emergence may take 1–3 weeks and the worm manifests as a thin whitish filament in the centre of the ulcer and can be up to 1 m long. Emergence is accompanied or preceded by burning and itching of the area around the blister and frequently, fever, nausea, vomiting, diarrhoea and generalized urticaria (allergic skin rash). Ulcers are frequently subject to secondary bacterial infection.

No immunity to infection develops, and people in endemic areas suffer from repeated infections year after year, each infection lasting for about 1 year. Mortality is low unless secondary bacterial infections occur, leading to stiff joints, arthritis, and even permanent debilitating contractures of the limbs. Morbidity is, however, significant and people in endemic villages are incapacitated during periods of peak agricultural activity, which may affect their productivity for several weeks to months.

Infectious agent

Nematode (roundworm): *Dracunculus medinensis*.

Case definition

Suspected (clinical) case: A person reporting the emergence of one or more Guinea worms during the previous 12 months.

Confirmed (active) case: A person exhibiting the emergence of one or more Guinea worms.

Mode of transmission

The female worm discharges larvae on contact with stagnant water after immersion of the infected limb by an infected person. The larvae are ingested by crustacean copepods *(Cyclops* or "water fleas", 1–2 mm long and barely visible in a glass held

up to the light) and become infective after about 2 weeks. Humans ingest the infected copepods from infested shallow ponds and step wells. The copepods are dissolved by the acidity of the stomach, the larvae are liberated, penetrate the gut wall, develop and migrate through subcutaneous tissue, and become adults. The adult gravid worm then emerges through a blister and releases thousands of larvae when the leg is immersed in water, thus completing the life cycle (*1*).

Incubation period

About 1 year.

Period of communicability

No direct person-to person spread.

Time periods relevant to communicability:

- 2–3 weeks: the period from rupture of vesicle until larvae has been completely evacuated from the uterus of the gravid worm;
- About 5 days: the period during which larvae are infective for the copepods in water;
- 12–14 days to about 3 weeks after ingestion by copepods: the period during which the larvae become infective for humans (at temperatures exceeding 25 °C).

Reservoirs

Humans; no known animal reservoirs.

Epidemiology

Disease burden

Now found mostly in tropical Africa, the disease is prevalent where people bathe or wade in water used for drinking. Globally, the aim is to eradicate the Guinea worm. So far, 180 countries and territories have been certified free of disease transmission.

Côte d'Ivoire is one of four countries where disease transmission was interrupted in 2007: there were five cases of dracunculiasis in 2006 and none in 2007 (*2,3*).

Geographical distribution

Prevalence varies dramatically between locales within endemic countries.

Seasonality

The disease is seasonal; patterns of occurrence depend on climatic factors, especially rainfall, as this influences the type of water used by the human population. Transmission generally occurs in the dry season, when water sources are limited (generally December to May); the majority of cases are reported during the same season when the worm emerges after its 12-month incubation period.

Risk factors for increased burden

Population movement

Infected persons can contaminate previously uninfected water sources, leading to geographical spread of disease (provided the climatic and host factors that enable completion of the disease transmission cycle are present).

Overcrowding

Overcrowding can lead more people to share the same body of water infected with Guinea-worm larvae, thus increasing the risk of transmission.

Poor access to health services

Health education is required to discourage infected patients from bathing or washing in water sources, particularly during the long period of worm discharge. Secondary bacterial infection and remnant worm due to incomplete removal is common without access to health services. Lack of access to public health programmes contributes to sustained disease transmission.

Food shortages

Not relevant.

Lack of safe water, poor hygienic practices and poor sanitation

Among the most important factors are insufficient water and poor hygiene practices that lead to people drink and wash themselves in the same body of water. Human-made water-catchment ponds such as shallow wells and ponds are the main sources of transmission; use of these water sources largely determines the epidemiology of the disease.

Prevention and control measures

Case management

No drugs are currently available to kill the adult worm. Slow progressive extraction of the worm using a matchstick to roll it out gently (a few centimetres per day) until it has been completely removed (may take 10–20 days). Never break the worm, and never pull it. Local wound care with antibiotics for secondary bacterial infection if present, and tetanus toxoid. Surgical extraction is NOT recommended.

Educate the infected patient not to immerse the limb with the emerging worm into local sources of drinking-water.

Drugs such as thiobendazole, albendazole, ivermectin and metronidazole have no effect on the worm.

Prevention

Provision of sources of safe drinking-water is the most effective intervention. Ideally, all drinking-water should be boiled, chlorinated or filtered. Improvised filters include a 0.15 mm nylon mesh, a fine-meshed cloth filter or straw filter; ceramic-candle filters can also be used. Health education targeting endemic communities should emphasize that patients with blisters/ulcers should not enter any sources of drinking-water. Control copepod populations in ponds, tanks, reservoirs and step wells with regular application of the insecticide temephos.

Immunization

None available.

Epidemic control

Surveillance and health education. Identify infected individuals and contaminated water sources.

References

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CD-WGE technical focal point: Department of Control of Neglected Tropical Diseases (NTD)

HEPATITIS E

Description

Clinical description

An acute illness typically including acute jaundice, dark urine, anorexia, malaise, extreme fatigue and right upper-quadrant tenderness. Hepatitis E virus (HEV) cannot be distinguished reliably from other forms of acute viral hepatitis (A, B, C) except by specific serological testing. In general, hepatitis E is a self-limiting illness followed by recovery. Fulminant forms of hepatitis may occasionally occur. Case-fatality ratios (CFRs) range from 0.5% to 4%. The fulminant form of the disease occurs more frequently in pregnancy, when it is associated with a CFR of 20% in the third trimester. In endemic areas, the highest rates of clinically evident disease occur in young to middle-aged adults. Lower rates of disease in younger age groups may be the result of anicteric (without jaundice) and/or subclinical infection.

Infectious agent

Single-stranded RNA virus .

Case definition

Suspected case: A case that is compatible with the clinical description.

Confirmed case: A suspected case that is laboratory -confirmed positive for IgM anti-HEV.

Mode of transmission

Hepatitis E is primarily transmitted by the faecal–oral route (like hepatitis A). Consumption of faecally contaminated drinking-water has given rise to epidemics, and the ingestion of raw or uncooked shellfish has been the source of sporadic cases in endemic areas. Person-to-person transmission is uncommon. There is no evidence for sexual transmission or for transmission by transfusion. The risk factors for HEV infection are related to poor sanitation in large areas of the world, and HEV shedding in faeces. There is a possibility of zoonotic spread of the virus, since several non-human primates, pigs, cows, sheep, goats and rodents are susceptible to infection.

Incubation period

Usually 2–5 days, occasionally longer.

Period of communicability

Unknown. HEV has been detected in stools 14 days after the onset of jaundice and approximately 4 weeks after ingestion of contaminated food or water.

Reservoirs

Humans are the natural hosts, as well as some non-human primates. Infection has been described in several other species (pigs, chicken and cattle).

Epidemiology

Disease burden

Epidemic and sporadic cases of hepatitis E suggest a global distribution of multiple strains of hepatitis E virus of varying pathogenicity. The global and national burden is unknown.

Geographical distribution

Outbreaks and sporadic cases occur over a wide geographical area. Epidemics have been reported in central and south-east Asia, north and west Africa, and in Mexico, especially where faecal contamination of drinking-water is common.

Seasonality

Perennial.

Alert threshold

In the absence of a clear epidemic threshold, an epidemic should be suspected if:

- Multiple cases of confirmed disease are seen in a single geographical area;
- Deaths of pregnant women are reported with fever/jaundice syndrome.

Epidemics

The highest rates of infection occur in regions where low standards of sanitation promote transmission. An outbreak occurred in Côte d'Ivoire in 1992.

Risk factors for increased burden

Population movement

Population movement increases the likelihood of contaminated water and poor hygiene.

Overcrowding

Overcrowding is a very important risk factor and facilitates transmission.

Poor access to health services

Poor access to health services may delay detection and response to outbreaks.

Food shortages

Malnutrition increases the susceptibility of the gastrointestinal tract to invasion by the organism and also the severity of disease.

Lack of safe water, poor hygienic practices and poor sanitation

Overcrowding, lack of safe water, poor hygiene and inadequate sanitation increase the risk of infection. The risk of epidemics of hepatitis E is high in camp settings.

Prevention and control measures

Case management

Supportive and symptomatic

Epidemic control

Prevention and detection are of key importance, given that there is no known therapy to alter the course of the disease and that it is spread by the faecal–oral route. Determine mode of transmission, investigate water supply, identify populations at increased risk, improve sanitary and hygienic practices to eliminate contamination of food and water, provide appropriate health education.

Prevention

Protect, purify and chlorinate public water supplies. Education to promote household water treatment, sanitary disposal of faeces, and hand-washing after defecation and before handling food. See *Diarrhoeal diseases (others):* Prevention; and Annex 4*: Safe water and sanitation*.

Immunization

Vaccines to prevent hepatitis E are being developed, but none are yet commercially available. Immunoglobulin prepared from plasma collected in HEV-endemic areas has not been effective in preventing clinical disease during HEV outbreaks.

Further reading

Key messages for health education: Hepatitis E. Geneva, World Health Organization (http://www.who.int/csr/disease/hepatitis/education_messages/en/index.html, accessed August 2009).

CD-WGE technical focal point: Department of Immunization, Vaccines and Biologicals (IVB)

HIV/AIDS

Description

Clinical description

Acquired immunodeficiency syndrome (AIDS) is the late clinical stage of infection with the human immunodeficiency virus (HIV) and is defined as an illness characterized by one or more indicator diseases.

Infectious agent

HIV. Two types have been identified: HIV-1 and HIV-2, which have similar epidemiological characteristics.

Case definition

WHO revised the staging system for HIV infection in 2006 (Table 5) (*1*).

Table 5. **Revised WHO clinical staging of HIV/AIDS for adults, adolescents and children**

Adults and adolescents[1]	Children
Clinical stage 1	
Asymptomatic Persistent generalized lymphadenopathy (PGL)	
Clinical stage 2	
Herpes zoster Angular cheilitis Papular pruritus eruptions Recurrent oral ulcerations Fungal nail infections (of fingers in adults)	
Moderate unexplained weight loss (< 10% of presumed or measured body weight) Recurrent respiratory-tract infections (sinusitis, bronchitis, otitis media, pharyngitis) Seborrheic dermatitis	Unexplained persistent hepatosplenomegaly Lineal gingival erythema Extensive wart-virus infection Extensive molluscum contagiosum Unexplained persistent parotid enlargement Recurrent/chronic upper respiratory-tract infections (otitis media, otorrhoea, sinusitis or tonsillitis)

Clinical stage 3	
Oral hairy leukoplakia	
Persistent oral candidiasis (in children: after first 6–8 weeks of life)	
Pulmonary tuberculosis (must be current in adults)	
Unexplained chronic diarrhoea (for > 1 month in adults; ≥ 14 days in children)	
Unexplained persistent fever (> 37.6 °C in adults or > 37.5 °C in children; intermittent or constant, for > 1 month)	
Unexplained anaemia (< 8 g/dL), neutropenia (< 0.5 ´ 10^9/l) or chronic thrombocytopenia (<50 ´ 10^9/l)	
Unexplained severe weight loss (> 10% of presumed or measured body weight)	Unexplained moderate malnutrition or wasting not adequately responding to standard therapy
Severe bacterial infections (such as pneumonia, empyema, pyomyositis, bone or joint infection, meningitis or bacteraemia)	Acute necrotizing ulcerative gingivitis or periodontitis
Acute necrotizing ulcerative stomatitis, gingivitis or periodontitis	Lymph-node tuberculosis
	Severe recurrent bacterial pneumonia
	Symptomatic lymphoid interstitial pneumonitis
	Chronic HIV-associated lung disease including brochiectasis
Clinical stage 4	
Pneumocystis pneumonia	
Extrapulmonary tuberculosis	
Disseminated non-tuberculous mycobacterial infection	
Chronic herpes simplex infection (orolabial, genital or anorectal of > 1 month duration or visceral at any site)	
Oesophageal candidiasis (or candidiasis of trachea, bronchi or lungs)	
Cytomegalovirus infection - retinitis or infection of other organs (in children: onset after 1 month of life)	
Chronic cryptosporidiosis (with diarrhoea)	
Chronic isosporiasis	
Symptomatic HIV-associated nephropathy or symptomatic HIV-associated cardiomyopathy	
Extrapulmonary cryptococcosis including meningitis	
Disseminated mycosis (coccidiomycosis or histoplasmosis)	
Central nervous system toxoplasmosis (after 1 month of life)	
Progressive multifocal leukoencephalopathy	
HIV encephalopathy	
Kaposi sarcoma	
Cerebral or B-cell non-Hodgkin lymphoma	
HIV wasting syndrome	Unexplained severe wasting, stunting or severe malnutrition not responding to standard therapy
Recurrent severe bacterial pneumonia	Recurrent severe bacterial infections (such as empyema, pyomyositis, bone or joint infection or meningitis but excluding pneumonia)
Recurrent non-typhoidal Salmonella bacteraemia	
Other solid HIV-associated tumours	
Invasive cervical carcinoma	
Atypical disseminated leishmaniasis	

Source: *WHO case definitions of HIV for surveillance and revised clinical staging and immunological classification of HIV-related disease in adults and children*. Geneva, World Health Organization, 2007 (http://www.who.int/hiv/pub/guidelines/hivstaging/en/index.html, accessed June 2009).

Laboratory confirmation of HIV

HIV infection is most commonly detected by testing for HIV antibody in serum samples using enzyme-linked immunosorbent assay (ELISA, or enzyme immunoassay, EIA). After infection, there is a period called the "window" during which the body starts to produce antibodies, but the levels are too low to be detected by current HIV tests. However, during this time the virus replicates actively and HIV infection can be transmitted. This window period lasts about 3 months.

When the ELISA or EIA test for HIV antibody gives a positive result, 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 of the first.

The rapid tests that are recommended by WHO have been evaluated at WHO collaborating centres and have levels of sensitivity and specificity that are comparable to those of WHO-recommended ELISA tests. The use of rapid HIV tests affords several advantages in emergency and disaster settings including:

- 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 where relatively few tests are performed.
- Many rapid tests require no laboratory equipment and can be performed in settings where electrical and water supplies need not be guaranteed.
- Some rapid tests can detect HIV antibodies in whole blood (finger-prick samples) as well as serum/plasma, and testing may therefore be performed by non-laboratory personnel with adequate training and supervision.

Mode of transmission

- Sexual intercourse (vaginal or anal) with an infected partner, especially in the presence of a concurrent ulcerative or non-ulcerative sexually transmitted infection (STI). The primary route of HIV infection is heterosexual.
- Infected mother to her child during pregnancy, labour and delivery or through breastfeeding (mother-to-child transmission).
- Transfusion of infected blood or blood products.
- 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), through accidental

injury of patients or service providers in occupational health, or those who are intravenous-drug users (IDUs).

Incubation period

Variable. On average, the time from HIV infection to clinical AIDS is 8–10 years, although AIDS may be manifested in less than 2 years or be delayed in onset beyond 10 years.

Incubation times are shortened in resource-poor settings, in infected infants and in older people.

Period of communicability

Any HIV-infected person may transmit the virus, beginning early after acquisition and throughout their lifetime, even when seemingly asymptomatic. Whilst the risk of transmitting infection is higher with a higher HIV viral load, a very low or an undetectable viral load does not equate to an absence of risk. The presence of concurrent sexually-transmitted illnesses (particularly ulcerative) in either partner increases transmission risk.

Reservoir

Humans.

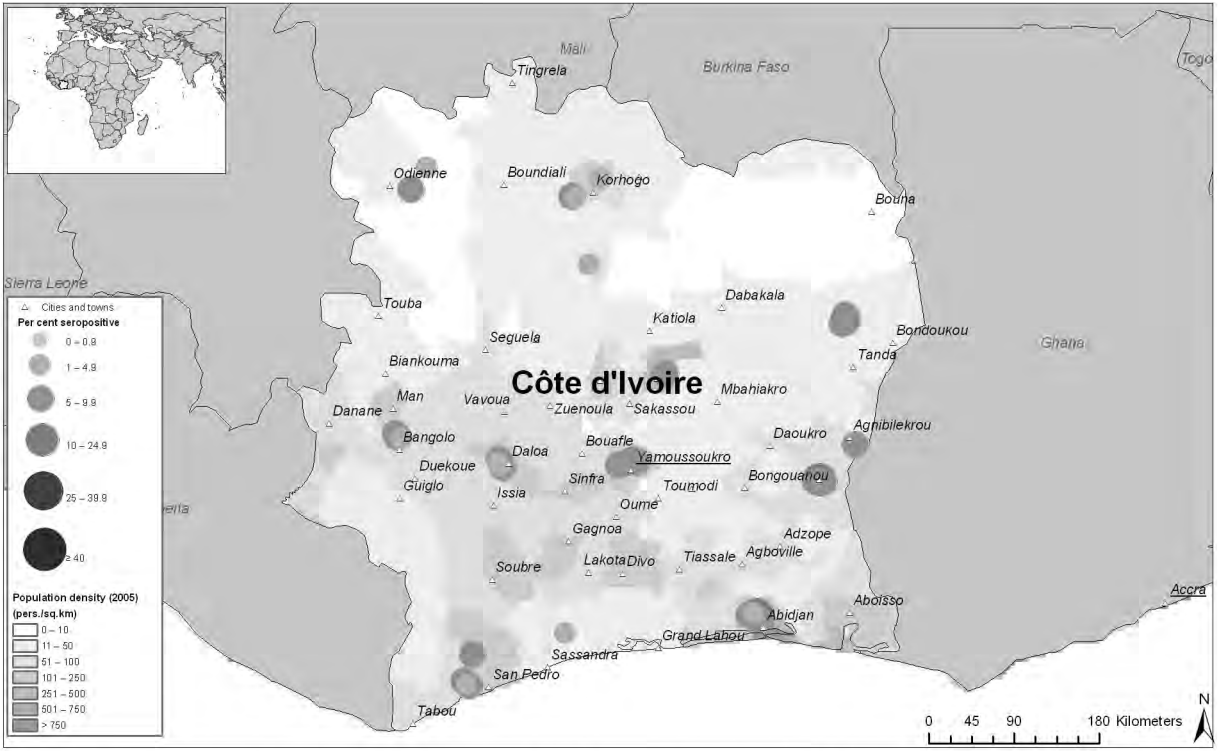
Epidemiology

Disease burden

Sub-Saharan Africa remains the worst-affected region in the world. With just over 10% of the world's population, it is home to 63% of all people living with HIV. At the end of 2006, 25.8 million people in sub-Saharan Africa were infected with HIV. In 2005, an estimated 3.2 million people in the region became newly infected, while 2.4 million adults and children died of AIDS (*2*). About 59% of all adults living with HIV in sub-Saharan Africa are women (13.2 million).

In 2007, UNAIDS estimated the HIV prevalence among adults aged 15–49 years to be 3.9%, decreasing from a peak of > 6% in 1998, with 424 260 persons living with HIV; 46 600 deaths attributable to AIDS and 420 943 children orphaned because of AIDS. The rate of seroprevalence is higher among women (6.4%) than men (2.9%) and this is the case for all for age groups.

Fig. 5 **HIV sentinel surveillance in pregnant women, Côte d'Ivoire, 2002–2006**



World Health Organization

The boundaries and names shown and the designations used on this map do not imply the expression of any opinion whatsoever on the part of the World Health Organization concerning the legal status of any country, territory, city or area or of its authorities, or concerning the delimitation of its frontiers or boundaries. Dotted lines on maps represent approximate border lines for which there may not yet be full agreement.

Data source: UNAIDS, WHO, CIESIN/FAO/CIAT, DCW, USCB
Map production: Public Health Information and Geographic Information Systems (GIS)
World Health Organization

Geographical distribution

HIV infection is reported throughout the country, but the prevalence is higher in urban areas (5.4%: 7.4% among women, 3.2% among men) than in rural areas (4.1%: 5.5% among women, 2.5% among men). It is lower in the north-west (1.7%), higher in the centre (5.8%) and in Abidjan (6.1%), and intermediate in the mid-west (3.7%) (see Fig. 5).

Risk factors for increased burden

Population movement

In emergency situations, exposure to distress, violence, lack of resources, and altered social networks may be associated with high-risk sexual behaviour and sexual violence. Lack of information and education, and shortages of basic commodities for preventing HIV, such as condoms, can also increase the risk of HIV transmission.

Poor access to health services

Lack of education and communication results in decreased opportunities for prevention among those not infected with HIV. Important materials for HIV prevention, such as condoms, are likely to be lacking in an emergency situation. Without adequate medical services, STIs, if left untreated in either partner, greatly increase the risk of acquiring HIV. Interruption of programmes preventing mother-to-child transmission.

Lack of testing and counselling services delays diagnosis. Failure to treat concomitant opportunistic infections or illnesses and interruption or delayed commencement of antiretrovirals risk increasing the burden of illness and death among those already infected.

Health-service quality may be compromised, with increased chances of transmission in the health-care setting owing to failure to observe universal precautions and to unsafe blood transfusion.

Food shortages

The need for food is paramount in emergency situations; exchange of sex for money and food can occur. Malnutrition has an adverse impact on the health of the people living with HIV/AIDS. People living with HIV/AIDS need a balanced diet with extra energy requirements compared with a person who is not infected:

- Energy requirements are likely to increase by 10% to maintain body weight and physical activity in asymptomatic HIV-infected adults, and growth in asymptomatic children.
- During symptomatic HIV infection and subsequently during AIDS, energy requirements increase by approximately 20–30% to maintain adult body weight.
- Energy intakes need to be increased by 50–100% above normal requirements in children experiencing weight loss.
- Energy intake should be increased by 20–50% during the convalescent period following opportunistic infection for children and adults.
- In food-secure as well as in non-food-secure areas, nutrition counselling promoting a diet of quality for HIV patients and carers has a key role to play in alleviating the side-effects of HIV (e.g. diarrhoea, nausea, sore mouth etc) as well the side-effects of antiretroviral treatment (ART) (e.g. insulin resistance, hyperlipidaemia etc).

Lack of safe water, poor hygienic practices and poor sanitation

People living with HIV and AIDS may suffer from disease and death as a consequence of limited access to food, clean water, and good hygiene, owing to their weakened immune systems.

Prevention and control measures

HIV prevention and care in an emergency response should include progressive implementation of the following:

- Safe disposal of medical waste (particularly needles and syringes) and universal precautions in the health-care setting;
- Condom distribution;
- Safe blood transfusion;
- Syndromic management of STIs;
- Clinical management of rape, including emergency contraception, and presumptive treatment of STIs, and later when antiretroviral drugs are available, post-exposure prophylaxis (PEP);
- Health education and behavioural-change interventions on safe sexual practices;
- Home-based care and support;
- Treatment of tuberculosis (TB) and other opportunistic infections;

- Voluntary counselling and testing (VCT);
- Prevention of mother-to-child transmission;
- Prophylaxis for opportunistic infections;
- ART;
- More comprehensive interventions to combat drug and alcohol problems;
- Monitoring and surveillance (including behavioural surveillance), in line with national strategies.

See Annex 5: *Injection safety.*

Case management

Voluntary counselling and testing

The establishment of voluntary counselling and testing (VCT) services to help individuals make informed decisions about HIV prevention and treatment should be considered when relative stability is restored. At present, the coverage of VCT is low in Côte d'Ivoire. In a 2007 survey of 2361 sex workers in Côte d'Ivoire, only half had had a HIV test in the last 12 months.

People must be fully informed and freely consent to testing and have counselling before and after testing. Confidentiality of the test result must be ensured.

At times, people can be coerced into testing or are required to make decisions about testing when they are suffering acute or post-traumatic stress disorders. As displaced persons 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 residency status is contingent on the applicant having HIV-antibody seronegative status.

Counselling before and after testing is essential for people with seronegative or seropositive results. Displaced persons and conflict survivors who are already traumatized may require additional psychosocial support if the test result is positive. Typically, the support networks of displaced persons are disrupted, and suicide-risk assessment therefore forms an important part of post-test counselling in a refugee or conflict context.

Testing of orphaned minors should be carried out with the consent of their official guardians, only where there is an immediate health concern or benefit to the child. There should be no mandatory screening before admittance to substitute care.

A positive test result is the gateway to treatment, and/or in the case of pregnancy, prevention of mother-to-child transmission (see later, under prevention).

Essential care

Where ART is not available or indicated, essential care includes:

- Psychosocial counselling and support;
- Disclosure, partner notification, and testing and counselling;
- Co-trimoxazole prophylaxis;
- Preventing fungal infections;
- Treating sexually transmitted and other reproductive-tract infections;
- Preventing malaria;
- Vaccinating against selected vaccine-preventable diseases (e.g. hepatitis B, pneumococcal, influenza vaccine);
- Nutritional care (see above under food shortages);
- Family planning;
- Needle-syringe programmes and opioid substitution therapy;
- Adequate water, sanitation and hygiene.

Treatment of opportunistic infections

Screen carefully for opportunistic infections and continue to monitor for their development. Common infections such as acute lower respiratory-tract infections, acute diarrhoeal illnesses, and malaria are common in HIV-infected persons. Non-typhi *Salmonella* species are the most common cause of bacteraemias in people living with HIV in many parts of Africa. TB is the most common co-infection in Côte d'Ivoire. All people living with HIV should be tested for TB, and all people presenting with TB should be offered VCT where available. Concurrent hepatitis B is an under-recognized co-infection.

Antiretroviral treatment (ART)

The repertoire of antiretrovirals available is currently limited in Côte d'Ivoire. Coverage of antiretrovirals in Côte d'Ivoire has increased from < 5% (2004) to about 30% (2007) (*2*,*3*).

ART treatment programmes in resource-poor settings have efficacy rates that are similar to those reported for developed countries. Country-specific national guidelines on use of ART that have been developed should be implemented. ART results

in improvement in clinical status and brings about effective reversal of the clinical stage in those with symptomatic disease.

When should ART be started?

The decision on when to start treatment with antiretrovirals is a complex one. The WHO classification of HIV-associated clinical disease has recently been revised in order to provide greater consistency between the adult and paediatric classification systems. Clinical staging is intended for use in emergency and crisis situations after HIV infection has been confirmed by HIV-antibody testing. It should form part of the baseline assessment (first visit) on entry into a care and treatment programme and is used to guide decisions on when to start co-trimoxazole prophylaxis and when to start and switch to ART in situations where a CD4-lymphocyte count is not available (see Table 6).

After a patient has tested positive for HIV, and meets the criteria for initiating ART (Table 6), the process of initiating ART involves assessing patient readiness to commence therapy and understanding its implications (lifelong therapy, adherence and toxicities). Support from family and peer support from individuals ("treatment buddy") groups is critical when ART is initiated, as adherence to treatment is difficult but essential to treatment efficacy and also to prevent the development of drug resistance.

Table 6. **Recommendations for initiating antiretroviral therapy in adults and adolescents, in accordance with clinical stages and the availability of immunological markers**

WHO clinical stage	Classification	CD4 testing	
		Not available	Available
			Start treatment if CD4 count is:
1	Asymptomatic	Do not treat	CD4 count, < 200 cells/mm^3
2	Mild	Do not treat	
3	Advanced	Treat	Consider treatment if CD4 count is < 350 cells/mm^3; and initiate ART before CD4 count drops below 200 cells/mm^3
4	Severe	Treat	Treat irrespective of CD4 cell count

Source: *Antiretroviral therapy for HIV infection in adults and adolescents: recommendations for a public health approach. 2006 Revision*. Geneva, World Health Organization, 2006 (http://www.who.int/hiv/pub/guidelines/artadultguidelines.pdf; accessed August 2009).

ART, antiretroviral treatment.

Current WHO ART recommendations require that the first-line regimen for adults and adolescents contains two nucleoside reverse-transcriptase inhibitors (NRTIs) plus one non-nucleoside reverse-transcriptase inhibitor (NNRTI) (see Further Reading: *Antiretroviral therapy for HIV infection in adults and adolescents in resource-limited settings: towards universal access recommendations for a public health approach*). These combinations are usually efficacious, generally less expensive, have generic formulations, are often available as fixed-dose combinations and do not require a cold chain. In addition, they preserve a potent new class of drugs (protease inhibitors) for second-line treatments. These recommendations should be used in conjunction with country-specific national guidelines for antiretroviral treatment.

There are various ART precautions that must be observed:

- Monotherapy or dual therapy should NOT be used to treat chronic HIV infection; they may be used only in the setting of prevention of mother-to-child transmission and post-exposure prophylaxis.
- A few specific nucleoside reverse transcriptase inhibitor (NRTI) combinations should be avoided, including concurrent stavudine (d4T) + AZT (proven antagonism), d4T + didanosine (overlapping toxicities) and lamivudine (3TC) + FTC (are very similar drugs).
- Avoid the combinations of TDF + 3TC + ABC and TDF + 3TC + didanosine as these select for the K65R mutation in HIV and are associated with high incidences of early virological failure.
- The combinations of TDF + didanosine + any non-nucleoside reverse transcriptase inhibitors (NNRTI) are also associated with high rates of early virological failure.
- The use of didanosine should be reserved for second-line treatment, in which situation it is possible to consider TDF + didanosine with boosted protease inhibitor (PI), provided that caution and close monitoring are practised.
- In the case of toxic side-effects of intolerance, and pregnant women on regimes containing efavirenz (EFV), the first-line regimen will need to be substituted with another first-line regimen.
- In the case of immunological and virological failure, a switch to second-line therapy should be made.
- After commencement of ART, monitor for the development of immune restoration disease where a previously known infection/condition paradoxically

worsens or an unrecognized infection/condition becomes starkly obvious. Immune restoration disease accounts for a large number of deaths in the first few months of ART.

When and how should ART be stopped?

If a person living with AIDS has exhausted all available options in terms of antiretroviral and opportunistic treatment and is clearly in a terminal condition because of advanced HIV infection or has distressing or intolerable side-effects of therapy, it is reasonable to stop giving antiretrovirals and institute an active palliative and end-of-life care plan.

In case of an emergency or disaster where drug shortages are likely, people on ART should be given strategies to help manage a treatment disruption. It is important that education and adherence counselling is instigated to prevent alterations in the drug regime, such as change in dosages, irregular treatment, or drug sharing.

If supplies of antiretrovirals are running out, the treatment should be stopped completely. People receiving antiretrovirals should be told not to conserve medications, to change dosing regimens, and to avoid acquiring antiretrovirals from unofficial sources, as the quality of the drugs acquired in this way cannot be guaranteed.

In case of disruption of a treatment regimen including nevirapine (NVP) or EFV, experts recommend that patients be provided with an additional 7-day supply of the two other drugs included in the treatment – NRTI drugs, e.g. 3TC and D4T, or 3TC and zidovudine (ZDV). This "wash-out" period of treatment with two NRTI drugs is designed to cover the time it takes for the NNRTIs to be eliminated from the system (as the half-life of NNRTIs in the bloodstream is longer than that of NRTIs). For other regimens, treatment should be stopped as soon as any drug in the regimen is not available and people on antiretrovirals should be counselled not to take the remaining doses of incomplete treatment.

Prevention

Universal precautions

Should be instituted in all stages of an emergency response.

Washing hands thoroughly with soap and water, especially after contact with body fluids or wounds.

Using protective gloves and clothing when there is a risk of contact with blood or other potentially infected body fluids.

Single-use needles and syringes should be employed. Safe handling and disposal of waste material, needles and other sharp instruments. Proper cleaning and disinfection of medical instruments between patients.

Blood safety

HIV testing of all transfused blood. Avoid non-essential blood transfusions. Recruitment of a pool of safe blood donors.

Reduce sexual transmission

Condom provision*:** good quality condoms should be made freely available to those already using condoms before the emergency. This can be done without health education in the immediate response to the emergency, accompanied by culturally appropriate ***condom promotion as the situation stabilizes. A household survey (EIS-CI: 4503 men and 5148 women) showed that less than one third used a condom during the last high-risk sexual contact (*4*).

STI management, including for sex workers, using the syndromic STI management approach, with partner notification and promotion of safer sex (*5*).

Clinical management of rape with a combination of emergency contraception (if presenting within 5 days), presumptive treatment of STIs, and post-exposure prophylaxis for HIV (PEP, if presenting within 72 hours), as well as appropriate counselling and follow-up care (*6*).

Education in awareness and life skills, especially for young people, ensuring that everyone is well informed of what does and does not constitute a mode of transmission; of how and where to acquire condoms free of charge and medical attention if necessary; and information on basic hygiene.

Reduce mother-to-child transmission of HIV

Coverage of antiretrovirals to HIV-positive pregnant women for the prevention of mother-to-child transmission (PMTCT) is about 12% (*7*).

Antenatal testing for HIV is poor with only 51% and 21% of women offered counselling in urban and rural settings, respectively. In the west, this is as low as 6%. Only 7% of the women who were counselled for HIV testing at an antenatal visit were tested and received the results of the test. In 2006, 11.2% of pregnant women Côte d'Ivoire infected with HIV received antiretrovirals; this increased to 17.2% in the first half of 2007 (*3*).

Most children living with HIV acquire the infection through mother-to-child transmission (MTCT), which can occur during pregnancy, labour and delivery or during breastfeeding (*8*). In the absence of any intervention, the risk of MTCT is 15–30% in non-breastfeeding populations. Breastfeeding by an infected mother increases the risk by 5–20% to 20–45%.

The risk of MTCT can be reduced to 2% or less by a combination of interventions:

- Antiretroviral prophylaxis given to HIV-positive women during pregnancy (preferably starting at 28 weeks) and labour and to the infant in the first weeks of life;
- Elective caesarean delivery (before the onset of labour and rupture of membranes); where elective caesarean delivery is not possible, avoidance of unnecessary obstetrically invasive procedures, such as artificial rupture of membranes or episiotomy;
- Complete avoidance of breastfeeding.

In many resource-constrained and emergency settings, elective delivery is seldom feasible and it is often neither acceptable nor safe for mothers to refrain from breastfeeding. In these settings, the efforts to prevent HIV infection in infants initially focused on reducing MTCT around the time of labour and delivery, which accounts for between one and two thirds of overall transmission. Many countries with a heavy burden of HIV have recently adopted more effective antiretroviral regimens beginning in the third trimester of pregnancy, which can reduce the risk of transmission during pregnancy and childbirth to 2–4%. Even when these regimens are used, however, infants remain at substantial risk of acquiring infection during breastfeeding (*9*). Where feasible, acceptable, affordable, sustainable and safe methods are available, infant-feeding practices should be modified. Otherwise, exclusive breastfeeding for the first months of life is recommended.

Avoiding unintended pregnancies and promoting access to family planning methods.

Where ART is available for the treatment of HIV/AIDS, ART should be continued in pregnant women who qualify (Table 6).

Post-exposure prophylaxis in health-care settings

There are strict eligibility criteria for the provision of antiretrovirals as post-exposure prophylaxis after a needle-stick injury (occupational and non-occupational) (*10*).

Prevention among injecting drug users

Ready access to sterile needles, syringes and other injection equipment (and disposal of used equipment).

HIV risk reduction education and counselling for injecting drug users (including peer outreach when possible).

Drug-dependency treatment services, including substitution treatment (e.g. methadone) where possible.

Access to STI and HIV/AIDS treatment for injecting drug users.

Physical protection

Protection of affected populations, especially women and children and the most vulnerable, from violence and abuse is not only an important principle of human rights but is also essential for reducing the risk of HIV infection.

Immunization

No vaccine against HIV is available currently, but work is in progress.

Asymptomatic HIV-infected children should be immunized with vaccines of the Expanded Programme on Immunization (EPI).

Symptomatic HIV-infected children should NOT receive either Bacille Calmette–Guérin (BCG) or yellow fever vaccine.

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INFLUENZA

In this chapter, influenza will be described under the following sections:

- Seasonal influenza
- Avian influenza, including highly pathogenic avian influenza A (H5N1) virus infection

Influenza, seasonal

Description

Infectious agent

Influenza viruses A, B and C. Influenza virus A has multiple subtypes, of which two (H1N1 and H3N2) are currently circulating widely among humans. Other influenza A subtypes of animal origin (16 HA and 9 NA subtypes), of which several (H5, H7 and H9) have caused sporadic human infections in some countries.

Case classification

Suspected case (clinical case definition): A person with rapid onset of fever of > 38 °C and cough or sore throat in the absence of other diagnoses. Diagnosis can be made on epidemiological characteristics: cases with similar clinical presentation usually cluster or form an epidemic typically with short intervals between case onset (1–4 days). The positive predictive value of this case definition is highest when influenza is circulating in the community (and is higher in adults or adolescents than in young children).

Confirmed case: A case that meets the clinical case definition and has been confirmed by laboratory test.

Laboratory criteria for diagnosis

Laboratory confirmation of influenza infection can be done by isolation of viruses from respiratory specimens (nasal, nasopharyngeal or throat swabs, gargle, tracheal aspirate etc) using cell culture or in embryonated eggs; direct identification of viral antigens in nasopharyngeal cells and fluids (fluorescent antibody [FA], test or enzyme-linked immunosorbent assay [ELISA]); rapid diagnostic test; or viral RNA amplification (reverse-transcriptase polymerase chain reaction, RT-PCR; see below). Demonstration of a fourfold or greater rise in specific antibody titre between acute and convalescent sera can also be used to confirm acute infection.

Ideally, respiratory specimens should be collected as early in the illness as possible. Virus shedding starts to wane by the third day of symptoms and in most cases virus is not detected after 5 days in adults, though virus shedding can occur for longer periods in children.

Antigen detection in respiratory specimens:

- *Rapid diagnostic tests (for A and B seasonal influenza).* Near-patient tests, or point-of-care rapid testing (enzyme immunoassays or neuraminidase assay) are commercially available. In general, the sensitivity of rapid tests is variable (median, 70–75%) and lower than that of virus culture, while their specificity is high (median, 90–95%). Because of low sensitivity, false negative results are a major concern with these tests. Results are available within 15–30 minutes.
- *RT-PCR.* RT-PCR assays detect viable and non-viable influenza virus RNA and are in general more sensitive than cell culture. Results can be available within a few hours.
- *Virus culture.* Considered as the "gold standard" and takes 2–10 days to complete. WHO provides standardized reagents to test the viruses. It is critical to provide information regarding circulating influenza subtypes and strains to formulate vaccine for the coming year, to make the reagents and to guide decisions regarding influenza treatment and chemoprophylaxis.

Antibody detection in serum specimens:

Rarely useful for immediate clinical management and used more commonly for retrospective diagnosis. Can be used for epidemiological purposes (detection of start of seasonal outbreak and studies). A fourfold rise in specific antibody titre of serum samples taken during the acute and convalescent phases suggests a recent infection (paired samples collected at least 2 weeks apart).

Influenza may be diagnosed clinically by typical symptoms during a recognized seasonal epidemic period when reliable surveillance data are available.

Mode of transmission

The transmission of human influenza viruses occurs through exposure to large particle (> 5 μm) respiratory droplets at distances closer than 1 metre and through direct, and possibly indirect, contact (e.g. fomites, with hand contamination and self-inoculation into nose or eye). Some evidence indicates that near-distance airborne transmission is possible. Relative contributions and clinical importance of the different modes of influenza transmission are currently unknown.

Incubation period

An infected person will develop symptoms in 1–7 days (usually 2 days).

Period of communicability

The patient may have detectable virus and possibly be infectious from 1–2 days before the onset of symptoms. Infectiousness can last for up to 7 days after the onset of illness in adults (perhaps longer if infection is caused by a novel virus subtype) and for up to 21 days after onset in children aged less than 12 years.

Reservoir

Humans normally form the primary reservoir for seasonal human influenza viruses.

Epidemiology

Disease burden

There is a lack of recent epidemiological and virological data on influenza in Côte d'Ivoire.

Geographical distribution

Influenza virus circulates globally.

Seasonality

In temperate countries, influenza epidemics peak during winter months. In some tropical countries, viral circulation occurs all year, with peaks during rainy seasons. High attack rates (47.4%) with a case-fatality ratio (CFR) of 1.5% were reported during seasonal influenza epidemics in the Democratic Republic of Congo and Madagascar (2002) (*1*). During the influenza outbreak in Madagascar (2002), despite rapid intervention within 3 months, more than 27 000 cases and 800 deaths were reported.

Alert threshold

An increase in the number of cases above what is expected for a certain period of the year or any increase in the incidence of cases of fever of unknown origin should be investigated, after eliminating other causes. Accumulated surveillance data are required to determine the threshold. Currently, no such data are available for Côte d'Ivoire.

Epidemics

No recent outbreaks or epidemics have been detected or reported from Côte d'Ivoire.

Risk factors for increased burden

Population movement

Influx of non-immune populations into areas where the virus is circulating or of infected individuals into areas with an immunologically naive population.

Overcrowding

Overcrowding with poor ventilation facilitates transmission and rapid spread.

Poor access to health services

Prompt identification, isolation and treatment of cases (especially treatment of secondary bacterial pneumonia with antimicrobials) are the most important control measures (see section on *Case management*). Without proper treatment, the CFR for complicated infection in people with chronic underlying conditions can be high. In countries where the burden of influenza disease is well documented, the most vulnerable populations are the elderly aged 65 years and older, those who are chronically immunocompromised, and infants and young children.

Food shortages

Low birth weight, malnutrition, vitamin A deficiency and poor breastfeeding practices are likely risk factors for any kind of infectious disease, and may prolong the duration of illness and give higher chances of complication.

Lack of safe water, poor hygienic practices and poor sanitation

Poor hand-washing either due to lack of access to safe water or to behaviour, lack of respiratory hygiene practices (e.g. cough etiquette) may facilitate spread.

Others

Lower temperatures and dry conditions contribute to longer virus survival in the environment. Low temperatures can also lead to crowded living conditions which can result in increased transmission (home confinement, increased proximity of individuals indoors, with insufficient ventilation of living spaces).

Smoking is a risk factor for complications and prolonged illness of acute respiratory illnesses (ARI) in general.

Immunocompromised individuals

Depending on the degree of immune compromise, viral replication could be protracted (weeks, and in rare cases, months), the frequency of complications is higher, and there is an increased probability that antiviral resistance will emerge during, and potentially enduring after, drug administration. Influenza infection itself can transiently depress immune cellular function, so that there is a potential risk for increased activity of HIV and possibly of reactivation of latent infections (such as tuberculosis).

Prevention and control measures

Case management

Early recognition, isolation of symptomatic patients and appropriate treatment of complicated cases are important.

For most people, influenza is a self-limiting illness that does not require specific treatment. Aspirin and other salicylate-containing medications should be avoided in children and adolescents aged less than 18 years in order to avoid the risk of a severe complication known as Reye syndrome. Paracetamol may be used for management of fever as clinically indicated. Antiviral drugs may be used for specific and early treatment.

M2 inhibitors (amantadine or rimantadine for influenza A only if the circulating virus is proven to be susceptible by local surveillance) and neuraminidase inhibitors (oseltamivir or zanamivir for influenza A and B) given within the first 48 hours can reduce symptoms and virus shredding. Neuraminidase inhibitors seem to have less frequent, less severe side-effects and are generally better tolerated than M2 inhibitors, reducing the frequency of complications that need antibiotic treatment and lead to hospitalization.

Antiviral resistance to treatment is more likely to develop with the use of M2 inhibitors, although oseltamivir-resistant A(H1N1) viruses have emerged and dominate in some parts of the world since the beginning of 2008). Where possible, neuraminidase inhibitors should be selected for treatment provided that they are registered for use in the country. If supplies are limited, antiviral treatment should be reserved for patients at high risk of complications (e.g. the elderly or those with underlying chronic conditions). WHO provides real-time antiviral drug resistance monitoring for influenza (*2*).

Patients should be monitored for the development of bacterial complications. Only then should antibiotics be administered accordingly. Other supportive therapies such as rehydration may be needed.

Isolation is impractical in most circumstances because of the highly transmissible nature of the virus and delay in diagnosis. However, ideally, all persons admitted to hospital with a respiratory illness, including suspected influenza, should be placed in single rooms or, if these are not available, placed in a room with patients with similar illness ("cohorting"). When cohorting is used, adequate spacing between beds should be provided for droplet precautions. For influenza, isolation should continue for the initial 5–7 days of illness, and possibly longer for patients who are severely immunocompromised and who may be infectious for longer periods. Both standard and droplet precautions are recommended (see Further reading: *Infection prevention and control of epidemic- and pandemic-prone acute respiratory diseases in health care. WHO interim guidelines.* Geneva, World Health Organization, 2007).

There is no need to adapt doses of the neuraminidase inhibitor, oseltamivir, for the elderly (Table 7). However, doses should be adapted for people with moderate renal failure (creatinine clearance, < 30 ml/minute). Oseltamivir should not be administered to any person who has experienced an allergic reaction to the drug in the past or to pregnant women, unless clinical circumstances indicate necessity (note the lack of safety data for this population). Nursing mothers are advised to refrain from breastfeeding during treatment.

Table 7. **Neuraminidase inhibitor: treatment schedule for oseltamivir**

Age	Dose[c]	Duration
Adults	75 mg twice per day	5 days
Children[a, b]		
≤ 15 kg	30 mg twice per day	5 days
> 15 kg to 23 kg	45 mg twice per day	5 days
> 23 kg to 40 kg	60 mg twice per day	5 days
> 40 kg	75 mg twice per day	5 days

[a] Use in children aged < 1 year has not been approved.

[b] Children aged 1 year or older require weight-adjusted doses.

[c] Doses should be reduced for individuals with decreased renal function.

Table 8. **M2 inhibitors: treatment schedules for amantadine and rimantadine**

Amantadine

Weight and/or age[a]	Dose	Duration
Age 1–9 years (≤ 45 kg)	5 mg/kg bw per day in two divided doses, up to a maximum of 150 mg/day	5 days
Age 10–65 years[b] (> 45 kg)	100 mg twice per day	5 days
Age > 65 years	100 mg once per day	5 days

Decreased renal function

Creatinine clearance (ml/minute per 1.73 m^2):	Dose
30–50	200 mg on first day and 100 mg per day thereafter
15–29	200 mg on first day and 100 mg on alternate days
< 15	200 mg every 7 days
Haemodialysis	200 mg every 7 days

[a] Amantadine should be used with caution in patients receiving treatment with neuro-psychiatric drugs and patients with seizure disorders, where the potential risks outweigh the benefits. This drug should not be used by women who are breastfeeding.

[b] In some jurisdictions, a once-daily dose of 100 mg is recommended e.g. the recommended regimen in the British National Formulary (BNF) and in Japan is 100 mg once-daily for patients aged > 10 years.

Rimantadine

Age (years)	Dose[b]	Duration
1–12[a]	5 mg/kg bw per day in two divided doses up to a maximum of 150 mg per day	5 days
13–64	100 mg twice per day	5 days
≥ 65	100 mg once per day	5 days

[a] Use in children less than 13 years of age has not been approved in some countries.

[b] Doses should be reduced for individuals with decreased renal function. Use with caution in patients with hepatic dysfunction.

Prevention

Non-pharmaceutical public health measures, including respiratory etiquette (covering coughs and sneezes) and hand hygiene, are the most feasible measures for the prevention of spread of influenza seasonal infection during epidemics.

Immunization

Vaccination with influenza vaccine is the primary measure to control seasonal influenza epidemics. The objective is to reduce disease morbidity and mortality for severe illness and death in at-risk groups (mainly the elderly, infant and young children and persons with chronic underlying conditions). This may be done through:

- Vaccination of at-risk individuals before the season (if burden of disease is known);
- Vaccination of caregivers (to prevent them from becoming the source of infection).

Immunization with available inactivated virus vaccines can provide 70–90% protection against illness in healthy young adults when the vaccine antigen closely matches the circulating strains of virus. A single dose suffices for those with prior exposure to influenza A and B viruses; two doses at least 4 weeks apart are essential for children aged less than 9 years who have not previously been vaccinated against influenza. Routine immunization programmes should focus efforts on vaccinating those at greatest risk of serious complications or death from influenza and those who might spread influenza (health-care personnel and household contacts of high-risk persons) to high-risk persons.

Proper health education and planning of yearly vaccination campaigns are recommended.

The recommended composition of seasonal influenza vaccines is reviewed twice per year by WHO.

Surveillance

Influenza is a disease that is under international surveillance. Countries are encouraged to report the disease activity and virus isolation to WHO through FluNet (*3*). Surveillance in Côte d'Ivoire is coordinated by the Department of Epidemic Viruses, Pasteur Institute, Abidjan.[2]

Surveillance of influenza is essential for:

- Characterization of the epidemic influenza pattern in terms of seasonality and identification of risk groups, to estimate burden of disease and impact, in

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order to allow for yearly planning of prevention (vaccination) and response activities (medical and non-medical interventions).

- Identification of changes in the epidemiological pattern over the year to allow timely implementation of planned medical and non-medical preparedness and response measures.
- Characterization of circulating strains of influenza virus to support updating of the composition of the annual seasonal influenza vaccine for the northern and southern hemispheres and allow early detection of new influenza A virus subtypes.
- Monitoring the emergence of viruses that are resistant to recommended anti-viral treatments.

Influenza, avian

Description

Avian influenza, or "bird flu", is a contagious disease of birds. Avian influenza viruses can be transported from farm to farm by the movement of live birds, carcasses, poultry equipment and products, people (contaminated shoes, clothing) and other items contaminated by infected birds or poultry products (vehicles, equipment, feed and cages). Several of the avian influenza viruses have been able to cross the species barrier to infect humans and lead to illness.

Avain influenza viruses are defined as "highly pathogenic" (HPAI) or "low pathogenicity" according to their pathogenicity in chickens. The "low pathogenicity" forms of avian influenza commonly cause mild symptoms in poultry and may easily go undetected. The highly pathogenic form (i.e. HPAI H5N1) spreads rapidly through poultry flocks, causing disease affecting multiple internal organs, and has a mortality that can approach 100%, often within 48 hours. Pathogenicity may differ between different poultry species. Ducks can be infected and contagious but often remain asymptomatic.

Only A(H5N1) and A(H7N7) have thus far been reported to cause human death; however, these and other avian influenza viruses are a cause of concern to human health not only because of their ability to cause morbidity and mortality in humans but because of the possibility that they could mutate into a form that spreads easily among humans, which could lead to an influenza pandemic. The first laboratory-confirmed human case of infection with HPAI A(H5N1) was reported in China, Hong Kong Special Administrative Region (SAR) in 1997 (18 cases including 6 deaths), during a period of outbreaks in poultry. Outbreaks occurred again in south-east Asia in 2003, followed by concurrent human infections at the end of 2003 in several countries (China, Thailand, Viet Nam). Since then, HPAI A(H5N1) has spread to wild birds and to poultry in Central Asia, Europe and Africa.

The first outbreak of HPAI A(H5N1) in animals in Africa was confirmed in poultry farms in northern Nigeria in early February 2006. The virus was also reported in poultry in Nigeria in 2007 and 2008. HPAI A(H5N1) was also confirmed in poultry and/or wild birds in Niger in February 2006, Cameroon in March 2006, and Sudan and Côte d'Ivoire in April 2006, Benin 2007 and 2008, Burkina Faso 2006, Djibouti 2006, Ghana 2007 and Togo 2007 and 2008). Egypt has been reporting H5N1 outbreaks in poultry since 2006, and the disease is now considered endemic in Egyptian poultry.

HPAI A(H5N1) can also infect several mammalian species besides humans, and the Food and Agriculture Organization of the United Nations (FAO) produces a regular information bulletin on the spread of HPAI A(H5N1) in animal populations.

Human infection with HPAI A(H5N1) virus

As of 2 March 2009, a total of 409 laboratory-confirmed cases (256 of which were fatal) have been reported since November 2003 from 15 countries including: Azerbaijan, Bangladesh, Cambodia, China, Djibouti, Egypt, Indonesia, Iraq, Lao People's Democratic Republic, Myanmar, Nigeria, Pakistan, Thailand, Turkey, Viet Nam (*4*).

As of March 2009, no cases of human infection with avian influenza virus had been recorded in Côte d'Ivoire.

Clinical description

A review article on human infection with avian influenza A(H5N1) was published by the WHO Writing Committee in 2008 (*5*). Guidelines on clinical management were updated with this additional experience in 2007 (*6*).

Common initial symptoms are fever (usually higher than 38 °C) and cough, plus signs and symptoms of lower respiratory-tract involvement including dyspnoea. Upper respiratory-tract symptoms such as sore throat and coryza are present only occasionally. Gastrointestinal symptoms were frequently reported in cases in Thailand and Viet Nam in 2004, but less frequently since 2005. Lower respiratory-tract manifestations often develop early in the course of illness and clinically apparent pneumonia with radiological changes is usually been found at presentation. The disease usually progresses rapidly and often progresses to an acute respiratory-distress syndrome. Median times of 4 days from the onset of illness to presentation at a health-care facility and 9–10 days until death in fatal cases have been reported. Atypical presentations have included fever and diarrhoea without pneumonia, and fever with diarrhoea and seizures progressing to coma.

Common laboratory findings include leukopenia, lymphopenia, mild-to-moderate thorombocytopenia, and elevated levels of aminotransferases. Lymphopenia and increased levels of lactate dehydrogenase at presentation have been associated with a poor prognosis. Of six infected pregnant women, four have died, and the two survivors had a spontaneous abortion. Mild illnesses such as upper respiratory illness without clinical or radiological signs of pneumonia have recently been reported more frequently in children. Limited seroepidemiology studies conducted since 2004 suggest that subclinical infection is uncommon. Overall case-fatality is high (63% as of July 2008).

Case definitions (August 2006)

A case of human influenza caused by a new subtype should be immediately notified to WHO under the International Health Regulations (IHR). For human infection with HPAI A(H5N1) virus, WHO has developed standardized case definitions to facilitate:

- Reporting and classification of human cases of H5N1 infection by national and international health authorities;
- Standardization of language for communication purposes;
- Comparability of data across time and geographical areas.

Application of the H5N1 case definitions:

- The current case definitions may change as new information about the disease or its epidemiology becomes available.
- National authorities should formally notify only probable and confirmed H5N1 cases to WHO. The case definitions for persons under investigation and suspected cases have been developed to help national authorities in classifying and tracking cases.
- The case definitions are not intended to provide complete descriptions of disease in patients but rather to standardize reporting of cases.
- In clinical situations requiring decisions concerning treatment, care or triage of persons who may have H5N1 infection, those decisions should be based on clinical judgment and epidemiological reasoning, and not on adherence to the case definitions. While most patients with H5N1 infection have presented with fever and lower respiratory complaints, the clinical spectrum is broad.

Person under investigation

A person whom public health authorities have decided to investigate for possible H5N1 infection.

Suspected H5N1 case

A person presenting with unexplained acute lower respiratory illness with fever (> 38 °C) and cough, shortness of breath or difficulty breathing **AND** one or more of the following exposures in the 7 days before symptom onset:

- Close contact (within 1 metre) with a person (e.g. caring for, speaking with, or touching) who is a suspected, probable, or confirmed H5N1 case;

- Exposure (e.g. handling, slaughtering, plucking, butchering, preparation for consumption) to poultry or wild birds or their remains or to environments contaminated by their faeces in an area where H5N1 infections in animals or humans have been suspected or confirmed in the last month;
- Consumption of raw or undercooked poultry products in an area where H5N1 infections in animals or humans have been suspected or confirmed in the last month;
- Close contact with a confirmed H5N1-infected animal other than poultry or wild birds (e.g. cat or pig);
- Handling samples (animal or human) suspected of containing H5N1 virus in a laboratory or other setting.

Probable H5N1 case (notify WHO)

Probable H5N1 case definition 1:

A person meeting the criteria for a suspected case **AND** one of the following additional criteria:

- Infiltrates or evidence of an acute pneumonia on chest radiograph plus evidence of respiratory failure (hypoxemia, severe tachypnea)

  OR
- Positive laboratory confirmation of an influenza A infection but insufficient laboratory evidence for H5N1 infection.

Probable H5N1 case definition 2:

A person dying of an unexplained acute respiratory illness who is considered to be epidemiologically linked by time, place, and exposure to a probable or confirmed H5N1 case.

Confirmed H5N1 case (notify WHO)

A person meeting the criteria for a suspected or probable case **AND** one of the following positive results conducted in a national, regional or international influenza laboratory whose H5N1 test results are accepted by WHO as confirmatory:

- Isolation of an H5N1 virus.
- Positive H5 PCR results from tests using two different PCR targets, e.g. primers specific for influenza A and H5 HA.

- A four-fold or greater rise in neutralization antibody titre for H5N1 based on testing of an acute serum specimen (collected 7 days or less after symptom onset) and a convalescent serum specimen. The convalescent neutralizing antibody titre must also be 1 : 80 or higher.
- A microneutralization antibody titre for H5N1 of 1 : 80 or greater in a single serum specimen collected at day 14 or later after symptom onset and a positive result using a different serological assay, for example, a horse red-blood-cell haemagglutination inhibition titre of 1 : 160 or greater or an H5-specific Western-blot positive result.

Application of H5N1 case definitions:

- The case definitions apply to the current phase of pandemic alert (phase 3) and may change as new information about the disease or its epidemiology becomes available.
- National authorities should formally notify only probable and confirmed H5N1 cases to WHO. The case definitions for persons under investigation and suspected cases have been developed to help national authorities in classifying and tracking cases.
- The case definitions are not intended to provide complete descriptions of disease in patients but rather to standardize reporting of cases.
- In clinical situations, decisions concerning treatment, care or triage of persons who may have H5N1 infection, should be based on clinical judgment and epidemiological evidence, and not on adherence to case definitions. While most patients with H5N1 infection have presented with fever and lower respiratory complaints, the clinical spectrum is broad.

For further details, see *WHO case definitions for human infections with influenza A(H5N1) virus (7).*

Mode of transmission

Most human infection is reported to be after exposure to infected birds. Infected birds also shed the virus in large quantities in their respiratory secretions and in their faeces. All parts of the animal and its blood may contain the virus. Human infection may occur through touching, slaughtering, plucking and butchering of infected birds and probably contact with contaminated environments. Unprotected manipulation or consumption of raw meat and eggs of infected birds should also be considered risk factors for possible infection, especially if there are poor hygiene

practices (hands, tools, environment, etc.). In some cases, infected birds (especially ducks) may not appear to be ill. Freezing does not kill the virus. Cooking thoroughly will kill the virus (*8*).

Human-to-human transmission was suspected in several clusters (cases related in time and place and documented as probable in Thailand in 2004, Indonesia in 2006, Pakistan and China in 2007). Human-to-human transmission, when suspected, is likely to have occurred in the context of intimate unprotected prolonged contact between a severely ill patient and the contact(s) to whom he/she transmitted the infection (for example, when taking care of the patient or sharing a bedroom with a patient).

Incubation period

After exposure to infected poultry, the incubation period generally appears to be 7 days or less, in many cases 2–5 days. In clusters in which limited, human-to-human transmission has probably occurred, the incubation period appears to be approximately 3–5 days.

Period of communicability

Limited data suggest that patients may remain infectious for as long as 3 weeks, perhaps even longer in immunosuppressed patients (i.e. those using corticosteroids). The longest documented period has been 27 days after the onset of illness, based upon detection of virus antigen in a patient's respiratory specimens.

Risk assessment

No predisposing factors for infection have been identified that can explain the low incidence of H5N1 observed in humans to date, despite extensive exposure. However, the risk for infection through inappropriate handling of ill birds remains. So far, no domestic mammals have been identified as a source of infection; however, cats and dogs can become infected.

Concern that additional human cases may occur in affected parts of Africa is high given the close contact between people and poultry (estimated 1.1 billion chickens in the African continent, mostly in backyard farming systems).

Throughout much of Africa, rapid detection and investigation of outbreaks is hampered by the absence of an early warning system for avian influenza in animals or humans, inadequate diagnostic capacity, and difficulties in shipping specimens, both locally and internationally, for diagnostic confirmation. Population movement and food insecurity increase the risk of importation from neighbouring countries to Côte d'Ivoire.

Prevention and control measures

Case management

The patient should be ***isolated*** and ***strict infection-control measures*** applied

- Standard and droplet precautions should be the minimum level of precautions to be used in all health-care facilities when providing care for patients with acute febrile respiratory illness, regardless of whether infection with avian influenza is suspected. The most critical elements of these precautions include facial protection (nose, mouth and eyes if sprays/splashes of secretions are anticipated) and hand hygiene.
- Therefore standard plus droplet precautions should be applied for routine care of patients with suspected or confirmed infection with avian influenza , which comprise of adequate hand hygiene, use of gowns, clean gloves, medical mask and eye protection if splashes are anticipated. If aerosol-generating procedures are performed, personal protective equipment (PPE) should include particulate respirator instead of medical mask.
- The WHO infection-control guidelines for health-care facilities are currently under revision *(9)*.

Treatment with antivirals should be given in case of suspected infection (clinical presentation and notion of exposure) in the absence of an alternative diagnosis.

- Oseltamivir is the primary recommended antiviral treatment. Observational data on treatment with oseltamivir in the early stages of the disease suggest that it is useful in reducing A(H5N1) virus infection-associated mortality. Furthermore, evidence that the A(H5N1) virus continues to replicate for a prolonged period indicates that treatment with oseltamivir is also warranted when the patient presents for clinical care at a later stage of illness.
- Modified regimens of oseltamivir treatment, including twofold higher doses (i.e. 150 mg twice per day for adults), longer duration and possibly combination therapy with amantadine or rimantadine (in countries where A(H5N1) viruses are likely to be susceptible to adamantanes) may be considered on a case-by-case basis, especially in patients with pneumonia or progressive disease. Ideally this should be done in the context of prospective data collection.

Additional supportive therapy

- Corticosteroids should not be used routinely, but may be considered for septic shock with suspected adrenal insufficiency requiring vasopressors (agent that

causes vasoconstriction and maintains or increases blood pressure e.g. norepinephrine, epinephrine or dopamine). Prolonged or high-dose corticosteroids can result in serious adverse events in A(H5N1) virus-infected patients, including opportunistic infection.

- Antibiotic chemoprophylaxis should not be used. However, when pneumonia is present, antibiotic treatment is appropriate initially for community-acquired pneumonia according to published evidence-based guidelines. When available, the results of microbiologic studies should be used to guide antibiotic usage for suspected bacterial co-infection in patients with A(H5N1) virus infection.
- Monitoring of oxygen saturation should be performed whenever possible at presentation and routinely during subsequent care (e.g. pulse oximetry, arterial blood gases), and supplemental oxygen should be provided to correct hypoxemia.
- Therapy for A(H5N1) virus-associated acute respiratory distress syndrome (ARDS) should be based upon published evidence based guidelines for sepsis-associated ARDS, specifically including lung-protective mechanical ventilation strategies (*6*).

Management of contacts

Chemoprophylaxis: Antiviral chemoprophylaxis should generally be considered according to the risk stratification.

High-risk exposure groups are currently defined as:

- Household or close family contacts of a strongly suspected or confirmed H5N1 patient, because of potential exposure to a common environmental or poultry source as well as exposure to the index case.

Moderate-risk exposure groups are currently defined as:

- Personnel involved in handling ill animals or decontaminating affected environments (including animal disposal) if PPE may not have been used properly.
- Individuals with unprotected and very close direct exposure to ill or dead animals infected with the H5N1 virus or to particular birds that have been directly implicated in human cases.
- Health-care personnel in close contact with strongly suspected or confirmed H5N1 patients, for example during intubation or performing tracheal suctioning, or delivering nebulized drugs, or handling inadequately screened/sealed body fluids without any or with insufficient PPE. This group also includes laboratory personnel who might have an unprotected exposure to virus-containing samples.

Low-risk exposure groups are currently defined as:

- Health-care workers not in close contact (distance greater than 1 metre) with a strongly suspected or confirmed H5N1 patient and having no direct contact with infectious material from that patient.
- Health-care workers who used appropriate PPE during exposure to H5N1 patients.
- Personnel involved in culling non-infected or likely non-infected animal populations as a control measure.
- Personnel involved in handling ill animals or decontaminating affected environments (including animal disposal), who used proper PPE.

To assist countries in prioritizing the use of antiviral drugs for chemoprophylaxis, particularly where their availability is limited, a three-tier risk categorization for exposure was developed.

Where neuraminidase inhibitors are available:

- In high-risk exposure groups, including pregnant women, oseltamivir should be administered as chemoprophylaxis, continuing for 7–10 days after the last exposure (strong recommendation); zanamivir could be used in the same way (strong recommendation) as an alternative.
- In moderate-risk exposure groups, including pregnant women, oseltamivir might be administered as chemoprophylaxis, continuing for 7–10 days after the last exposure (weak recommendation); zanamivir might be used in the same way (weak recommendation).
- In low-risk exposure groups oseltamivir or zanamivir should probably not be administered for chemoprophylaxis (weak recommendation). Pregnant women in the low-risk group should not receive oseltamivir or zanamivir for chemoprophylaxis (strong recommendation).
- Amantadine or rimantadine should not be administered as chemoprophylaxis (strong recommendation).

Where neuraminidase inhibitors are not available:

- In high- or moderate-risk exposure groups, amantadine or rimantadine might be administered for chemoprophylaxis if local surveillance data show that the virus is known or likely to be susceptible to these drugs (weak recommendation).

- In low-risk exposure groups, amantadine and rimantadine should not be administered for chemoprophylaxis (weak recommendation).
- In pregnant women, amantadine and rimantadine should not be administered for chemoprophylaxis (strong recommendation).

In the elderly, people with impaired renal function and individuals receiving neuropsychiatric medication or with neuropsychiatric or seizure disorders, amantadine should not be administered for chemoprophylaxis (strong recommendation).

Health monitoring is recommended for close contacts of cases up to 7 days after the last exposure and consists of monitoring temperature and symptoms such as cough. It is also required for health-care professionals who have had contact with patients, their body fluids and secretions, their room or with potentially contaminated equipment.

Quarantine of close contacts of suspected cases during the health-monitoring period is not necessary unless there is suspicion of human-to-human transmission.

Prevention

Reduce human exposure to H5N1

For individuals, the risk of bird-to-human transmission of avian influenzas can be reduced through proper precautions; hand hygiene, hygiene precautions when handling birds (especially when ill or dead) or their products for consumption or when in environments which may be contaminated with faeces of ill birds. In communities, the risk can be reduced by control of spread of the infection in the animal population, and reduction of human contact with infected birds.

Human-to-human transmission of the H5N1 can be prevented through early detection and isolation of suspected and confirmed cases in a dedicated health-care facility and application of infection-control measures.

Food safety precautions have been described by WHO (*10, 11*).

Humanitarian agencies could:

- Contribute to reducing human exposure to avian influenza A(H5N1) by informing communities affected by avian influenza in birds of risks of exposure to ill or dead animals (particularly poultry/birds) and of strategies for risk avoidance including avoiding close contact with ill/dead animals and their remains, or to environments contaminated by their faeces, avoiding consumption of raw or undercooked poultry products, and performance of hand hygiene after handling, slaughtering, plucking, butchering, or preparing poultry/wild birds;

- Ensure that the information they deliver is done in close coordination with the animal and public health authorities to prevent discrepancies in preventive messages.
- Promote immediate reporting to relevant local and national animal health authorities of unexpected illness/deaths in birds/animals.
- Investigate people developing unexplained acute respiratory illness after exposure to ill/dead birds should be investigated for H5N1 infection.

Agencies could support such efforts through integration of these activities into other field programmes such as agriculture, livelihoods, food security, water and sanitation.

Surveillance

Strengthen the early warning system.

- Humanitarian agencies should facilitate the early detection, notification and early response to initial suspected cases and/or clusters in humans of H5N1 avian influenza or a novel influenza virus.
- It is important that relevant authorities are notified immediately in case of any suspect die-off or severe unexplained illness in animals, especially if affecting birds.

Relevant authorities and WHO should also be informed immediately upon suspicion of human infection with an avian influenza virus so as to ensure rapid and adequate case management, and relevant further planning and action as necessary.

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CD-WGE technical focal point: Global Influenza Programme (GIP)

LEPROSY

Description

Clinical description

Leprosy is a chronic bacterial disease primarily affecting the skin, peripheral nerves, and the surface membranes of the nose and mouth. The clinical presentation of leprosy may vary in a continuous spectrum between two main forms, known as tuberculoid leprosy (paucibacillary) and lepromatous leprosy (multibacillary). In tuberculoid leprosy, the skin lesions are few and small, sharply demarcated, anaesthetic or hypoanaesthetic. Involvement of peripheral nerves tends to be bilateral, asymmetrical and severe. In lepromatous leprosy, the more severe form of the disease, the lesions may be much more diffuse, widespread and many with symmetrical and bilateral nodules and papules. Ocular involvement may result in iritis and keratitis leading to blindness.

Infectious agent

Bacterium: *Mycobacterium leprae*

Case definition

A case of leprosy is defined as a person showing hypopigmented or reddish skin lesion(s) with definite loss of sensation.

The WHO operational case definition includes:

- Persons who have failed to adhere to the treatment regimen with signs of active disease;
- Relapsed cases, patients having previously completed a full course of treatment.

Case classification

Suspected case (clinical):

Paucibacillary leprosy: one to five patches or lesions on the skin.

Multibacillary leprosy: more than five patches or lesions on the skin.

Confirmed case:

Laboratory criteria for confirmation

In practice, laboratories are not essential for the diagnosis of leprosy.

Mode of transmission

Not clearly established: organisms are probably transmitted from the nasal mucosa of an untreated infected person to another person through the mucous membranes of the upper respiratory tract and possibly through broken skin, during frequent and close contact.

Incubation period

Ranges from 9 months to 40 years; 5–7 years on average.

Period of communicability

If not treated: possible infectivity; the risk is higher for contacts of multibacillary cases than for contacts of paucibacillary cases.

Treated: infectivity is lost within a day of treatment with multidrug therapy (MDT) in most cases.

Reservoirs

Humans. Naturally acquired leprosy has been found in a mangebey monkey in Nigeria, in a chimpanzee in Sierra Leone and in feral amardillos in the United States of America (USA).

Epidemiology

Disease burden

During the 1980s, most African countries were highly endemic for leprosy, with an average national prevalence exceeding 2% (leprosy is considered to be a public health problem when the prevalence surpasses 1 per 10 000 population). The WHO African Region made substantial progress towards the elimination of leprosy during the 1990s. These efforts are ongoing in order for leprosy to be eliminated in each country.

Globally, annual new case detection continues to show a sharp decline. Most countries where leprosy has been endemic have managed to successfully eliminate leprosy to the target rate of < 1 case per 100 000 population. There were 254 525 new cases of leprosy reported worldwide in 2007, with 31 037 of these from Africa, giving a regional prevalence of 0.47 per 100 000 population. Côte d'Ivoire is one of 17 countries that reported more than 1000 new cases of leprosy in 2007; together these 17, largely African and Asian countries, account for > 95% of cases globally.

Leprosy is endemic in Côte d'Ivoire. However, like many endemic countries, detection of new cases continues, although the numbers may be low, emphasizing the importance of sustaining efforts towards elimination.

Geographical distribution

Specific data on geographical distribution within Côte d'Ivoire are not available.

Seasonality

No seasonality has been demonstrated.

Epidemics

No epidemic potential.

Risk factors for increased burden

Population movement

Movement of untreated individuals into areas with susceptible individuals or less well-established leprosy elimination programmes may increase the risk of disease spread.

Overcrowding

Increases the risk of transmission.

Poor access to health services

Lack of early diagnosis and treatment caused by difficulties (geographical, financial, security) in accessing health services and diagnostic methodology increases the disease load and the risk of transmission. Inadequate health education provided by the health services to dispel myths related to leprosy is still a significant contributory factor, as the enduring stigma associated with the disease further delays the presentation of the patient to health-care facilities.

Food shortages

Not relevant.

Lack of safe water, poor hygienic practices and poor sanitation

Poor hygienic practices may lead to secondary bacterial infections of crusty lesions or ocular lesions.

Prevention and control measures

Case management

Treat with MDT according to case classification. This treatment is highly effective, has few side-effects and there is virtually no recurrence. Free and convenient monthly calendar blister-packs are available. Patients are no longer infectious after the first dose. Compliance is important.

Multibacillary leprosy: 12 months of MDT with the following combinations:

Adults: – rifampicin: 600 mg once per month
– dapsone: 100 mg once per day
– clofazimine: 50 mg once per day and 300 mg once per month.

Children must receive appropriately scaled-down doses (in child blister-packs).

Paucibacillary leprosy: 6 months of multidrug treatment with the following combination:

Adults: – rifampicin: 600 mg once per month
– dapsone: 100 mg once per day

Children must receive appropriately scaled-down doses (in child blister-packs).

A core element of the elimination strategy is to make diagnosis and MDT available at all health centres, to all existing leprosy patients. MDT is provided free of charge by WHO. Any interruption of treatment schedules has serious implications for treatment outcome.

Prevention

The following actions are essential for prevention of leprosy and maintenance of an ongoing elimination campaign:

- Ensuring that accessible and uninterrupted MDT services are available to all patients through flexible and patient-friendly drug-delivery systems;
- Guaranteeing that MDT services are sustainable by integrating leprosy services into the general health services and building the ability of general health workers to treat leprosy;
- Encouraging self-reporting and early treatment by promoting community awareness and changing the image of leprosy;

- Monitoring the performance of MDT services, quality of patient care and progress made towards elimination through national disease-surveillance systems.

The enduring stigma associated with the disease remains an obstacle to self-reporting and early treatment. The image of leprosy has to be changed at the global, national and local levels. Reducing contact with known leprosy patients is of dubious value and can lead to stigmatization. It is important to create an environment in which patients will not hesitate to come forward for diagnosis and treatment at any health facility.

Immunization

Bacille Calmette–Guérin (BCG) vaccination can induce protection against the disease; this is part of the tuberculosis programme in Côte d'Ivoire and it is therefore not necessary to undertake BCG vaccination specifically for leprosy.

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CD-WGE technical focal point: Department of Control of Neglected Tropical Diseases (NTD)

LYMPHATIC FILARIASIS

Description

Clinical description

In its most obvious manifestations, lymphatic filariasis results in genital damage, especially hydrocoele and elephantiasis of the penis and scrotum among 10–50% of men in communities where the disease is endemic. Elephantiasis of the entire leg, the entire arm, the vulva, or the breast – swelling of up to several times normal size – can affect up to 10% of men and women in these communities. These symptoms generally appear in adults and in men more often than in women.

The disease may take years to become manifest. In fact, many people never acquire outward clinical manifestations of their infections. Although there may be no clinical symptoms, studies have now disclosed that such victims, outwardly healthy, actually have hidden lymphatic pathology and kidney damage. The asymptomatic form of infection is most often characterized by the presence in the blood of thousands or millions of larval parasites (microfilariae) and adult worms located in the lymphatic system.

Acute episodes of local inflammation involving the skin, lymph nodes and lymphatic vessels often accompany chronic lymphoedema or elephantiasis. Some of these are caused by the body's immune response to the parasite, but most are the result of bacterial infection of the skin where normal defences have been partially lost due to underlying lymphatic damage.

Infectious agent

Nematode *Wuchereria bancrofti*. *Brugia malayi* and *Brugia timori* also cause lymphatic filariasis but are not usually found in Africa.

Case classification

Suspected case: Hydrocele or lymphoedema in a resident of an endemic area for which other causes of these findings have been excluded.

Confirmed case: A person with positive laboratory criteria even if he/she does not meet the clinical case definition.

Laboratory criteria

Positive parasite identification by:

- Direct blood examination; or
- Ultrasound; or
- Positive antigen detection test using monoclonal antibody specific for *W. bancrofti*.

Mode of transmission

Repeated bites of infected blood-feeding female mosquitoes (mainly *Anopheles*, also *Culex* species), which transmit immature larval forms of the parasitic worms from human to human.

Incubation period

Microfilariae may not appear in the blood until 6–12 months after infection (known as the pre-patent period).

Period of communicability

Not directly transmitted from human to human. Humans may infect mosquitoes if microfilariae are present in the peripheral blood (from 6–12 months to 5–10 years after the infective bite). Mosquitoes become infective 12–14 days after an infected blood meal. A large number of infected mosquito bites are required to initiate infection in the host.

Reservoirs

Humans. No other reservoirs for *Wuchereria bancrofti*.

Epidemiology

Disease burden

The burden of lymphatic filariasis, as measured in disability-adjusted life years (DALYs), is the highest of all tropical diseases after malaria. Countries are categorized as endemic when prevalence is > 1% microfilaraemia or antigenaemia. As of 31 December 2007, the total population at risk for lymphatic filariasis is estimated to be 1.3 billion people in 81 endemic countries and territories. At least 38% of the global burden of lymphatic filariasis is found in Africa, with 382 million people at risk and 43 million already infected; 4.6 million have lymphoedema and additional 10 million have hydrocele. Côte d'Ivoire is one of the 39 countries in the WHO African Region where lymphatic filariasis is endemic. Elimination programmes have begun in 10 of the endemic countries, and plans to commence such a pro-

gramme in Cote d'Ivoire were advanced in 2008. Mapping of the disease was completed in 2004. 5023 persons were examined, giving an average prevalence of 8.4% (range, 1.4–45.5%).

Geographical distribution

Of the 67 districts surveyed in Cote d'Ivoire, lymphatic filariasis is endemic in 33, and the status of 34 districts is still unknown. Data on reported cases is not

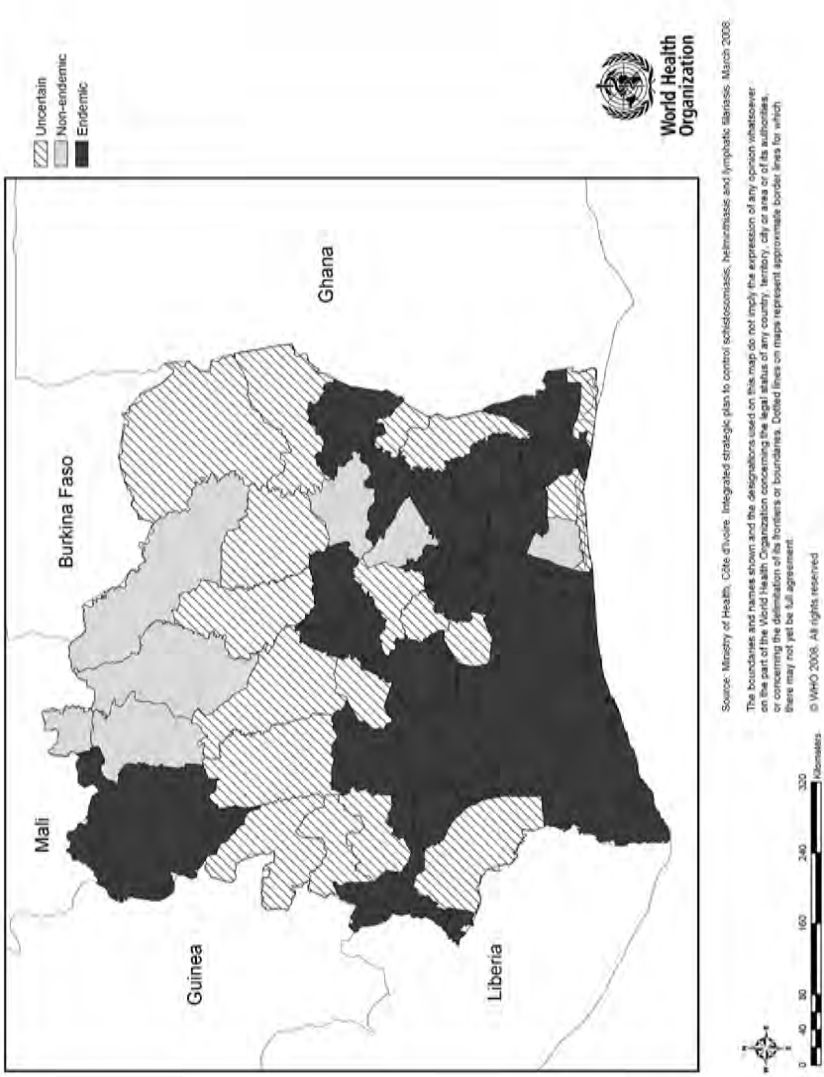


Fig. 6 **Endemicity of lymphatic filariasis by department, Côte d'Ivoire, 2002**

available but, generally, the entire population in the southern part of the country is at risk, a small proportion in the north has been identified as being at risk, while mapping in the central part of the country has yet to be conducted (Fig. 6).

A study based in west Africa (excluding Côte d'Ivoire) has suggested there is a negative spatial association between the prevalence of *Wuchereria bancrofti* and that of *Plasmodium falciparum (1)*. Interspecies competition between parasites, seasonality, differences in the distribution and vector competence of *Anopheles* vectors, agricultural practices and insecticide resistance may be factors driving current (and potentially future) spatial distributions. This may have serious implications for elimination programmes.

Seasonality

The rainy season is likely to be associated with a higher risk of transmission. The frequency of acute adenolymphangitis (ADL) attacks also increases during the wet season.

Epidemics

The disease is not prone to outbreaks.

Risk factors for increased burden

Population movement

Movement of disease-free individuals into endemic areas put them at risk. Movement of chronically infected individuals into disease-free areas increases the risk of geographical spread.

Migration of infected-mosquitoes into disease-free areas may also lead to disease spread.

Overcrowding

Living in crowded conditions increases the risk of transmission.

Poor access to health services

Lack of early diagnosis and treatment owing to difficulties in accessing health services (for geographical, financial or security reasons) and diagnostic methodology increases the risk of transmission and morbidity. Shame and stigma associated with the disease often further delays presentation to health-care services.

Food shortages

Not directly, but malnutrition leading to hypoalbuminaemia may compound limb oedema and delay wound healing.

Lack of safe water, poor hygienic practices and poor sanitation

Providing safe water is a means of secondary prevention of complications such as adenolymphangitis attacks and secondary bacterial infections. It is essential for some of the hygienic measures recommended for the affected body parts. Poor sanitation may contribute to creating breeding sites for mosquito vectors (especially *Culex* spp.).

Other

Lymphatic filariasis is closely associated with the economies and infrastructure of endemic communities. There is an established link between reduced productivity, poverty and the prevalence of lymphatic filariasis.

Prevention and control measures

Case management

Identification of microfilariae in a blood smear by microscopic examination confirms the diagnosis of active infection. Blood collection should be done at night, when microfilariae circulate in peripheral blood. However, as lymphoedema develops many years after infection, the results of laboratory tests are most likely to be negative for these patients. A rapid immunology-based card test has been developed.

Drug regimen for microfilaria-positive patients:

- Diethylcarbamazine citrate (DEC) as a single dose at 6 mg/kg for 12 days, repeated at 1–6 month intervals if necessary. A single dose at 6 mg/kg is equally effective for killing the adult worm and in reducing the number of microfilaria. *However, for bancroftian filariasis patients who live in areas endemic for onchocerciasis or loasis, as in Cote d'Ivoire, it is important to exclude co-infection with these agents before treatment with DEC as it may worsen eye disease in the former and cause serious adverse reactions in the latter.*
- Alternatively, ivermectin and albendazole can be used. *However, those suffering from Loa loa co-infection should not be exposed to ivermectin as it promotes massive destruction of microfilariae, which may cause an allergic reaction and even encephalopathy).*

- Alternative regimes (used outside national elimination programmes) include a single dose of DEC plus albendazole or multiweek doxycycline.

Knowledge of the geographical distribution of lymphatic filariasis and Loa loa is useful for decision-making on appropriate treatment. WHO guidelines on preventive chemotherapy in human helminthiasis should be consulted (*1*).

Lymphoedema and elephantiasis are not indications for DEC treatment. Treatment for large hydroceles is mostly surgical.

Hygiene measures for the affected body parts (and, when necessary, antibiotics and antifungal agents) can reduce the risk of ADL:

- Washing the affected parts twice per day with soap and clean water, and keeping the affected part dry;
- Raising the affected limb(s) at night;
- Exercising to promote lymph flow;
- Keeping nails short and clean;
- Wearing comfortable footwear;
- Using antiseptic, antibiotic, or antifungal creams to treat small wounds or abrasions (in severe cases, systemic antibiotics may be necessary).

Prevention

Transmission of infection can be interrupted by

- Minimizing contact between humans and vectors (through vector control and avoiding mosquito bites;
- Reducing the amount of infectious agent the vector can acquire (by treating the human host).

The Global Programme to Eliminate Lymphatic Filariasis, launched in 2000, has two main goals: to interrupt transmission of infection and to prevent disability caused by the disease. This includes efforts to map affected communities, mass drug administration (MDA) using annual two-drug combinations (ivermectin and albendazole in Africa) for elimination, for at least 5 years. Côte d'Ivoire has undergone mapping and is ready to start MDA in 2009. Only Liberia among its neighbouring countries has not yet commenced MDA (*2*). In MDA, since the entire at-risk population must be treated for a period long enough to ensure that

levels of microfilariae in the blood remain lower than necessary to sustain transmission, a yearly, single-dose MDA of the following drugs is recommended:

In areas with concurrent onchocerciasis (as in Côte d'Ivoire):

- 400 mg of albendazole + 150 µg/kg of ivermectin, once per year for at least 5 years, or until the prevalence of microfilaraemia is brought down to less than 1% in most areas and less than 1 per 1000 school-entry children showing infection, whichever occurs later. Note that onchocerciasis has recently re-emerged in the south-western regions of Côte d'Ivoire.

In areas with concurrent loasis:

- MDA with ivermectin cannot be applied owing to possible serious adverse effects.

Immunization

None available.

Epidemic control

Because of relatively low infectivity and long incubation, epidemics of lymphatic filariasis are unlikely.

References

1. *Preventive chemotherapy in human helminthiasis. Coordinated use of anthelminthic drugs in control interventions: a manual for health professionals and programme managers.* Geveva, World Health Organization, 2006 (http://whqlibdoc.who.int/publications/2006/9241547103_eng.pdf, accessed June 2009).
2. The Global Alliance to Eliminate Lymphatic Filariasis. (http://www.filariasis.org, accessed June 2009).

CD-WGE technical focal point: Department of Control of Neglected Tropical Diseases (NTD)

MALARIA

Description

Clinical description

Malaria usually begins with non-specific symptoms: headache, fatigue, abdominal discomfort and muscle and joint aches, followed by fever, chills, perspiration, anorexia, and vomiting.

If treatment for falciparum malaria is ineffective or delayed, the parasite will multiply and severe malaria may result. Features of severe malaria include: coma (cerebral malaria), metabolic acidosis, severe anaemia, hypoglycaemia and, in adults, acute renal failure or acute pulmonary oedema. If untreated, severe malaria is almost always fatal; with treatment, fatality is about 15–20%. Patients can progress from having minor symptoms to severe disease within a few hours.

In Côte d'Ivoire, young children are at risk of severe malaria, as are pregnant women in whom immunity is reduced. Adults are less likely to become severely ill because transmission is stable (i.e. populations are continuously exposed to more than 10 infectious bites per person per year) and most people acquire some degree of immunity during childhood.

Infectious agent

The main parasite species in Côte d'Ivoire is *Plasmodium falciparum*. This causes the most life-threatening form of the disease and accounts for > 90% of all deaths attributable to malaria. Other species that cause human malaria globally are *P. ovale, P. malariae,* and *P. vivax*.

Case definition

Uncomplicated malaria

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, muscle pains and fatigue).

Severe malaria

Patient with 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).

Confirmed case (uncomplicated or severe)

Patient with uncomplicated or severe malaria with laboratory confirmation of diagnosis by blood film for malaria parasites or other diagnostic tests for *Plasmodium* antigen.

Mode of transmission

This is a vector-borne disease, the vector being female *Anopheles* mosquitoes, which bite mainly between dusk and dawn. The major *Anopheles* species in Cote d'Ivoire are: *arabiensis, brochieri, coustani, funestus, gambiae, hancocki, hargreavesi, melas, moucheti, nili, paludis, and pharoensis. A. gambiae* accounts for more than 90% of transmission (see Table 9 for important biological features).

Rarely, malaria may also be transmitted through transfusion by injection of infected blood and in utero or during delivery.

Table 9. **Important biological features of major malaria vectors in Africa**

Anopheles species	Resting location	Feeding time and location	Host preferences	Breeding sites	Insecticide susceptibility
A. gambiae	Mainly indoors	Mainly late Indoors	Mainly humans	Sunlit temporary pools, rice fields	Resistance to DDT, HCH, and recently to pyrethroids in west Africa
A. arabiensis	Indoors and outdoors	Mainly late Indoors and outdoors	Humans and animals	Temporary pools, rice fields	Resistance to DDT, and to malathion in Sudan
A. funestus	Indoors	Mainly late Indoors	Mainly humans	Semi permanent and permanent water, especially with vegetation swamps, slow streams, ditch edges	Resistance to DDT, and recently to pyrethroids in southern Africa.

Source: Malaria control in complex emergencies. An inter-agency field handbook. Geneva, World Health Organization, 2005 (http://www.who.int/malaria/interagencyfieldhandbook.html, accessed June 2009).

DDT, **d**ichloro**d**iphenyl**t**richloroethane; HCH, hexachlorocyclohexane

Incubation period

The average incubation period for mosquito-transmitted *P. falciparum* is 9–14 days; *P. vivax,* 12–17 days (up to 6–12 months); *P. ovale*, 16–18 days or longer; and *P. malariae,* 18–40 days or longer.

Malaria should be considered in all cases of unexplained fever that start at any time between 1 week after the first possible exposure to malaria risk and 2 months (or even later in rare cases) after the last possible exposure.

Period of communicability

Transmission is related to the presence of infective female *Anopheles* mosquitoes and of infective gametocytes in the blood of patients. Untreated or insufficiently treated patients may be a source of infection for mosquitoes for up to 1–2 years for *P. falciparum*, 1.5–5 years for *P. ovale* and *P. vivax*, and 3–50 years for *P. malariae*.

Reservoirs

There are no significant animal reservoirs. *P. malariae* is common to humans and some primates.

Epidemiology

Disease burden

Malaria remains a major public health problem globally. In 2006 there were an estimated 247 million cases and 881 000 deaths; 91% of deaths occurred in Africa and 85% were of children aged less than 5 years. In most countries, reported cases under-represent the actual total number of malaria cases, since many cases are not reported to national health-information systems nor captured by public health services as the patient seeks care at private pharmacies or from traditional healers.

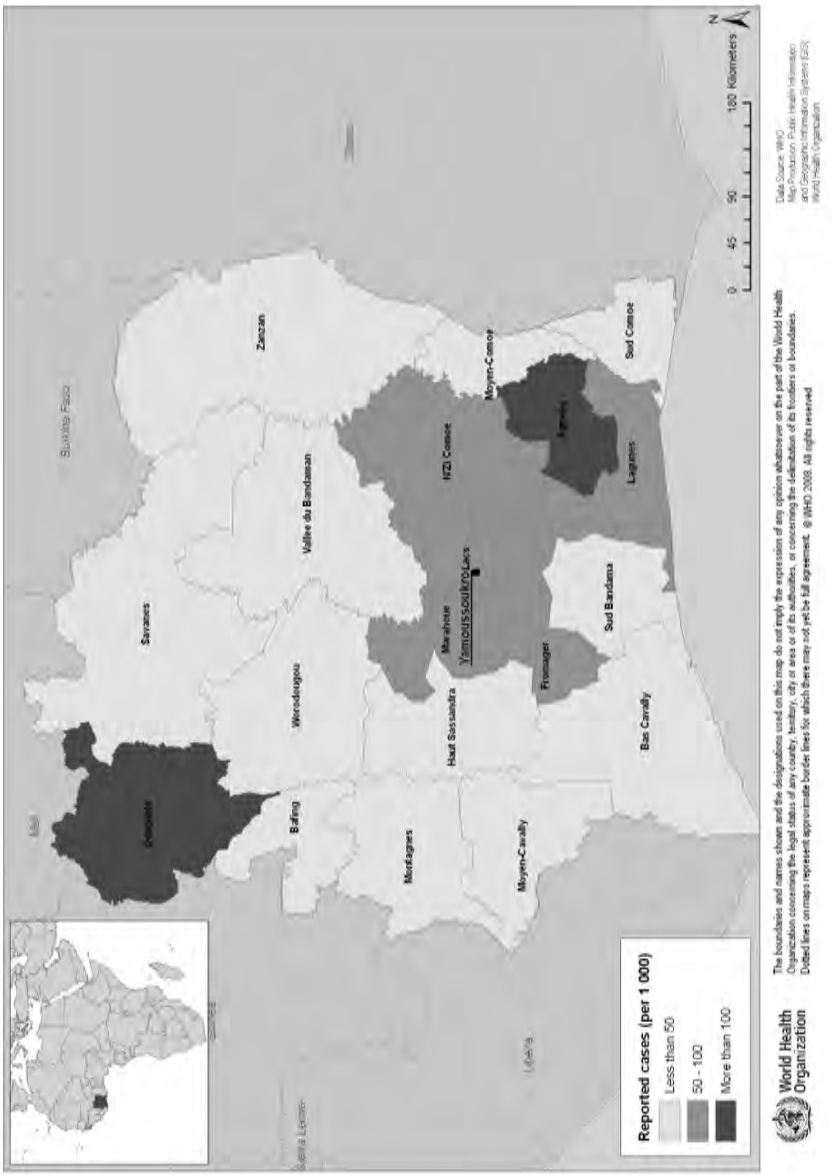
Table 10. **Estimated numbers of malaria cases and deaths, Côte d'Ivoire, 2006**

Parameter	Age group	Estimated number of cases	Lower estimate number cases	Upper estimate number cases
Fever suspected of being malaria	All ages < 5 years	21 572 000 12 056 000	13 289 000 2 016 000	30 133 000 23 275 000
Malaria cases	All ages < 5 years	7 029 000 3 928 000	4 330 000	9 818 000
Malaria deaths	All ages < 5 years	20 000 18 000	10 000 9 400	30 000 28 000
Malaria case-fatality ratio (%)	All ages < 5 years	0.28 0.46	— 	—

Source: *World malaria report 2008*. Geneva, World Health Organization, 2008 (*http://malaria.who.int/wmr2008/malaria2008.pdf*, accessed June 2009).

Côte d'Ivoire had an estimated 7 million malaria cases in 2006 (Table 10), i.e. 3% of all cases in the WHO African Region. Of the 1.3 million cases reported, none were confirmed as malaria. There was no evidence of a systematic decline in the number of malaria cases during 2001–2006. The number of deaths increased,

Fig. 7 **Geographical stratification of the burden of disease for malaria, Côte d'Ivoire, 2006**



perhaps due to improved reporting. Malaria is the most frequent cause of medical visits and hospitalization in Côte d'Ivoire and accounts for 33% of all hospital deaths. Children aged less than 5 years probably have around one to six malaria episodes each year (with an average of three episodes), and adults have around one to three episodes per year (more in rural settings). Malaria contributes to anaemia, neurological impairment and complications in pregnancy.

Geographical distribution

Malaria transmission occurs all over Cote d'Ivoire, ranging from high to very high. The geographical stratification of the disease burden is presented in Fig. 7.

Seasonality

Factors that influence transmission include altitude, rainfall, humidity, temperature and vegetation. Transmission occurs all year round throughout the country, but is more seasonal in the north, with upsurges during and just after the rainy season; heavy unseasonal rain may lead to an increase in the number of cases.

Outbreaks

Malaria transmission is stable so there is no risk of epidemic in the general population. Displaced populations arriving from areas of lower or no transmission of falciparum malaria may be at risk of an outbreak.

Alert threshold

Among populations displaced from areas of low endemicity, the following observations may be used to trigger an outbreak investigation:

- A doubling of the number of cases compared with the baseline (average weekly number of cases reported over the previous 2–3 weeks), adjusted for fluctuations in clinic attendance due to external factors such as a sudden population influx.
- An increase in the incidence of severe cases and an increase in the incidence of cases in children aged > 5 years and in adults.

Risk factors for increased burden

Population movement

Increased transmission and incidence associated with influx of less-immune populations from an area of lower endemicity to an area of higher endemicity.

Overcrowding

Increased population density may lead to increased exposure to mosquito bites in temporary shelters.

Poor access to health services

Delay in access to effective treatment increases the likelihood of severe disease and death. This delay also increases the pool of carriers of the malaria gametocyte (the mature sexual stage of the parasite in humans that, once picked up in the blood meal of a mosquito, develops into the infective stage for transmission to another human).

Food shortages

Malnutrition increases vulnerability to severe malaria once infected, and can mask the signs and symptoms of malaria, delaying clinical diagnosis and treatment and increasing mortality.

Lack of safe water, poor hygienic practices and poor sanitation

Temporary standing water may increase opportunities for breeding of the malaria vector, especially in arid environments (different vectors have different preferences for breeding sites).

Others

HIV co-infection worsens the manifestations and severity of malaria.

Prevention and control measures

Case management

Prompt access to effective treatment is one of the cornerstones of malaria control.

In children, the probability that fever in a child is due to malaria is high. Children under 5 years of age should therefore be treated on the basis of a clinical diagnosis. According to the Integrated Management of Childhood Illness (IMCI) guidelines, children aged less than 5 years with fever (and without runny nose, rash, or other cause of fever) should be treated with antimalarials according to the chart below (Table 11). The first dose of antimalarial (and paracetamol, if fever is > 38.5 °C) should be given in the clinic. Caregivers should be counselled on how to give the remaining doses of antimalarials, danger signs requiring immediate response, and when to return for follow up.

In older children and adults, including pregnant women, parasitological confirmation of the diagnosis is recommended before treatment is started. In all suspected cases of severe malaria, parasitological confirmation of the diagnosis is recommended. However, in the absence of or a delay in obtaining parasitological confirmation, patients should be treated for severe malaria on clinical grounds.

Artemesinin-based combination therapy (ACT) for the treatment of uncomplicated malaria was introduced in 2003 (replacing chloroquine as first-line therapy). The protocol is given in Table 11, and dosing schedules in Tables 12–14.

ACT is still not widely available. A survey conducted in Côte d'Ivoire in 2006 reported that only 3% of febrile children were given ACT. To improve access to effective antimalarials, community-based IMCI, including malaria treatment, has been initiated by the United Nations Children's Fund (UNICEF) through two field offices, one in Buoake, covering the central and northern part of the country and another in Man, covering the western regions.

Table 11. **Treatment guidelines for falciparum malaria in Côte d'Ivoire**

Case	Recommended treatment[a]	Comments
Uncomplicated malaria, unconfirmed	Artesunate and amodiaquine (AS+AQ)[b]	Separate tablets containing 50 mg of artesunate and 153 mg base of amodiaquine.
Uncomplicated malaria, laboratory confirmed	Artesunate and amodiaquine (AS+AQ)[a]	Dose: 4 mg/kg of artesunate and 10 mg base/kg of amodiaquine given once per day for 3 days (co-formulated tablets or co-blister packages are now available and recommended by WHO)
Treatment failure	Artemether-lumefanthrine (AL)[b]	Co-formulated tablets (20 mg of artemether + 120 mg of lumefantrine) 6 dose regime twice per day for 3 days
Severe malaria	Quinine (QN) (7 days)	Loading dose IV 20 mg QN salt/kg; maintenance at 30 mg QN salt/kg per day divided into three doses, given every 8 hours
Malaria in pregnancy,[a] treatment	Quinine (QN)	
Malaria in pregnancy,[a] prevention	Sulfadoxine-pyrimethamine intermittent prevention therapy SP (IPT)	See later section on prevention

Source: Global Antimalarial Drug Policies (AMDP) database. AFRO. Geneva, World Health Organization, updated May 2008 (*http://www.who.int/malaria/amdp/amdp_afro.htm*, accessed June 2009); *Guidelines for the treatment of malaria*. Geneva, World Health Organization, 2006 (http://whqlibdoc.who.int/publications/2006/9241546948_eng.pdf; accessed August 2009).

[a] Current Côte d'Ivoire guidelines recommend quinine throughout pregnancy. Based on the side-effects and poor adherence associated with quinine, WHO recommends quinine ± clindamycin for the first trimester and for the second and third trimesters, either the ACT being used in the country/region, or artesunate + clindamycin, or quinine + clindamycin. Lactating women should receive standard treatment for uncomplicated malaria.

[b] Policy adopted, not currently being deployed, implementation process ongoing.

Table 12. **Dosing schedule for artesunate + amodiaquine as separate tablets**

Age group	Dose in mg (number of tablets)					
	Artesunate (50 mg)			Amodiaquine (153 mg)		
	Day 1	Day 2	Day 3	Day 1	Day 2	Day 3
5–11 months	25 (1/2)	25	25	76 (1/2)	76	76
≥ 1–6 years	50 (1)	50	50	153 (1)	153	153
≥ 7–13 years	100 (2)	100	100	306 (2)	306	306
≥ 13 years	200 (4)	200	200	612 (4)	612	612

Source: *Guidelines for the treatment of malaria*. Geneva, World Health Organization, 2006 (*http://www.who.int/malaria/docs/TreatmentGuidelines2006.pdf*, accessed June 2009)

Table 13. **Dosing schedule for coformulated tablets containing artesunate + amodiaquine**

Age (body weight in kg)	Artesunate + amodiaquine (tablet strength)	Number of tablets, once daily for 3 days of treatment
2–11 months (4.5–8 kg)	25 mg + 67.5 mg	1
1–5 years (9–17 kg)	50 mg + 135 mg	1
6–13 years (18–35 kg)	100 mg + 270 mg	1
≥ 14 years (≥ 36 kg)	100 mg + 270 mg	2

Table 14. **Dosing schedule for artesunate + amodiaquine as separate tablets**

Body weight (kg)	Age (years)	Number of tablets at approximate timing of dosing[a]					
		0 hours	8 hours	24 hours	36 hours	48 hours	60 hours
5–14	< 3	1	1	1	1	1	1
15–24	≥ 3–8	2	2	2	2	2	2
25–34	≥ 9–14	3	3	3	3	3	3
> 34	≥ 14	4	4	4	4	4	4

Source: Guidelines for the treatment of malaria. Geneva, World Health Organization, 2006 (http://whqlibdoc.who.int/publications/2006/9241546948_eng.pdf accessed June 2009).

[a] The regimen can be expressed more simply for ease of use at the programme level as follows: the second dose on the first day should be given any time between 8 hours and 12 hours after the first dose. Dosage on the second and third days is twice per day (morning and evening).

Treatment failure within 14 days should be treated with the second-line therapy – artesunate-lumefanthrine. Treatment failure after 14 days should be treated with the first-line therapy.

Globally, resistance has arisen to every class of antimalarial drugs including the artemisinin derivatives. Indiscriminate usage of antimalarial drugs as monotherapy needs to be addressed; unfortunately, Côte d'Ivoire is one of 42 countries that still allow marketing of oral artemisinin-based monotherapies for treatment of uncomplicated malaria. As antimalarial-drug pressure varies between regions, incoming resistant strains of malaria can readily become established in new areas. Côte d'Ivoire is part of the West African Network for Monitoring Antimalarial Treatment (WANMAT II).

Laboratory capacity

Laboratory diagnosis is by demonstration of malaria parasites in a peripheral blood film (thick or thin smear) or by rapid diagnostic test. Laboratory tests for the diagnosis of malaria in Côte d'Ivoire are not widely available. Microscopy services are available at regional and district hospitals, other district health facilities and private laboratories. Malaria diagnosis is done by rapid diagnostic tests (RDTs) in some clinics. There are several types of RDTs. Tests that detect histidine-rich protein-II (HRP-II) are commonly used for identifying falciparum infection. They may continue to produce positive test results for up to 14 days after effective treatment of a malaria infection, even when patients no longer have detectable parasites on microscopy. RDTs should therefore not be used to assess parasite clearance or for re-screening treated patients. RDTs may lose their sensitivity when stored in hot and humid conditions and should usually be stored at < 30 °C. It is recommended that heat-stability data should be requested from the manufacturer before purchase.

Prevention

Key preventive strategies include:

Intermittent preventive treatment in pregnancy (IPTp)

Côte d'Ivoire has adopted **IPTp** with sulfadoxine-pyrimethamine (SP) at least twice during pregnancy (during the second and third trimesters). IPTp is recommended for pregnant women living in areas where transmission is high and where SP is > 80% effective and is best implemented through ante-natal care.

Health education

Active health education at community level is important to improve rapid treatment-seeking behaviour for fever cases, for effective use of insecticide-treated nets, and improving acceptability of indoor residual spraying with insecticide.

Vector control

- *Long-lasting insecticidal nets (LLINs*): Insecticide-treated nets provide personal protection for all those who sleep under the net. They also have the potential to significantly reduce the adult mosquito population when coverage is greater than 80% (community impact), thereby reducing transmission and subsequent morbidity and mortality. Insecticide-treated nets can be distributed with integrated mass vaccination campaigns or as stand-alone distributions. Health education on proper use and care is vital for the success of programmes distributing insecticide-treated nets. LLINs are insecticide-treated nets that do not need to be retreated with insecticide every 6 months; they are therefore the intervention of choice, especially when working with displaced and conflict-affected populations.

Currently, only a small portion of the population uses insecticide-treated nets. The National Malaria Control Programme (NMCP) distributed only 370 000 LLINs in 2006. Only 27% of households owned a mosquito net in 2006, and just 6% had an insecticide-treated net.

It is a matter for concern that pyrethroid resistance in *Anopheles gambiae* has been recognized in Côte d'Ivoire since 1993. The effectiveness of pyrethroids for malaria control in Côte d'Ivoire needs to be monitored.

- *Indoor residual spraying (IRS):* Periodic indoor spraying of shelters with residual insecticide can reduce transmission when applied according to WHO recommendations and when the following conditions are met:
  - A high percentage of the structures in an operational area have adequate sprayable surfaces, and can be expected to be well sprayed;
  - The majority of the vector population is endophilic, i.e. rests indoors;
  - The vector is susceptible to the insecticide in use.

The main purpose of IRS is to reduce transmission by reducing the survival of malaria vectors entering houses or sleeping units. IRS is not applicable during acute phases of emergencies. It can be useful in well-organized temporary settle-

ments or camps (*1*). IRS is not currently part of the national malaria control strategy in Côte d'Ivoire.

- *Environmental control:* Environmental control may be difficult during the acute phase of an emergency except on a local scale, and impact is often limited. To reduce the number of vector breeding sites:
  - Drain clean water around water tap stands and rain water drains;
  - Use larvicides in vector breeding sites if these are limited in number (seek expert advice);
  - Drain ponds (although this may not be acceptable if ponds are used for washing and/or for animals).

Immunization

None available at present, several candidate vaccines are under investigation.

References

1. *Indoor residual spraying. Use of indoor residual spraying for scaling up global malaria control and elimination*. Geneva, World Health Organization, 2006 (http://apps.who.int/malaria/docs/IRS/IRS-position.pdf, accessed June 2009).

Further reading

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CD-WGE technical focal point: Global Malaria Programme (GMP)

MEASLES

Description

Clinical description

Measles is a highly communicable disease, characterized by a prodrome of fever, conjunctivitis, coryza, cough and small whitish spots on the buccal mucosa (Koplik spots), and a characteristic red blotchy rash appearing on day 3–7 of illness. The rash usually begins on the face, then spreads to rest of the body, and lasts 4–7 days.

Most children will have uncomplicated measles. Disease tends to be severe in infants and adults. Complications of measles include acute otitis media, diarrhoea, bronchopneumonia, and laryngotracheobronchitis and encephalitis. They are more likely to occur in the very young, in malnourished children, or those who are immunocompromised.

The case-fatality ratio (CFR) in developing countries is generally 1–5%, but may be as high as 25% in populations with high levels of malnutrition and poor access to health care.

Infectious agent

Measles virus (genus *Morbillivirus*, family Paramyxoviridae).

Case definition

Suspected (clinical) case:

Any person with:

- Fever **and**
  - Maculopapular rash (i.e. non vesicular), **and**
  - Cough, coryza (i.e. runny nose) **or** conjunctivitis (i.e. red eyes);
- or any person in whom a clinical health worker suspects measles infection.

Confirmed case:

A case that meets the clinical case definition and has laboratory-confirmed presence of measles-specific IgM antibodies.

Confirmed through epidemiological linkage
A case that meets the clinical case definition and is from the same district or zone where an outbreak has been laboratory-confirmed during the previous 30 days.

Cases may be clinically further classified as **uncomplicated** (simple) or **complicated** by the presence or absence of medical complications (pneumonia, diarrhoea, stomatitis, malnutrition, encephalitis, otitis media, croup, or, with a long delay, subacute sclerosing panencephalitis).

Mode of transmission

Airborne by droplet spread (coughing, sneezing), direct contact with nasal and throat secretions of infected people or via objects (e.g. toys) that have been in close contact with an infected person. The virus remains viable in the air or on infected surfaces for about 2 hours.

Incubation period

Generally 10–12 days (range, 7–18 days) from exposure to the onset of fever.

Period of communicability

Measles is most infectious from 4 days before the rash until 4 days after rash onset; minimal after the second day of rash.

Reservoirs

Humans.

Epidemiology

Disease burden

Measles is one of the most contagious diseases known, and remains a leading cause of death among children worldwide. In 2006, an estimated 242 000 deaths were caused by measles globally. Death due to measles mostly occurs in countries that are poor, have a weak health infrastructure or, are experiencing or recovering from war, civil strife or natural disaster. Infection rates soar as a result of deterioration of routine immunization coverage and increased transmission among internally displaced persons due to crowded living conditions.

The 2005 World Health Assembly, as part of the Global Immunization Vision and Strategy document (*1*), adopted an ambitious global goal: to achieve a 90% reduction in measles mortality by 2010 compared with 2000.

Côte d'Ivoire is one of 47 priority countries targeted by the *Global Plan for reducing measles mortality 2006-2010*; together these countries account for more than 95% of measles deaths globally (*2*).

Disruption of immunization programmes due to civil unrest, culminating in the 2002 civil war, led to a rise in rates of measles in Côte d'Ivoire and also had negative repercussions on neighbouring countries, as exemplified by the measles outbreak in Burkina Faso in 2002. Between 2000 and 2003, measles accounted for 4% of all deaths in children aged less than 5 years in Côte d'Ivoire.

With concerted efforts to increase routine immunization services and a nationwide supplementary immunization activity in 2005 that reported 88% coverage among children aged 9 months to 14 years, the annual number of reported cases has steadily declined from 5729 cases in 2000 to 5 cases in 2007 (Table 15).

Côte d'Ivoire conducted its first follow-up campaign in 11–15 November 2008, targeting 3.2 million children aged 9–59 months, combined with distribution of vitamin A, de-worming medications, and insecticide-treated bed nets in selected districts.

Table 15. **Measles cases reported and vaccination coverage, Côte d'Ivoire, 2003–2007**

Year	Annual number of reported cases	
2007	5	67
2006	11	73
2005	117	51
2004	4010	49
2003[a]	5207	56
2002	7633	56
2001	5790	61
2000	5729	73

Source: *Measles reported cases*. Geneva, World Health Organization, updated 2 June 2009 (http://www.who.int/Immunization_monitoring/en/globalsummary/timeseries/tsincidencemea.htm, accessed June 2009); *Measles-containing vaccine. Reported estimates of MCV coverage*. Geneva, World Health Organization, updated 2 June 2009 (http://www.who.int/Immunization_monitoring/en/globalsummary/timeseries/tscoveragemcv.htm, accessed June 2009).

[a] Case-based surveillance started in 2003. Figures for 2000–2004 indicate suspected cases; confirmed cases are 86 for 2003 and 87 for 2004

Geographical distribution

Reported cases by department for the years 2003–2007 are shown in Fig. 8.

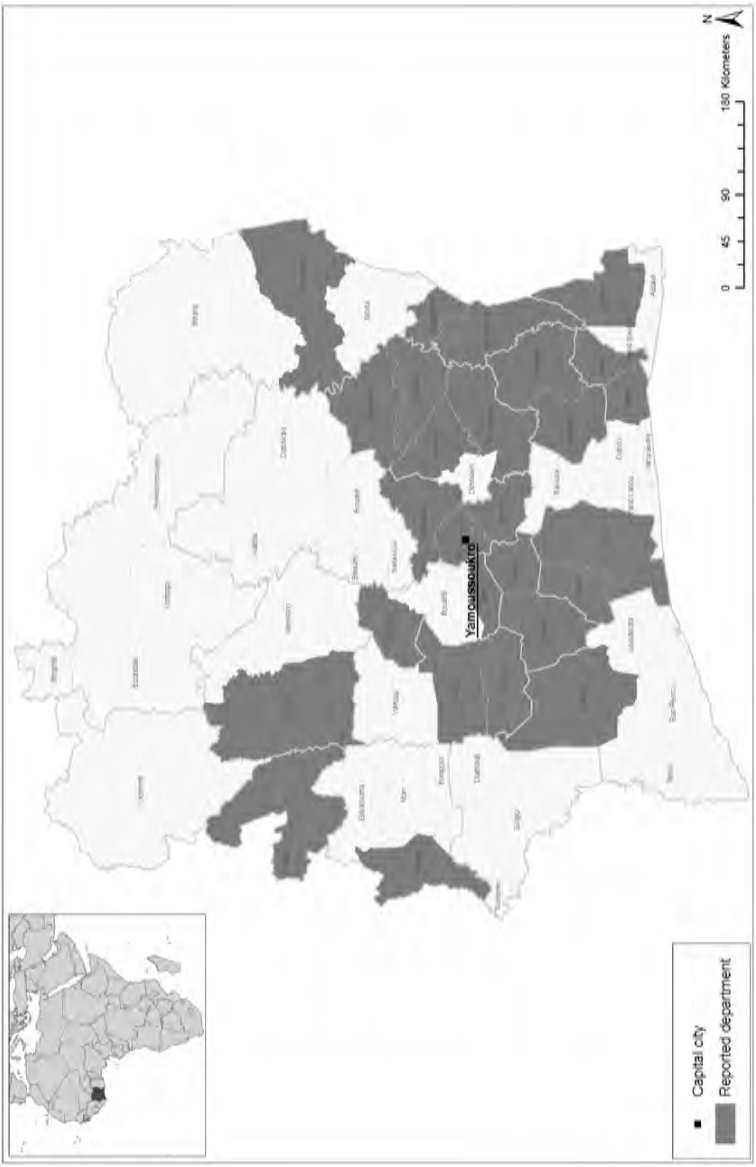


Fig. 8 **Departments reporting cases of measles, Côte d'Ivoire, 2003–2007**

Seasonality

The highest incidence of cases is usually observed between the end of the dry season and the beginning of the rainy season (March/April).

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 to be laboratory-confirmed.

Risk factors for increased burden

Population movement

Migration of non-immune populations into areas where the pathogen is circulating or of infected individuals into areas where the population is not immunized leads to increased risk of transmission. Large population migrations, particularly in association with war and unrest, compromise vaccination programmes.

Overcrowding

Crowded conditions and poor indoor ventilation facilitate rapid transmission.

Poor access to health services

Weak immunization programmes with low coverage increase the numbers of people susceptible to measles infection. Delayed case recognition delays implementation of patient isolation and immunization of contacts. The risk of case fatality increases in the absence of effective management of severe cases. Poor access to child health-care services, as a result of which nutrition and growth of young children is not assessed and malnutrition is not accordingly corrected, may contribute to a higher risk of complications following measles.

Food shortages

Disease is more severe among children with malnutrition and vitamin A deficiency; it can trigger acute protein-energy malnutrition and worsen vitamin A deficiency; malnourished children are at a higher risk of complications and death following measles.

Lack of safe water, poor hygienic practices and poor sanitation

Children living under these conditions usually have poor nutrition and repeated attacks of communicable diseases often precipitate acute malnutrition.

Prevention and control measures

Case management

Report to local health authority; isolate where possible (e.g. do not send to school). As there is no specific antiviral treatment, vitamin A supplementation is essential in resource-poor settings as it minimizes the complications of the disease (Table 16).

For uncomplicated cases

- Give vitamin A at a treatment dose on days 1, 2 and 15 (the second dose can be given to the caregiver to give at home).
- Advise the parent to treat the child at home (control fever and provide nutritional feeding).

For cases with non-severe eye, mouth or ear complications

- Children can be treated at home.
- Give vitamin A at a treatment dose on days 1, 2 and 15 (the second dose can be given to the caregiver to give at home).
- If pus is draining from the eyes, clean eyes and treat with 1% tetracycline eye ointment.
- If there are mouth ulcers, treat with gentian violet.
- If pus is draining from the ear, clean ear discharge and treat with antibiotics for 5 days (first-line, amoxicillin; or second-line, co-trimoxazole), according to national policy for acute respiratory infections and the Integrated Management of Childhood Illness (IMCI) guidelines.
- Treat malnutrition and diarrhoea, if present, with sufficient fluids and high-quality diet.

For cases with severe, complicated measles (any general danger signs such as inability to drink or breastfeed, repeated vomiting, convulsions, lethargy or unconsciousness, clouding of cornea, deep or extensive mouth ulcers, pneumonia)

- 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.
- Give vitamin A at a treatment dose on days 1, 2 and 15.

Vitamin A should not be given to females who may be pregnant.

Table 16. **Doses of vitamin A for measles treatment regimens**

Age	Vitamin A dose	
	Immediately on diagnosis	Following day
Infants aged < 6 months	50 000 IU	50 000 IU
Infants aged 6–11 months	100 000 IU	100 000 IU
Children aged > 11 months	200 000 IU	200 000 IU

Source: *Vitamin A supplements. A guide to their use in the treatment and prevention of vitamin A deficiency and xeropthalmia*. Geneva, World Health Organization, 1997 (http://whqlibdoc.who.int/publications/1997/9241545062.pdf; accessed August 2009).

Prevention

Immunization is the key to prevention. Improving and maintaining a high coverage of routine immunization is important to prevent outbreaks.

Immunization

Currently, measles vaccine is part of the national immunization schedule and is administered as a single dose to infants at age 9 months. This is a live vaccine and should induce active immunity in 85–90% of susceptible patients, probably for life. Regular follow-up vaccination campaigns every 3–4 years that target children born since the last campaign provide a second opportunity for measles immunization of those children who missed their first dose or did not develop an immune response after the first dose. After two doses of measles vaccine, 99% of children are immune to measles. About 5-15% of non-immune individuals may develop fevers and malaise within 5–12 days after vaccination and that lasts 1–2 days.

The live measles vaccine should also be administered to contacts within 72 hours of exposure. Immunoglobulin – where available – has been used after exposure for immunocompromised patients, for whom live vaccines are relatively contraindicated.

Epidemic control

In an acute emergency, immunize the population at risk as soon as possible if vaccination coverage is less than 80–90% or unknown. The priority is to **immunize children aged 6 months to 15 years**, regardless of vaccination status or disease history. Expansion to older age groups is of lower priority and should be based on evidence of high susceptibility among the age group. If vaccine supplies are limited, the age range may be reduced (e.g. 6 months to 12 years, or 6 months to 5 years). Vitamin A supplementation should be provided as above to those aged less than 5 years and should be considered in older children if clinical deficiency of vitamin A is suspected. Children who are vaccinated against measles at less than 9 months of age must receive a second measles vaccination, which should be given as soon as possible after reaching age 9 months, with an interval of at least 1 month between doses.

Outbreak response should include the following:

- Inform health authorities if one or more suspected cases are identified.
- Confirm the suspected outbreak following WHO guidelines (3) (also see Annex 2).
- Investigate suspected case: check whether case fulfils the case definition, record date of onset, age and vaccination status.
- Confirm the diagnosis: collect blood specimens from three to five initial reported cases.
- Assess the extent of the outbreak and the population at risk.
- Implement outbreak-response measures as follows:
  - Give priority to proper case management and immunization of groups at highest risk (e.g. children aged 6 months to 15 years) as soon as possible, even in areas not yet affected where the outbreak is likely to spread. Reduce age range if vaccines are limited.
  - Through social mobilization, ensure parents bring previously unvaccinated children for immunization.
  - The presence of several cases of measles in an emergency setting is an indication for a measles immunization campaign. Even among individuals who have already been exposed, and are incubating the natural virus, measles vaccine, if given within 3 days 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.

References

1. Global Immunization Vision and Strategy. Geneva, World Health Organization, 2009 (http://www.who.int/immunization/givs/en/index.html; accessed August 2009).
2. *WHO/UNICEF joint statement - global plan for reducing measles mortality 2006-2010*. Geneva, World Health Organization, 2006 (http://www.who.int/immunization/documents/WHO_IVB_05.11/en/index.html; accessed August 2009).
3. *WHO Guidelines for Epidemic Preparedness and Response to Measles Outbreaks*. Geneva, World Health Organization, 1999 (http://whqlibdoc.who.int/hq/1999/WHO_CDS_CSR_ISR_99.1.pdf; accessed August 2009).

CD-WGE technical focal point: Department of Immunization, Vaccines and Biologicals (IVB)

MENINGOCOCCAL DISEASE

Description

Clinical description

Meningococcal disease is an acute bacterial disease, which can present in three main clinical forms: the meningeal syndrome, the septic form and pneumonia. Meningeal syndrome is the most common presentation, while meningococcal sepsis is the more severe form of infection leading to disseminated intravascular coagulation and multi-organ failure. The onset of symptoms is sudden and death can follow within hours. In as many as 10–15% of survivors, there are persistent neurological defects, including hearing loss, speech disorders, intellectual impairment, and paralysis.

Infectious agent

Bacterium *Neisseria meningitidis*. Serogroups A, B, C, X, Y, W135.

Case definition

Suspected case:

An illness with sudden onset of fever (rectal, > 38.5 °C; axillary, > 38.0 °C) **and one or more** of the following:

- Neck stiffness;
- Altered consciousness;
- Other meningeal sign **or** petechial or purpural rash.

In patients aged less than 1 year, meningitis should be suspected when fever is accompanied by a bulging fontanelle.

Probable case:

A suspected case as defined above **and:**

- Turbid cerebrospinal fluid (CSF), with or without positive Gram stain; **or**
- Continuing epidemic.

Confirmed case:

A suspected or probable case with laboratory confirmation through

- Positive detection of antigen in CSF; **or**
- Positive bacterial culture.

Mode of transmission

Direct contact with respiratory droplets; 5–10% may have asymptomatic nasopharyngeal carriage.

Incubation period

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. Asymptomatic carriers are the most important source of infection.

Reservoirs

Humans.

Epidemiology

Disease burden

The highest burden of meningococcal disease occurs in sub-Saharan Africa in an area known as the "meningitis belt", which covers 15 countries between Ethiopia and Senegal. In the last decade, outbreaks of meningococcal disease have been reported in areas adjacent to the classical meningitis belt and also in several countries in eastern Africa and southern Africa, suggesting that the meningitis belt may be expanding.

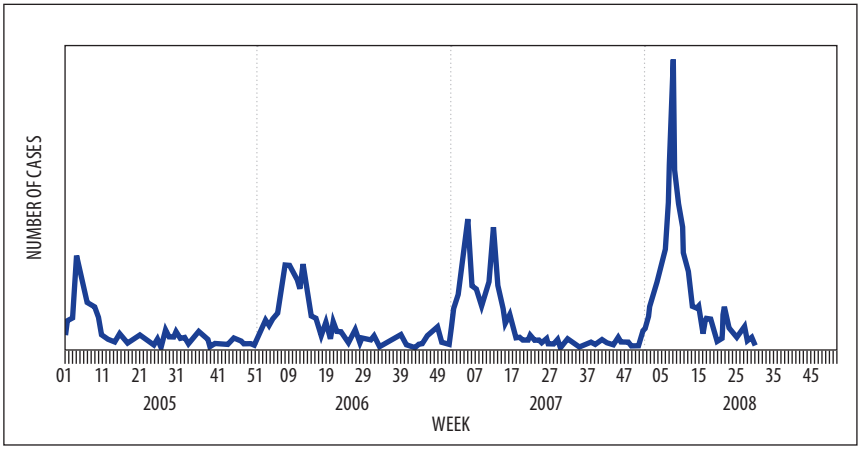
Twelve subtypes or serogroups of *Neisseria meningitidis* have been identified and five (*N. meningitidis* A, B, C, X and W135) are recognized as causing epidemics. Pathogenicity, immunogenicity, epidemic capabilities and vaccines differ according to serogroup. Thus the identification of the serogroup responsible for a sporadic case is crucial for epidemic containment.

In 1996, Africa experienced the largest recorded outbreak of epidemic meningitis in history, with more than 250 000 cases and 25 000 deaths registered. In 2008,

countries in the African meningitis belt reported a total of 27 985 cases, including 2578 deaths (case-fatality ratio, CFR, 9.2%) from 1 January to 25 May 2008. Burkina Faso reported the most cases (> 9000). The number of cases increased in 2008 in Côte d'Ivoire (Fig. 9) compared with previous years. *Neisseria meningitidis* A (Nm A) was identified as the causative agent in all confirmed epidemics in 2008. NmW135 was responsible for sporadic cases in Benin, Côte d'Ivoire, Chad and Togo, while NmX was detected in a few cases in Niger.

The CFR for meningococcal disease in Côte d'Ivoire is high, ranging from 16% to 25% (Table 17). Long-term neurological sequelae compound the associated morbidity of meningococcal disease.

Fig. 9 **Number of cases of meningococcal disease reported in Côte d'Ivoire by week, 2005–2008 (up to week 31)**



Source: MDSC Meningitis Weekly Bulletin (*http://www.who.int/csr/disease/meningococcal/Bulletin%20Meningite%202008_S27_31%20juillet.pdf*, accessed June 2009).

Table 17. **Morbidity, mortality and case fatality caused by meningococcal disease in Côte d'Ivoire, 2004–2008**

Year	Reported cases	Reported deaths	Case-fatality ratio (%)	Epidemic districts
2008 (up to 3 August)	1020	167	16.4	2
2007	760	190	25.0	2
2006 (up to 19 November)	656	105	16.0	1
2005	527	100	19.0	1
2004	464	74	15.9	1

Source: *Meningitis season 2007–2008: moderate levels of meningitis activity*. Geneva, World Health Organization, dated 9 July 2008 (http://www.who.int/csr/disease/meningococcal/meningitisesepidreport2007_2008/en/index.html, accessed June 2009).

Geographical distribution

Only the northern region of Côte d'Ivoire lies within the meningitis belt, and it is this area that has been affected by outbreaks in the past. In 2008, regions bordering Burkina Faso were particularly affected.

Seasonality

Outbreaks are seen at the time of the year when absolute humidity is low and when a hot, dusty wind (the Harmattan) blows from the Sahara. In Côte d'Ivoire, the peak season is from January to April.

Alert threshold

Population > 30 000: five cases per 100 000 inhabitants per week, or a cluster of cases in an area

Population < 30 000: two cases in 1 week or an increase in the number of cases compared with previous non-epidemic years.

Intervention: Inform authorities; investigate; confirm; treat cases; strengthen surveillance; prepare for epidemic.

Epidemic thresholds

Population > 30 000:

10 cases per 100 000 inhabitants per week **if**

- No epidemic for 3 years and vaccination coverage < 80%;
- Alert threshold crossed early in the dry season.

15 cases per 100 000 inhabitants per week in other situations

Population < 30 000:

- Five cases occurring in 1 week; **or**
- Doubling of the number of cases each week in a 3-week period; **or**
- For mass gatherings, refugees and displaced persons, two confirmed cases in 1 week are enough to initiate vaccination of the population;
- Other situations should be studied on a case-by-case basis.

Intervention: Mass vaccination; distribute treatment to health centres; treat according to epidemic protocol; inform the public.

Risk factors for increased burden

Population movement

Population movement of infected persons or asymptomatic carriers facilitates the circulation of strains within a country, or from country to country.

Overcrowding

Crowding of susceptible people and poor indoor ventilation are important risk factors for outbreaks. Crowding during complex emergencies, or because of cattle or fishing-related activities, or in military camps and schools, facilitates spread of the disease.

Poor access to health services

Case identification is crucial for rapid implementation of control measures. CFR in the absence of treatment can be very high (50%).

Food shortages

Not relevant.

Lack of safe water, poor hygienic practices and poor sanitation

Poor respiratory hygiene practice with regards to sneezing and coughing facilitates transmission.

Prevention and control measures

Case management

Meningococcal disease (either meningitis or septicaemia) is potentially fatal and should always be considered as a medical emergency.

Non-epidemic conditions

- 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.
- As infectivity of patients is moderate and disappears quickly after 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 (*N. meningitidis, Streptococcus pneumoniae and Haemophilus influenzae*) **until bacteriological results are available** (see Table 18).

Table 18. **Initial empirical anti-microbial therapy for presumed bacterial meningitis**

Age group	Probable pathogens	Antimicrobial therapy	
		First choice	Alternative
Epidemic situations			
Adults and children aged > 5 year	*N. meningitidis*	Oily chloramphenicol or ceftriaxone	Ampicillin or amoxicillin
Children aged 2 to 5 years	*H. influenzae* *S. pneumoniae* *N. meningitidis*	Oily chloramphenicol or ceftriaxone	Ampicillin or amoxicillin
Children aged 2 to 23 months	*H. influenzae* *S. pneumoniae* *N. meningitidis*	Ceftriaxone	Ampicillin or amoxicillin
Neonates	*S. agalactiae* *S. pyogenes* *Enterobacteria*	Ceftriaxone	Ampicillin or amoxicillin

Age group	Probable pathogens	Antimicrobial therapy	
		First choice	Alternative
Non-epidemic situations			
Adults and children aged > 5 years	*N. meningitidis* *S. pneumoniae*	Ceftriaxone	Ampicillin or amoxicillin
Children aged 2 months to 5 years	*H. influenzae* *S. pneumoniae* *N. meningitidis*	Ceftriaxone	Ampicillin or amoxicillin
Neonates	*S. agalactiae* *S. pyogenes* *Enterobacteria*	Ceftriaxone	Ampicillin or amoxicillin

Source: *Standardized treatment of bacterial meningitis in Africa in epidemic and non epidemic situations.* Geneva, World Health Organization, 2007 (WHO/CDS/EPR/2007.3) (http://www.who.int/csr/resources/publications/meningitis/WHO_CDS_EPR_2007_3/en/index.html, accessed June 2009).

Once diagnosis of meningococcal disease has been established treatment should be adapted to the causative pathogen and its antibiotic sensitivity, according to Table 18.

Prevention

Chemoprophylaxis may be appropriate in a small cluster of cases, but not in an epidemic. See section on epidemics and control for details on vaccination as prevention.

Immunization

There are a number of polysaccharide vaccines available – quadrivalent with serogroups A,C,Y,W-135, trivalent (A,C,W135) and bivalent (A,C). Polysaccharide vaccines are useful in outbreak control. Ensuring that vaccination takes place early in the course of an outbreak is difficult.

Meningococcal conjugate vaccines, which induce immunological memory and, in contrast to polysaccharide vaccines, have a substantial effect on nasopharyngeal carriage, provide a possible means of finally eliminating African meningococcal epidemics. A new monovalent group-A conjugate vaccine is being developed in India, specifically for use in Africa (*1*). The latter vaccine should be ready for introduction in 2009–2010, allowing for a true prevention strategy to be implemented.

Epidemic control

Epidemic conditions

During epidemics of confirmed meningococcal disease, case management needs to be simplified to permit the health system to respond to rapidly increasing numbers of cases.

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 having meningococcal meningitis.

Treatment: Refer to Table 18 and to *Standardized treatment of bacterial meningitis in Africa in epidemic and non epidemic situations* (*2*) for the appropriate protocol.

A mass vaccination campaign can halt an epidemic of meningococcal disease if carried out appropriately. Laboratory diagnosis and confirmation of epidemic serogroups will guide the type of vaccine needed, either meningococcal polysaccharide bivalent A,C (if serogroup A or C is confirmed as the epidemic serogroup), or meningococcal polysaccharide trivalent A,C,W135 (if serogroup W135 is confirmed). Vaccination should be concentrated in the area where the epidemic is maximal.

- **Camp settings:** After confirmation (serogroup identified) of two cases, mass vaccination is recommended with the appropriate vaccine containing the responsible serogroup, either the bivalent (A,C) or trivalent (A,C,W135) vaccine. At-risk populations 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.

Chemoprophylaxis: Chemoprophylaxis of contacts of meningitis patients is not warranted during an epidemic in Africa. In small clusters or outbreaks among closed populations (e.g. extended household, boarding schools), chemoprophylaxis may still be appropriate.

References

1. *The Meningitis Vaccine Project*. Geneva, World Health Organization, posted 2004 (http://www.who.int/vaccines/en/olddocs/meningACproject.shtml; accessed August 2009).
2. *Standardized treatment of bacterial meningitis in Africa in epidemic and non epidemic situations.* Geneva, World Health Organization, 2007 (WHO/CDS/EPR/2007.3) (http://www.who.int/csr/resources/publications/meningitis/WHO_CDS_EPR_2007_3/en/index.html, accessed June 2009).

CD-WGE technical focal point: Department of Global Alert and Response (GAR)

ONCHOCERCIASIS (RIVER BLINDNESS)

Description

Onchocerciasis is not a fatal disease, but its socioeconomic impact on affected populations can be severe if left untreated. It is the world's second leading infectious cause of blindness.

Clinical description

People suffering from onchocerciasis may experience:

- **Skin lesions:** dermal changes are secondary to tissue reaction to the motile larvae as they migrate subcutaneously or to their destruction in the skin.
- **Itching:** the pruritus of onchocerciasis is the most severe and intractable that is known. In lightly-infected individuals, this may persist as the only symptom.
- **Rashes:** the rash usually consists of many raised papules, which are due to microabscess formation, and may disappear within a few days or may spread. *Sowda*, from the Arabic for black or dark, is an intensely pruritic eruption usually limited to one limb and including oedema, hyperpigmented papules and regional lymphadenopathy.
- **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, usually measuring 0.5–2.0 cm in diameter but can exceed 6 cm in some cases. They harbour adult worms, usually a male and two females. They occur most frequently over bony prominences: in Africa, the nodules are often located over the hips and lower limbs. They may be located on the cranium, particularly, in children.
- **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. Involvement of all tissues of the eye has been described, and many changes in both anterior and posterior segments of the eye can occur. The more serious lesions lead to serious visual impairment including blindness.

- **General debilitation:** onchocerciasis has also been associated with weight loss and musculoskeletal pain.

Infectious agent

Nematode (roundworm): *Onchocerca volvulus*

Case definition

Suspected case:

In an endemic area, a person with palpable fibrous nodules in subcutaneous tissues. These must be distinguished from lymph nodes or ganglia.

Confirmed case:

A suspected case that is laboratory-confirmed through the presence of one or more of the following:

- Microfilariae in skin snips taken from the iliac crest (Africa) or scapula (Americas);
- Adult worms in excised nodules;
- Positive result for a diethylcarbamazine citrate (DEC) patch test;
- Typical ocular manifestations, such as slit-lamp observations of microfilariae in the cornea, the anterior chamber or the vitreous body;
- Serology (especially for non-indigenous people).

Mode of transmission

Transmitted through the bites of infected female blackflies of *Simulium* species that carry immature larvae forms of the parasite from human to human. The blackfly lays its eggs in the water of fast-flowing rivers which hatch after 1–4 days, depending on the water temperature. Adult blackflies emerge from the larvae after 8–12 days and can live up to 4 weeks, during which time they can cover hundreds of kilometres in flight.

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 through the bite wound during a blood meal.

Infective larvae develop into adult parasites in the human body where adult forms of *O. volvulus* can live for up to 15 years and are often found encased in fibrous

subcutaneous nodules. Each adult female produces millions of microfilariae that migrate under the skin and to the eyes, producing a variety of dermal and ocular symptoms (see above).

Humans are the only reservoir. Other *Onchocerca* species found in animals cannot infect humans but may occur together with *O. volvulus* in the insect vector.

Incubation period

Microfilariae are found in the skin usually only after 1 year or more from the time of the infective bite. Vectors in Africa can be infective 7 days after an infective bite.

Period of communicability

No direct human-to-human transmission.

Human to blackfly: Blackflies can be infected after biting infected individuals, whose infection may last for 10–15 years after their last exposure to *Simulium* bites, if untreated.

Blackfly to human: Blackfly vectors become infective (i.e. able to transmit infective larvae) 7–12 days after the blood meal.

Reservoirs

Humans. *Onchocerca* species found in animals cannot infect humans but may occur with *O. volvulus* in the insect vector.

Epidemiology

Disease burden

The great majority (99%) of the 37 million people thought to be infected live in 30 countries in sub-Saharan Africa. Apart from the disease burden, onchocerciasis also has important socioeconomic consequences. In the 1960s and early 1970s, fear of blindness led to depopulation of fertile river valleys of the west African savannah, greatly diminishing agricultural production and increasing poverty and famine. The dramatic consequences of onchocerciasis in west Africa led the World Health Organization (WHO), the World Bank, the United Nations Development Programme (UNDP) and the Food and Agriculture Organization of the United Nations (FAO) with the support of the international donor community to launch the Onchocerciasis Control Programme (OCP) in 1974. OCP was officially closed in December 2002 after virtually stopping transmission of the disease in all the participating countries except Sierra Leone, where operations were interrupted by a decade-long civil war.

Although it was once thought that onchocerciasis had been brought under control in Côte d'Ivoire, the socio-political instability that broke out in September 2002 has made it impossible for the national programme to continue ivermectin treatments. The prevalence of onchocerciasis in Côte d'Ivoire ranges from 50% to 60% in the south. An epidemiological assessment carried out in July–September 2007 found that 14% of children under the age of 5 years were infected.

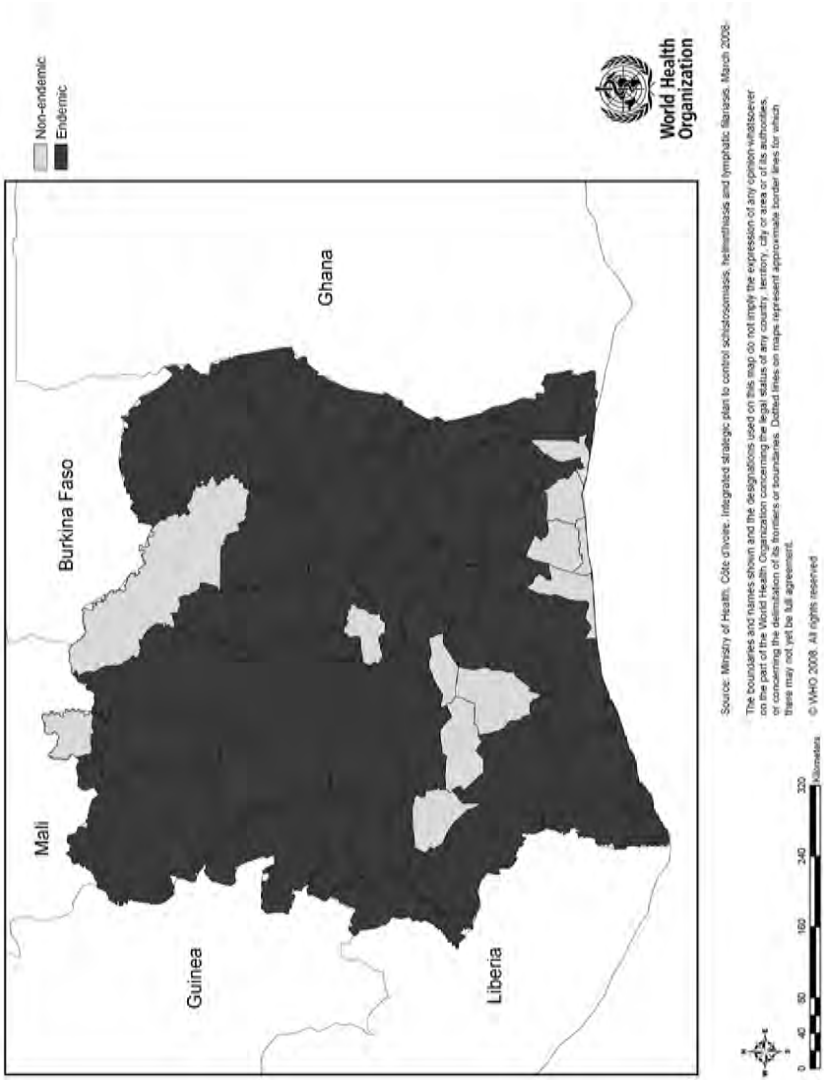


Fig. 10 **Endemicity of onchocerciasis, Côte d'Ivoire, 2007**

Geographical distribution

As a public health problem, the disease is mostly prevalent in west and central Africa, but it is also prevalent in Yemen and six countries in Latin America. It is estimated that some 120 million people are at risk, with 37 million infected throughout Africa. The geographical distribution of onchocerciasis in Côte d'Ivoire is shown in Fig. 10.

Seasonality

Currently not applicable.

Risk factors for increased burden

Population movement

Migration of infected persons into areas previously free of disease may lead to new transmission potential in the presence of infective blackflies.

Overcrowding

Not relevant.

Poor access to health services

Delayed presentation and diagnosis may lead to longer periods of communicability and increases associated morbidity.

Fortunately almost all mapped endemic areas are currently under mass community-directed treatment.

Food shortages

Not relevant.

Lack of safe water, poor hygienic practices and poor sanitation

Secondary bacterial infections may occur.

Prevention and control measures

Case management

Administration of ivermectin once per year over a period of at least 15–20 years will reduce infection to insignificant levels and prevent the appearance of clinical

manifestations. The recommended dose is equivalent to 150 µg/kg body weight (in practice, dosage is according to height, using one to four tablets of 3 mg formulation). Established clinical manifestations are also treated with ivermectin.

Treatment with ivermectin is **contraindicated** in:

- Children under age 5 years, weighing less than 15 kg, or who are less than 90 cm in height;
- Pregnant women;
- Lactating mothers of infants aged less than 1 week;
- Severely ill people;
- Concomitant Loa loa infection due to risk of encephalopathy. (Loa loa co-endemic areas should be treated according to the Mectizan Expert Committee Technical Consultative Committee of the African Programme for Onchocerciasis Control (MEC/TCC) guidelines (*1*).

Prevention

Efforts to prevent resurgence should focus on areas into which migration occurs. The two main strategies for prevention and control of onchocerciasis in Africa are:

- Vector control

  Destruction of S*imulium* larvae by application of insecticides such as temephos (Abate®) through aerial spraying to breeding sites in fast-flowing rivers, in order to interrupt the cycle of disease transmission. Once the cycle has been interrupted for 14–15 years, the reservoir of adult worms dies out in the human population, thus eliminating the source of the disease.

- Community-directed treatment with ivermectin

  This involves the annual administration of ivermectin (150 µg/kg body weight). The introduction of ivermectin in 1987 provided a feasible chemotherapeutic regimen for large-scale treatment of onchocerciasis for the first time. Ivermectin is an effective microfilaricide that greatly reduces the numbers of skin microfilariae for up to a year.

  Loa loa co-infection is a contraindication for the use of ivermectin as it promotes a massive destruction of microfilariae, which may cause an allergic reaction and in individuals with a high burden of Loa loa may cause encephalopathy. It is therefore important to be aware of the geographic distribution of both onchocerciasis and Loa loa. Mapping of the geographical distribution of Loa loa has not been completed in Côte d'Ivoire.

Immunization

None available.

Epidemic control

Recrudescence of transmission may occur and can be managed by administration of ivermectin if mass community-directed treatment programmes maintain good treatment coverage.

References

1. *Recommendations for the treatment of onchocerciasis with Mectizan® in areas co-endemic for onchocerciasis and loiasis. 2004.* http://www.mectizan.org/search/node/treatment)

Further reading

Guidelines for rapid assessment of Loa Loa. Geneva, Special Programme for Research & Training in Tropical Diseases, 2002 (TDR/IDE/RAPLOA/02.1; http://whqlibdoc.who.int/hq/2002/TDR_IDE_RAPLOA_02.1.pdf; accessed August 2009).

African Programme for Onchocerciasis Control (APOC). Geneva, World Health Organization (http://www.who.int/apoc/onchocerciasis/en/; accessed August 2009).

CD-WGE technical focal point: Department of Control of Neglected Tropical Diseases (NTD)

PERTUSSIS (WHOOPING COUGH)

Description

Clinical description

The initial stage – the catarrhal stage – is characterized by coryza (runny nose), sneezing, low-grade fever and a mild occasional cough, similar to the common cold. It has an insidious onset, with an irritating cough that gradually becomes paroxysmal, usually within 1–2 weeks, and lasts for 1–2 months or longer.

The patient has bursts or paroxysms of numerous rapid coughs; followed by a long inspiratory effort usually accompanied by a characteristic whoop. In younger infants, periods of apnoea (cessation in breathing) may follow the coughing spasms, and the patient may become cyanotic (turn blue).

The disease lasts 4–8 weeks. In the convalescent stage, recovery is gradual and 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 the disease.

Complications most commonly include pneumonia. Otitis, haemorrhages (subconjunctival petechiae and epistaxis), convulsions, encephalopathies and death occur more rarely.

Complications are more frequent and severe in younger infants. In developing countries, case-fatality ratios (CFRs) are estimated at 3.7% for children aged less than 1 year and 1% for children aged 1–4 years. Older individuals (i.e. adolescents and adults) and those partially protected by the vaccine may become infected, but usually have milder disease.

Infectious agent

Bordetella pertussis, the pertussis bacillus.

Case definition

Clinically confirmed:

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:

- Paroxysms (i.e. fits) of coughing;
- Inspiratory "whooping";
- Post-tussive vomiting (i.e. vomiting immediately after coughing) without other apparent cause.

Confirmed case:

Laboratory confirmed

A case that meets the clinical case definition and is laboratory-confirmed through

- Isolation of *B. pertussis;* **or**
- Detection of genomic sequences by polymerase chain reaction (PCR); **or**
- Positive paired serology.

Mode of transmission

Primarily by direct contact with discharges from respiratory mucous membranes of infected people via the airborne route.

Incubation period

Usually lasts 7–10 days, rarely more than 14 days.

Period of communicability

Pertussis is highly communicable in the early, catarrhal stage and has a high secondary attack rate in household contacts approaching 90%. Communicability gradually decreases after the onset of the paroxysmal cough. Patients may be contagious for up to 3 weeks after the onset of paroxysmal cough in the absence of treatment, or up to 5 days after onset of treatment.

Reservoirs

Humans are the only hosts.

Epidemiology

Disease burden

According to WHO estimates, in 2003, about 17.6 million cases of pertussis occurred worldwide, 90% of which were in developing countries, and about 279 000 patients died from the disease. WHO has also estimated that, in 2003, global vaccination against pertussis averted about 38.3 million cases and 607 000 deaths.

Outbreaks of *B. pertussis* are common in settings of population displacement, but definitive laboratory evidence is rare. This may be because of the difficulty of obtaining laboratory confirmation from suspected cases. Risk factors for transmission in settings like those found in Côte d'Ivoire include low levels of routine immunization, overcrowding of susceptible groups, malnutrition, and co-infection with other illnesses such as HIV, malaria, tuberculosis etc.

Examples of outbreaks that have occurred in humanitarian settings are:

- **Democratic Republic of the Congo, 2000:** 1136 cases, including 23 (2%) deaths. Vaccination coverage (with the first dose of diphtheria-tetanus-pertussis vaccine, DTP1) of infants aged < 12 months in the affected area was estimated to be 32%. Response activities consisted of case management support with provision of erythromycin, active surveillance and strengthening of routine Expanded Programme on Immunization (EPI) services. A vaccination campaign following the outbreak was not well-accepted by the population, due to fears of secondary effects.
- **Democratic Republic of the Congo, 2001:** 2633 cases, including 17 (0.6%) deaths, detected by active surveillance. Of these cases, 89% were aged ≤ 5 years. Cases were defined as having the characteristic coughing fits, "whooping", and vomiting after coughing for ≤ 2 weeks (suspected case) or longer than 2 weeks (probable case). Suspected cases were treated with erythromycin for 2 weeks. A vaccination campaign in one village targeted children aged 6–72 months and covered 81% of the targeted population.
- **Afghanistan, 2003:** 115 cases, including 17 (14.8%) deaths in an isolated border population with estimated vaccination coverage of < 40%. A 10-day treatment regimen of erythromycin was given to all children (regardless of immunization status, contact with cases, or presence of symptoms) under the age of 15 years in five affected subdistricts, involving 189 villages.
- **The Sudan, 2004:** Number of cases unknown, including 300 deaths, no definitive laboratory diagnosis but clinically diagnosed as pertussis. The affected populations lived in two remote counties not covered by health services. Outbreak-control measures included door-to-door mass treatment of cases and all children and contacts in the affected families with erythromycin.
- **The Sudan, 2005.** 419 cases, including 13 (3.1%) deaths. Response activities included mass treatment of cases and contacts with erythromycin. Routine vaccination of children aged less than 5 years was accelerated in the affected areas.

The CFR for pertussis in industrialized countries is very low (0.1%). However, in developing countries the CFR is 3.9% in infants and 1% in children aged 1–14 years. Severe disease and death is mainly reported in very young non-immune infants. However, in malnourished unvaccinated populations with a high prevalence of co-infections, CFR can reach 15%. Complications, most notably bronchopneumonia, occur most frequently in those aged less than 6 months. The incidence of pertussis-associated encephalopathy is 0.9 per 100 000.

The true disease burden in Côte d'Ivoire is not known. Under-reporting is common; an estimated 1% of cases are reported. As of 10 July 2009, no cases have been reported to WHO since 1998. The number of cases reported in 1995 was 1865; in 1996, 1559; and in 1997, 1284 (*1*).

Low vaccination coverage (less than 80%), increases the risk of outbreaks. Coverage with DTP3 (third dose of diphtheria-tetanus-pertussis vaccine) was 76% in 2007 (72% in 2000, dropping to 50–57% during 2001–2005, increasing again to 77% in 2006). Corresponding coverage in 2007 for AFRO region and the world is estimated at 74% and 81% respectively.

Geographical distribution

Pertussis is endemic worldwide. The specific geographical distribution of pertussis in Côte d'Ivoire is not known but is likely to be higher in areas of poor vaccine coverage (north and west).

Seasonality

In general, pertussis has no distinct seasonal pattern.

Alert threshold

One case is sufficient for an alert and must be investigated, especially if the case occurs in high-risk areas (i.e. with low vaccination coverage).

Risk factors for increased burden

Population movement

Mass population movement facilitates spread of *B. pertussis.*

Overcrowding

Crowded conditions facilitate transmission. The disease is usually introduced into a household by an older sibling or a parent.

Poor access to health services

Especially, poor access to routine immunization services. Susceptibility of non-immunized individuals is universal, and vaccination is the mainstay of pertussis control. Low vaccination coverage is a major risk factor for increased transmission and outbreaks.

Food shortages

Malnutrition increases the severity of illness.

Lack of safe water, poor hygienic practices and poor sanitation

Not relevant.

Prevention and control measures

Case management

The drug of choice for the treatment of pertussis is erythromycin, which should be administered for 7 days to **all cases** and **close contacts** of people with pertussis, regardless of age and vaccination status, and for all those living in households where there is an infant aged less than 1 year. Clarithromycin and azithromycin are also effective.

Drug administration 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 care are important.

Prevention

Immunization is the key to prevention. Administer antibiotics during outbreak management as above.

Immunization

Active primary immunization with the *whole-cell vaccine* (wP) is recommended in association with the administration of vaccines containing diphtheria and tetanus toxoids at age 6, 10 and 14 weeks, according to the national schedule. The efficacy of the vaccine in children who have received at least three doses is estimated to exceed 80%. Protection is greater against severe disease and begins to wane after about 5 years. A fourth dose is not part of the routine schedule but would be beneficial if resources permit.

Although the use of *acellular vaccines* (aP) is less commonly associated with adverse reactions, price considerations affect their use, and wP vaccines are the vaccines of choice for some countries. In general, wP is not given to individuals aged 7 years or older, since local reactions may be increased in older children and adults, and the disease is less severe in older children.

Except for cases in which prior pertussis vaccination resulted in anaphylactic reaction, there are no strict contraindications for this vaccine. All infants, including those who are HIV-positive, should be immunized against pertussis. There are no data to support the perception that previous encephalitis may be a contraindication for pertussis vaccination.

Despite its efficient prevention of clinical disease, the vaccine has a limited impact on the circulation of *B. pertussis,* even in countries with high vaccination coverage. Remaining non-immunized children and older individuals with waning immunity may serve as reservoirs for the infection and transmit *B. pertussis* to non-immunized young infants. Susceptible adolescents and adults allow the occurrence of pertussis outbreaks, although high vaccination coverage may prolong the inter-epidemic intervals.

Epidemic control

The highly contagious nature of the disease leads to large numbers of secondary cases among non-immune contacts. Prophylactic antibiotic treatment (erythromycin) in the early incubation period may prevent disease, but difficulties of early diagnosis, costs involved and concerns related to the occurrence of drug resistance all limit prophylactic treatment to selected individual cases.

Priority must be given to:

- Protecting children aged less than 1 year and pregnant women in the final 3 weeks of pregnancy because of the risk of transmission to the newborn; and
- Stopping infection among household members, particularly if these include children aged less than 1 year and pregnant women in the final 3 weeks of pregnancy.

The strategy relies on chemoprophylaxis of contacts within a maximum of 14 days after 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 commencing

treatment or for up to 3 weeks after onset of paroxysmal cough, or until the end of cough, whichever comes first.

All cases and contacts must have their immunization status verified and brought up to date.

References

1. *Pertussis reported cases.* Geneva, World Health Organization, updated 10 July 2009. (http://www.who.int/Immunization_monitoring/en/globalsummary/timeseries/tsincidenceper.htm, accessed June 2009).

Further reading

Global and regional immunization profile. Geneva, World Health Organization, 2008 (http://www.who.int/vaccines/globalsummary/Immunization/GS_GLOProfile.pdf?CFID=1223068&CFTOKEN=85495284, accessed June 2009).

WHO Vaccine Preventable Diseases Monitoring System. 2008 Global Summary. Immunization Profile – Côte d'Ivoire. Updated 10 July 2009. Geneva, World Health Organization, 2009 (http://www.who.int/vaccines/globalsummary/immunization/countryprofileselect.cfm, accessed August 2009).

CD-WGE technical focal point: Department of Immunization, Vaccines and Biologicals (IVB)

POLIOMYELITIS

Description

Clinical description

Poliomyelitis is a viral infection, which is often recognized by the acute onset of flaccid paralysis, although only less than 1% of the infected susceptible individuals would continue to develop paralysis. Most infections (more than 90%) remain asymptomatic or result in a non-specific febrile illness lasting a few days, corresponding to the **viraemic** phase of the disease. In a few cases, an abrupt onset of **meningitic** and **neuromuscular** symptoms, such as neck stiffness and pain in the limbs follow, associated with fatigue, headache, vomiting and constipation (or, less commonly, diarrhoea).

Flaccid paralysis, when it occurs, is of gradual onset (2–4 days); lower limbs are more commonly affected; and involvement is typically asymmetric, with the weakness being more marked proximally (at the top of the legs). Bulbar (brainstem) paralysis may also occasionally occur, leading to respiratory muscle involvement and death, unless artificial respiration is applied. This accounts for the 2–10% mortality rate associated with paralytic poliomyelitis. Risk factors for paralytic disease include a large inoculum of virus, increasing age, pregnancy, recent tonsillectomy, strenuous exercise and intramuscular injections during the incubation period.

After the acute illness, there is often a degree of recovery of muscle function; 80% of eventual recovery is attained within 6 months, although recovery of muscle function may continue for up to 2 years. After many years of stable neurological impairment, new neuromuscular symptoms (weakness, pain and fatigue, post-polio syndrome) may develop in 25–40% of patients.

Infectious agent

Poliovirus (Enterovirus group): types 1, 2, 3; all can cause paralysis. Type 1 is isolated from paralytic cases most often. Type 1 most frequently causes epidemics.

Case definition

Suspected case:

Acute flaccid paralysis (AFP) in a child aged less than 15 years, including Guillain–Barré syndrome;* **or**

Any paralytic illness in a person of any age when polio is suspected.

* For practical reasons, Guillain–Barré syndrome is considered as poliomyelitis until proven otherwise

Confirmed case:

AFP with laboratory-confirmed wild poliovirus in stool sample.

Polio-compatible: AFP clinically compatible with poliomyelitis, but without adequate virological investigation.

Mode of transmission

Poliovirus is highly communicable. Transmission is primarily via the faecal–oral route.

Incubation period

Commonly 7–14 days for paralytic cases; but the reported range is 3 to possibly 35 days.

Period of communicability

Virus is demonstrable in throat secretions as early as 36 hours and in faeces 72 hours after exposure to infection; virus persists in throat for 1 week and in faeces for 3–6 weeks. Cases are most infectious during the days before and after the onset of symptoms.

Reservoirs

Humans, most frequently people with asymptomatic (subclinical) infections, especially children.

Epidemiology

Disease burden

Although Côte d'Ivoire was polio-free in July 2000, an outbreak followed an importation of wild poliovirus in December 2003, and culminated in 17 cases in 2004 (Table 19), largely in the northern and western regions. Côte d'Ivoire then remained polio-free until 2008, when 38 cases were confirmed in previously polio-free countries of west Africa, including Côte d'Ivoire as well as Benin, Burkina Faso, Ghana, Mali, Niger and Togo and after several importations of virus from Nigeria (data as of 2 March 2009). The proximity of Nigeria, where polio remains

endemic, puts Côte d'Ivoire at constant risk. In response to this threat, a series of cross-border synchronized rounds of polio vaccination will be carried out in this belt of countries with importations of poliovirus of Nigerian origin, including Côte d'Ivoire.

Table 19. **Reported cases of poliomyelitis, Côte d'Ivoire, 2003–2008**

Year	Reported cases of acute flaccid paralysis	Cases of acute flaccid paralysis with adequate specimens (%)	Total confirmed cases of poliomyelitis	Wild-virus confirmed cases of poliomyelitis
2008	235	93	1	1
2007	258	91	0	0
2006	309	96	0	0
2005	290	79	0	0
2004	161	88	17	17
2003	116	98	1	1

Source: *Wild Poliovirus Weekly Update*. Updated 10 July 2009. Global Polio Eradication Initiative (http://www.polioeradication.org/casecount.asp, accessed August 2009).

Today, the disease has been eliminated from most of the world, and only four countries worldwide remain polio-endemic. This represents the lowest number of endemic countries with circulating wild poliovirus. At the same time, the areas of transmission are more concentrated than ever – 98% of all cases in the world are found in India, Nigeria and Pakistan. However, all countries will remain at risk of importations, regardless of their geographical proximity to polio-endemic countries. It is therefore important that certification-standard surveillance is widely maintained in order to avoid late detection of any importation of wild poliovirus.

- All countries are required to conduct a number of activities reaching previously non-immunized children and gaining access to all areas, including those that are inaccessible as a result of conflict;
- Sustaining high-quality surveillance for acute flaccid paralysis in order to detect any importation in a timely fashion and to improve targeting of supplementary immunization activities;
- Improving basic infrastructure for the EPI.

Geographical distribution

Substantial progress has been made towards eradicating poliomyelitis from the region. However, outbreaks of polio after importation of wild poliovirus into polio-free areas and ongoing transmission of wild poliovirus in endemic areas pose a constant risk to the achievement of polio eradication globally, the success of which depends largely on progress in Afghanistan, India, Nigeria and Pakistan. The longer it takes to interrupt transmission in these countries, the greater the danger of wild poliovirus being exported to areas in the region.

The last cases of polio reported in Côte d'Ivoire occurred in the northern and western regions of the country.

Seasonality

Increased transmission during the rainy season.

Alert threshold

Any case of AFP must be notified and investigated.

Risk factors for increased burden

Population movement

Facilitates transmission from infected to non-immune populations.

Overcrowding

Important in promoting transmission.

Poor access to health services

Limited access to routine immunization services will lead to low vaccination coverage (< 80%) for routine EPI, thereby increasing the proportion of susceptible persons and the risk of outbreaks.

Risk of undetected circulation of poliovirus .

Food shortages

Not relevant.

Lack of safe water, poor hygienic practices and poor sanitation

As polio is spread by the faecal–oral route, lack of water, poor hygiene and sanitation enhance the risk of transmission.

Prevention and control measures

Case management

Management of the acute phase of paralytic poliomyelitis is supportive and symptomatic:

- Close monitoring of respiration; respiratory support in case of respiratory failure or pooling of pharyngeal secretions;
- Moist hot-packs for muscle pain and spasms;
- Passive physical therapy to stimulate muscles and prevent contractures;
- Anti-spasmodic drugs; and
- Frequent turning to prevent bedsores.

If hospitalization is required, the patient should be isolated, particularly avoiding contact with children.

Safe disposal of discharge and faeces, disinfection of any soiled articles and immediate reporting of further cases are essential.

Prevention

Immunization is the key to prevention.

Immunization

The routine immunization schedule in Côte d'Ivoire includes oral poliovirus vaccine (OPV) given at birth, and at age 6, 10 and 14 weeks. This oral vaccine is based on live attenuated strains of all three virus types and is easily administered, induces a good humoral (antibody) and mucosal (intestinal) immune response and is considerably cheaper than the injectable inactivated poliovirus vaccine (IPV).

In endemic countries, WHO recommends the use of national supplementary immunization campaigns administering two doses of OPV, 1 month apart, to all children aged less than 5 years, regardless of previous immunization history (preferably in the cooler, dry season). Targeted mop-up campaigns use the same strategy. In camp settings, all children aged 0–59 months should be vaccinated on arrival.

Vaccine-related paralytic poliomyelitis, which can occur among vaccine recipients or their healthy contacts, is extremely rare, affecting approximately 1 in every 2.5 million doses administered.

Epidemic control

Every country should have standard operating procedures in place to mount rapid mop-up campaigns upon confirmation of a polio case. Such plans are also a prerequisite for polio-free certification.

In case of suspected outbreak, undertake:

Investigation

- Clinical and epidemiological investigation
- Rapid virological investigation (two stool samples within 14 days of onset of symptoms must be sent to a WHO-accredited laboratory).
- Outbreak confirmation will be based on the isolation of wild poliovirus from a stool specimen obtained from an AFP case.

Intervention

- House-to-house mop-up campaigns with OPV covering a wide geographical area (at least province involved and relevant neighbours) should be conducted within 4 weeks after confirmation of the wild poliovirus case. Mop-up campaigns target a minimum of 500 000–1 million children.
- If national immunization days (NIDs) or sub-national immunization days (SNIDs) are already planned, a major focus on quality of supplementary immunization activities (SIA) should be made for the area of the outbreak and adjacent districts.
- Surveillance should be enhanced through intensive monitoring of all reporting units to ensure active surveillance and reporting of zero cases, extensive retrospective record reviews and active case-finding in surrounding areas.

CD-WGE technical focal point: Polio Eradication Initiative (POL)

RABIES

Description

Clinical description

Paresis or paralysis, delirium, convulsions

Without medical attention, death in about 6 days, usually due to respiratory paralysis

Infectious agent

Rabies virus, a Rhabdovirus of the genus *Lyssavirus*.

Case definition

Human rabies

Suspected case:

An acute neurological syndrome (encephalitis) dominated by forms of hyper-activity (furious rabies) or paralytic syndrome (dumb rabies) which progresses towards coma, respiratory failure and death, within 7–10 days after the first symptom. Bites or scratches from an animal suspected to have rabies can usually be traced in the patient's medical history.

Probable case:

A suspected case plus history of contact with a suspected rabid animal.

Confirmed case:

A suspected case that is laboratory-confirmed by **one or more** of the following:

- Detection of rabies viral antigens by direct fluorescent antibody (FA) or by enzyme immunoassay (EIA) in clinical specimens, preferably brain tissue (collected post mortem);
- Detection by FA test on skin biopsy (collected ante mortem);
- FA-positive result after inoculation of brain tissue, saliva or cerebrospinal fluid (CSF) in cell culture, or after intracerebral inoculation in mice or in suckling mice;
- Detectable rabies-neutralizing antibody titre in the serum or the CSF of an unvaccinated person;

- Detection of viral nucleic acids by polymerase chain reaction (PCR) on tissue collected post mortem or in a clinical specimen (brain tissue or skin, cornea, urine or saliva).

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

Usually by the bite of an infected mammalian species (e.g. dog, cat, fox, bat): bites or scratches introduce virus-laden saliva into the human body.

No human-to-human transmission has been documented.

Incubation period

Usually ranges from 2 to 10 days but may be as long as 7 years.

Period of communicability

In dogs and cats, the disease is usually communicable 3–7 days before the onset of clinical signs (rarely more than 4 days) and throughout the course of the disease. Longer periods of excretion before the onset of clinical signs have been observed in other animal species.

Reservoirs

In Africa, dogs are the main hosts and are responsible for most of the deaths caused by rabies in humans worldwide.

Epidemiology

Disease burden

In the WHO African and South-East Asia regions, human mortality from endemic canine rabies has been estimated to be 55 000 per year, with 44% of these deaths

occurring in Africa. An estimated 10 million people receive post-exposure treatments each year after being exposed to animals suspected to have rabies. The number of deaths officially reported in most developing countries greatly underestimates the true incidence of the disease. Although effective and economical control measures are available, the disease has not been brought under control throughout most of the affected countries. The application of effective and economical control measures is hampered by a range of economic, social and political factors. Lack of accurate data on the true public-health impact of the disease is a major factor contributing to low commitment to rabies control.

Risk of cases in humans is significant if individual cases or outbreaks of rabies are reported in dogs or other susceptible animals in the same zone. Children aged 5–15 years are the group at most risk.

Côte d'Ivoire is considered to be endemic for rabies. The actual burden of rabies is difficult to quantify as the group most affected are children in rural settings where there is a lack of knowledge of the disease, hence most do not present to health services and die at home. Reported cases by year (Table 20) are probably a gross under-representation of the situation in Côte d'Ivoire.

Table 20. **Incidence of human rabies in Côte d'Ivoire, 2002–2006**

Year	Annual number of reported cases
2006	1
2005	1
2004	0
2003	-
2002	1

Source: *Rabies in humans*. Geneva, World Health Organization (http://www.who.int/rabies/human/en/index.html, accessed May 2009).

- not available

Geographical distribution

The majority of deaths attributable to rabies in Africa and Asia occur in rural areas.

Seasonality

No seasonality has been reported.

Alert threshold

One case in a susceptible animal species and/or human must lead to an alert.

Epidemics

Rabies is a sporadic illness in humans. No data are available on outbreaks in Côte d'Ivoire.

Risk factors for increased burden

Population movement

Movement of populations into areas with high numbers of rabies-infected dogs may increase the risk of exposure.

Overcrowding

In overcrowded settings, an infected animal has the opportunity to bite more people; the density of the dog population parallels that of the human population.

Poor access to health services

Prompt administration of vaccine after exposure (plus immunoglobulin if heavy exposure) is the only way to prevent the death of an infected person.

Food shortages

Not relevant.

Lack of safe water, poor hygienic practices and poor sanitation

Lack of safe water and poor hygiene result in poor wound care; thorough cleansing of the wound with soap and water, detergent or iodine after an animal bite can minimize the risk of infection with rabies.

Prevention and control measures

Case management

There is no specific treatment for rabies once symptoms have started. Rabies is an almost invariably fatal disease.

The most effective way to prevent rabies is to wash and flush the wound or point of contact with soap and water or detergent, and then apply ethanol or tincture or aqueous solution of iodine.

When caring for patients with rabies, universal barrier nursing practices need to be applied. Health authorities should be notified.

Table 21 summarizes the recommended treatment strategies according to type of contact with animal suspected to have rabies. Anti-rabies vaccine should be given as soon as possible for category-II and -III exposures, according to WHO-recognized regimens.

Table 21. **Recommended treatments for human rabies according to type of contact with animal suspected to have rabies**

Category of exposure	Type of contact with a suspected or confirmed rabid domestic or wild animal, or animal unavailable for testing	Type of exposure	Recommended treatment
I	Touching or feeding of animals Licks on intact skin	None	None, if reliable case history is available
II	Nibbling of uncovered skin Minor scratches or abrasions without bleeding	Minor	Administer vaccine immediately Stop treatment if animal remains healthy throughout an observation period of 10 days or if animal is proven to be negative for rabies by a reliable laboratory using appropriate diagnostic techniques
III	Single or multiple transdermal bites or scratches, licks on broken skin Contamination of mucous membrane with saliva (i.e. licks) Exposures to bats	Severe	Administer rabies immunoglobulin and vaccine immediately. Stop treatment if animal remains healthy throughout an observation period of 10 days or if animal is found to be negative for rabies using appropriate diagnostic techniques

Source: *Current WHO guide for rabies pre- and post-exposure prophylaxis in humans.* Geneva, World Health Organization (http://www.who.int/rabies/PEProphylaxisguideline.pdf, accessed May 2009).

Prevention

- Post-exposure treatment immunization and, when appropriate, anti-rabies immunoglobulin;
- Pre-exposure immunization;
- Increased availability of safe and effective rabies vaccine; and
- Elimination of dog rabies through mass vaccination of dogs, and dog-population management.

Immunization

Post-exposure immunization

- *Intramuscular administration*: 0.5 ml to 1.0 ml should be given on post-exposure days 0, 3, 7, 14 and 28 into the deltoid region (or anterolateral thigh in small children). The abbreviated multi-site schedule is acceptable: one dose into the left arm and another into the right arm on day 0, then one dose on days 7 and 21.
- *Intradermal administration:* 0.1 ml at eight sites on post-exposure day 0, at four sites on day 7, and one site on days 30 and 90 or two sites on day 30. An alternative regimen consists of 0.1 ml at two sites on days 0, 3, 7 and 28.

Anti-rabies immunoglobulin should be applied for category III (severe) exposures only. In cases where immunoglobulin is necessary, immunoglobulin should be infiltrated into the depths of the wound and around the wound as anatomically feasible. Any remaining immunoglobulin should be injected at an intramuscular site distal from that of vaccine inoculation. Suturing of the wound may have to be delayed. Anti-tetanus treatment and antimicrobials may be necessary for prevention of other bacterial infections.

Pre-exposure immunization

- *Intramuscular administration:* 1.0 ml intramuscular rabies vaccine given on days 0, 7 and 28 (may be advanced to day 21 if time is limited).
- *Intradermal administration:* 0.1 ml on days 0, 7, and 28 or advanced as for intramuscular administration.

Vaccination should be considered in individuals at high risk (e.g. veterinarians, wildlife-conservation personnel, laboratory workers working with rabies).

Mass preventive vaccination in humans is generally not recommended, but can be considered under certain circumstances for the age group 5–15 years.

Epidemic control

Immediate notification if one or more suspected cases are identified. Confirm the outbreak, following WHO guidelines (Annex 2). Confirm diagnosis and ensure prompt management.

CD-WGE technical focal point: Department of Control of Neglected Tropical Diseases (NTD)

SCHISTOSOMIASIS

Description

Clinical description

The disease manifestations associated with schistosomiasis are dependent on the host response to larvae and eggs. In people who have been exposed previously, a protective response is directed against the schistosomula after cercarial penetration of the skin, thus killing most organisms and inducing a papular dermatitis.

Acute schistosomiasis, which occurs in primary infections and just before or at the time of egg deposition, may present as high fever, chills, headache, muscle pain, abdominal pain, diarrhoea and occasional bloody stools. Chronic schistosomiasis may occur months to years after initial infection and may be asymptomatic. Patients may have indirect symptoms, such as chronic malnutrition, anaemia and impaired cognitive function.

Schistosoma haematobium results in haematuria (blood in urine) and dysuria (difficulty in passing urine), granulomas, hydronephrosis and eventual renal impairment. Heavy infections with *Schistosoma mansoni* may lead to colicky abdominal pain with intermittent diarrhoea and fatigue. Hepatosplenomegaly with portal hypertension occurs with chronic infection. Rare cases of pulmonary and central nervous system infections have occurred.

Concurrent illness such as *Salmonella* bacteraemia, HIV, hepatitis B or C co-infection affects the clinical course of schistosomiasis disease.

Infectious agent

Helminths: *Schistosoma haematobium* (agent of urinary schistosomiasis); *Schistosoma mansoni* (agent of intestinal schistosomiasis). All are blood fluke-worms belonging to the class Trematoda.

Intestinal schistosomiasis due to *Schistosoma intercalatum* may be endemic in the tropical rain-forest areas of the subregion.

Case definition

Urinary schistosomiasis

Endemic areas (moderate or high prevalence)

Suspected case: Not applicable.

Probable case: Not applicable.

Confirmed case: A person with:

- visible haematuria; **or**
- positive reagent strip for haematuria; **or**
- *S. haematobium* eggs in urine (microscopy).

Non-endemic areas and areas of low prevalence

Suspected case: A person with:

- Visible haematuria; **or**
- Positive reagent strip for haematuria; **and**
- Possible contact with infective water.

Probable case: Not applicable.

Confirmed case: A person with *S. haematobium* eggs in urine (microscopy).

Intestinal schistosomiasis

Endemic areas (moderate or high prevalence)

Suspected case: A person with nonspecific abdominal symptoms, blood in stool, hepato(spleno)megaly.

Probable case: A person who meets the criteria for presumptive treatment, according to the locally-applicable diagnostic algorithms.

Confirmed case: A person with eggs of *S. mansoni* in stools (microscopy)

Non-endemic areas and areas of low prevalence

Suspected case: A person with nonspecific abdominal symptoms, blood in stool, hepatosplenomegaly **and** possible contact with infective water

Probable case: Not applicable

Confirmed case: A person with eggs of *S. mansoni* in stools (microscopy)

Mode of transmission

Water-borne disease. Patients with schistosomiasis discharge the schistosome eggs in their urine (*S. haematobium*) or faeces (*S. mansoni*, occasionally *S. haematobium*). When the eggs reach a body of fresh water, they liberate first-stage larvae (miracidia) that penetrate suitable snail hosts (*Bulinus* spp. and *Biomphalaria* spp.) and develop

into final-stage larvae (cercariae). The cercariae emerge from the snail and penetrate human skin, usually while the person is swimming, working or wading in water (mainly among people engaged in agriculture and fishing).

Bulinus snails live mainly along the grassy riverbanks away from the main current and the *Biomphalaria* snails more particularly in the swampy lateral pools.

Transmission is focal in endemic areas and most intense in poor rural areas with inadequate sanitation and water supplies. Schistosomiasis also occurs in urban areas.

Incubation period

In primary infections:

Within 4 days: localized dermatitis at the site of cercarial penetration.

Within 2–8 weeks: acute febrile reaction (Katayama fever; uncommon).

From 3 months to several years: manifestations of chronic illness.

Period of communicability

As long as eggs are discharged by patients. This may be from 10 to 12 weeks to more than 10 years after infection. Any infected (acute and chronic) patient may discharge eggs via faeces or urine thereby contaminating water sources. In endemic areas, most persons have a low worm burden with only a small proportion (usually children aged 5–14 years) having heavy infections. No human-to-human spread.

Reservoirs

There are two snail hosts for *S. haematobium* in Côte d'Ivoire – *Bulinus globosus* and *Bulinus truncates*. The former has been found at Buona in the north-east, at Boundiali and Odienne in the north-west, at Danané in the Ouest region, at Kossou in the central region, at San Pedro in the south-west and at Adzopé in the south-east. The latter has been found mainly in the central region, the north region and the north-east (least humid regions).

Biomphalaria pfeifferi is the only snail host for *S. mansoni* in the country and can be found throughout, but its density is lower than that of the two *Bulinus* species, particularly in the zones with an annual rainfall of less than 1500 mm.

S. mansoni is also found in rodents and primates, but human beings are the main host.

Epidemiology

Disease burden

An estimated 200 million people in 74 countries have schistosomiasis, 85% of these people live in sub-Saharan Africa. WHO estimates that the annual number of deaths caused by schistosomiasis could be as high as 200 000.

The most intense transmission occurs where populations concentrate around water sources. Such patterns of disease may be mirrored and amplified among internally displaced persons and refugee populations who also congregate around water sources, as demonstrated in 2007 when infected Somali populations moved to Kenya.

As schistosomiasis may be co-endemic with soil-transmitted helminthiasis, coordinated treatment for both may be appropriate. The mapping of pathogen distribution is therefore vital.

S. haematobium and *S. mansoni* are endemic in Côte d'Ivoire, with the former being more common. There is no available data on current rates but small parasitological surveys in selected areas of Côte d'Ivoire suggest that about one third of school-aged children are infected with schistosomiasis eggs. Higher prevalence is seen along major waterways, dams and irrigated rice-cultivation zones.

Geographical distribution

Figures 11 and 12 show the geographical distribution of *S. haematobium* and *S. mansoni* in Côte d'Ivoire in 2000. Since then, there have been major water resources schemes leading to increased schistosomiasis transmission, and insecurity may have displaced infected populations into areas where transmission could be established.

In general, cases tend to occur:

- Along major river valleys;
- In settlements in irrigated areas;
- In recurrent lake formations due to seasonal changes;
- In settlements near perennial or semi-permanent ponds or lakes;
- Around water-resource development schemes.

Fig. 11 **Prevalence of schistosomiasis (*S. haematobium*) infection, by district, Côte d'Ivoire, 1995–2000**

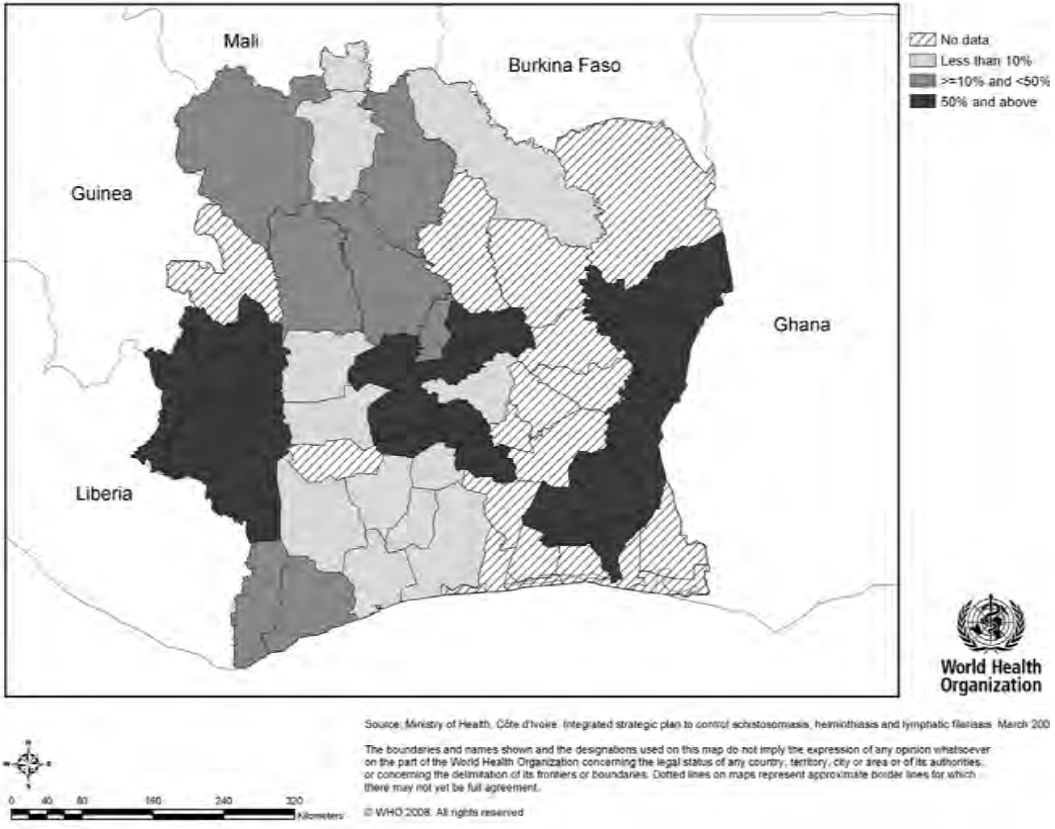
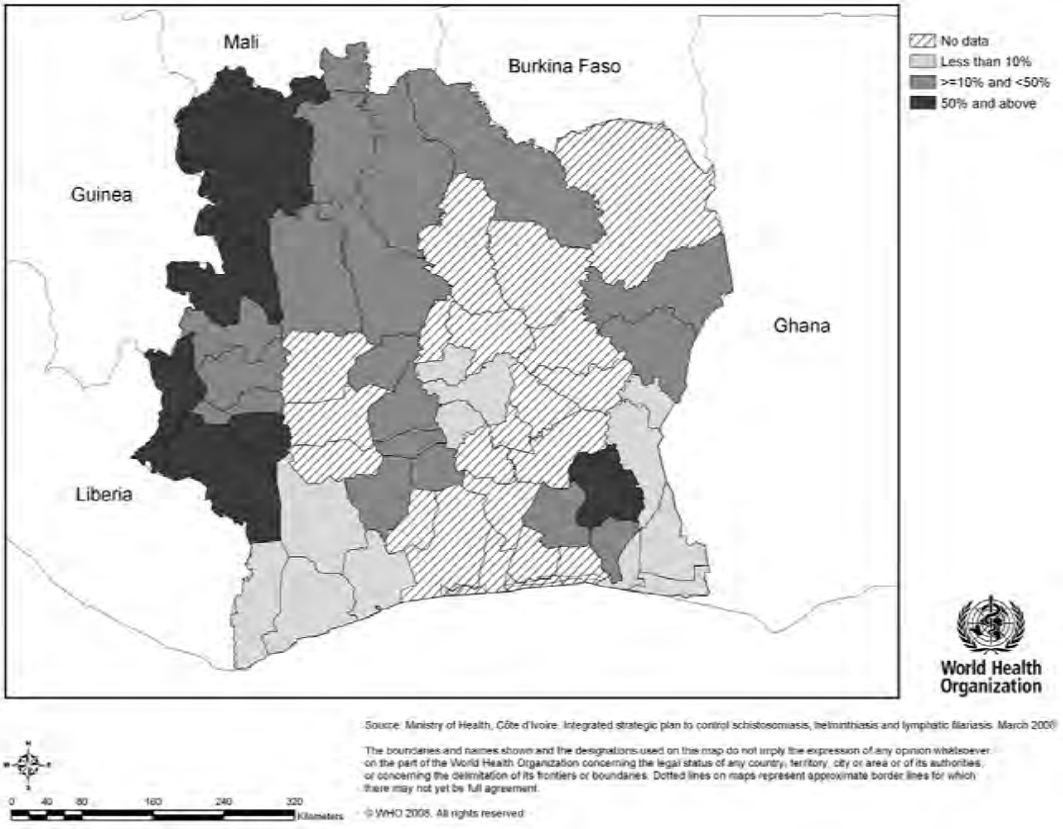


Fig. 12 **Prevalence of schistosomiasis (*S. mansoni*), by district, Côte d'Ivoire, 1995–2000**



Seasonality

Overall, transmission of schistosomiasis occurs throughout the year. Transmission in semi-permanent ponds or lakes is linked to the rainy season. In certain conditions, dry periods tend to increase transmission of the disease as a result of higher cercarial densities in bodies of water and of drying of wells, with consequent increased use of infested water.

Epidemics

May potentially occur after repeated flooding. Changes in ecology, such as the building of dams, lead to marked increase in the prevalence of schistosomiasis.

Risk factors for increased burden

Population movement

Can lead to the introduction of *S. mansoni* and/*or S. haematobium* in areas previously free or endemic for only one of the species.

Overcrowding

Higher human population densities lead to increased discharge of schistosome eggs in water bodies, increasing the risk of transmission.

Poor access to health services

Regular preventive treatment (preventive chemotherapy) and treatment of cases has proved effective in reducing egg discharge, thus limiting introduction of *Schistosoma* spp. into schistosome-free areas. Reduced egg discharge also prevents late-stage complications of schistosomiasis in infected individuals.

Food shortages

Malnutrition and schistosomiasis have a synergic role in causing iron-deficiency anaemia.

Lack of safe water, poor hygienic practices and poor sanitation

Contamination of water by urination/defecation and use of surface water infested by cercariae are essential for the transmission of schistosomiasis.

Prevention and control measures

Case management

Praziquantel is the drug of choice against all schistosome parasites. A single oral dose of 40 mg/kg is generally sufficient to produce cure rates of between 80% and 90% and dramatic reductions in the average number of eggs excreted. Praziquantel treatment for one person requires, on average, three tablets of 600 mg in one dose. A dose pole (for calculating dosage according to height) is available to facilitate the delivery of praziquantel in schools or in community-based programmes.

The cost of a tablet (as of September 2008) is now less than US$ 0.10, bringing the total drug cost of treatment to about US$ 0.30. Drug costs decrease when the entire population is included in prevention programmes.

Prevention

Control of helminths occurs as coordinated mass drug administration, generally for children and women of childbearing age. See Table 22 for the recommended treatment strategy.

In addition to preventive chemotherapy, other recommended interventions are:

- Community diagnosis (through primary school surveys) and regular treatment of endemic populations 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 and proper disposal of excreta.
- Reduction of snail habitats and snail contact (through irrigation and agricultural practices, and environmental management).
- Treatment of snail-breeding sites with molluscicide.

Table 22. **Recommended treatment strategy for preventive chemotherapy of schistosomiasis**

Community category	Prevalence among school-age children	Action to be taken	
High-risk community	≥ 50% by parasitological methods (intestinal and urinary tract schistosomiasis) or ≥ 30% by questionnaire for visible haematuria (urinary schistosomiasis)	Treat all school-age children (enrolled and not enrolled) once per year	Also treat adults considered to be at risk (from special groups to entire communities living in endemic areas)
Moderate-risk community	≥ 10% but < 50% by parasitological methods (intestinal and urinary schistosomiasis) or < 30% by questionnaire for visible haematuria (urinary schistosomiasis)	Treat all school-age children (enrolled and not enrolled) once every 2 years	Also treat adults considered to be at risk (special risk groups only)
Low-risk community	< 10% by parasitological methods (intestinal and urinary schistosomiasis)	Treat all school-age children (enrolled and not enrolled) twice during their primary schooling age (e.g. once on entry and once on exit)	Praziquantel should be available in dispensaries and clinics for treatment of suspected cases.

Source: *Preventive chemotherapy in human helminthiasis: coordinated use of anthelminthic drugs in control interventions. A manual for health professionals and programme managers*. Geneva, World Health Organization, 2006.

Immunization

Not available.

Epidemic control

Examine for schistosomiasis and treat all who are infected (particularly those with disease), those who are heavily infected and children. Prohibit contamination of water and provide sources of clean water. Educate. Control snail population in areas of high density.

Further reading

For documents and publications on schistosomiasis, see the WHO site for schistosomiasis, soil transmitted helminths, and the Partners for Parasite Control (PPC) (http://www.who.int/worm control/en/, accessed June 2009).

CD-WGE technical focal point: Department of Control of Neglected Tropical Diseases (NTD)

SOIL-TRANSMITTED HELMINTHIASES: ASCARIASIS, HOOKWORM INFECTION, AND TRICHURIASIS

Description

Clinical description

Soil-transmitted helminth (STH) infections are usually asymptomatic. Overt infection may produce a wide range of symptoms, including intestinal manifestations (diarrhoea, abdominal pain), general malaise and weakness, which may affect working and learning capacities and impair physical growth. All STHs compete with the host for nutrients, causing malabsorption of fats, proteins, carbohydrates and vitamins, and directly contributing to malnutrition. Among infants and children, they can cause growth retardation.

Ascaris infection exacerbates vitamin A deficiency; elimination of ascarids may result in rapid clinical improvement in night blindness and dryness around the eye.

Persons acquiring **hookworm** infections may develop an itchy maculo-papular rash at the site of larval penetration. Transient pneumonitis, epigastric pain, anorexia, diarrhoea and eosinophilia may occur, complicated by iron-deficiency anaemia caused by chronic intestinal blood loss.

Heavy ***Trichuris*** infection may cause diarrhoea and severe malabsorption. In endemic regions, typically only 10% have heavy worm burdens and tend to be the ones who suffer from the disease. Those with a heavy burden of worms have a friable intestinal mucosa leading to tenesmus and bloody mucoid stools. Recurrent rectal prolapse, iron-deficiency anaemia, malnutrition, and growth retardation may be seen.

Infectious agent

The four major STHs are:

- The roundworm *Ascaris lumbricoides* (white or pinkish adult worm measuring 15–30 cm in length).
- Hookworms – *Ancyclostoma duodenale and Necator americanus* (small, cylindrical, grayish-white nematodes measuring 7–13 mm in length).
- The whipworm *Trichuris trichiura* (pinkish-grey adult worm, measuring 4 cm in length).

Case definition

Ascariasis

Suspected case: Abdominal (mild abdominal discomfort, dyspepsia, loss of appetite, nausea, malnutrition) or respiratory symptoms (non-productive cough, chest discomfort, eosinophilic pneumonitis) **and** history of passing worms.

Confirmed case: Suspected case **and** passage of *A. lumbricoides* (anus, mouth and nose), **or** presence of *A. lumbricoides* eggs in stools (microscopic examination).

Hookworm infection (*Ancyclostoma duodenale* and *Necator americanus*)

Suspected case: Severe anaemia for which there is no other obvious cause.

Confirmed case: Suspected case **and** presence of hookworm eggs in stools (microscopic examination).

Trichuriasis

Suspected case: bloody and mucoid stools.

Confirmed case: Suspected case **and** presence of *T. trichiura* eggs in stools.

Mode of transmission

Ingestion of eggs, mainly as a contaminant of food: *A. lumbricoides* and *T. trichiura*.

Active penetration of skin by larvae in the soil: hookworms.

Incubation period

4–8 weeks for *A. lumbricoides*.

From a few weeks to many months for hookworm disease.

From a few weeks to 3 months for *T. trichiura*.

Period of communicability

A. lumbricoides eggs appear in the faeces 45–75 days after ingestion and become infective in soil after 14–21 days. *Ascaris* eggs can remain viable for up to 6 years in moist loose soil and can survive freezing winter temperatures and short periods of desiccation. 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).

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 (3–5 years for *Necator*, and about 1 year for *Ancyclostoma*).

T. trichiura 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 1–3 years.

Reservoirs

Humans.

Epidemiology

Disease burden

STH infections are among the most prevalent infections in the world and a leading cause of morbidity, particularly in the developing world. Because STH-induced blood loss (especially in hookworm infections) as well as iron and protein deficiencies are typically insidious and chronic and do not usually result in death, until recently STHs were often overlooked as significant causes of morbidity. Recent estimates suggest that *A. lumbricoides* infects over 1 billion people, *T. trichiura* 795 million, and hookworms (*Ancylostoma duodenale* and *Necator americanus*) 740 million. The greatest numbers of STH infections occur in sub-Saharan Africa, the Americas, east Asia and China (*1*). STHs cause malnutrition, anaemia, growth retardation and increased susceptibility to other infections, thereby reducing work capacity.

Available data show that the prevalence of hookworm in endemic communities of Côte d'Ivoire can be as high as 100%, that of ascariasis as high as 71.6%, while that of *Trichuris* can reach 24%.

Geographical distribution

Occurs worldwide in developing countries with poor sanitation.

Ascaris and *Trichuris* occur in both urban environments, especially urban slums, and in rural areas. In some instances, the prevalence of *Ascaris* infection is actually greater in urban environments. In contrast, high rates of hookworm infection are typically restricted to areas where rural poverty predominates.

Only scarce data are available on the prevalence and intensity of STH infections in Côte d'Ivoire; however, they are known to be widespread throughout the entire national territory.

Seasonality

Distribution is influenced by environmental parameters, especially temperature, humidity and soil dryness, factors that influence the survival of eggs and larvae in the environment. Transmission is most intense immediately after rainy seasons and is lowest during prolonged dry seasons.

Risk factors for increased burden

Population movement

Population displacement for extended periods leading to living conditions with unsatisfactory sanitary facilities increases the risk of infection. Populations must remain in the same place for long enough for eggs to be discharged by an infected person and to become infective themselves.

Overcrowding

Overcrowding may facilitate transmission as a result of poor sanitation and poor hygienic practices, related to the number of people defecating in one area and unsafe disposal of faeces.

Poor access to health services

Treatment of entire endemic communities at regular intervals has a limited effect on breaking the transmission cycle and reducing overall transmission rates; however, such action has been shown to be highly effective in preventing development of morbidity due to STH infections in infected individuals (preventive chemotherapy).

Food shortages

May lead to consumption of unsafe food and water, increasing the risk of infection. Malnutrition and STH infections have a synergistic role in causing iron-deficiency anaemia and vitamin A deficiency.

Lack of safe water, poor hygienic practices and poor sanitation

The proportion of people using sanitation facilities effectively is the most important factor.

Prevention and control measures

Case management

STH infections can be controlled with very cheap interventions. The average cost of treatment during a school-based drug distribution campaign (including drugs, distribution and monitoring activities) is approximately US$ 0.10–0.15 per child.

For treatment, the following four drugs are recommended by WHO and can be safely administered to children after the first year of life:

- Albendazole, 400 mg single dose (200 mg in children aged 1–2 years); **or**
- Mebendazole, 500 mg single dose; **or**
- Levamisole, 2.5 mg/kg single dose; **or**
- Pyrantel, 10 mg/kg single dose.

Levamisole and pyrantel are less commonly used because they are less easy to administer in large-scale drug-distribution interventions.

The following points are also to be noted regarding these four drugs:

- These drugs must not be given during the first trimester of pregnancy;
- Where mass treatment with albendazole for lymphatic filariasis is envisaged, such albendazole will also treat STH infections;
- Iron supplementation is also recommended in communities with high prevalence of iron-deficiency anaemia (such as those where hookworm disease is highly endemic).

Occasionally, surgery may be required for intestinal obstruction or perforation.

Prevention

Adequate sanitation is the key to prevention.

Surveys will identify areas of particularly high endemicity where preventive chemotherapy interventions (large-scale distribution of anthelminthic drugs without individual diagnosis) will be warranted. In areas where STH infections are co-endemic with schistosomiasis and lymphatic filariasis, coordinated implementation of preventive chemotherapy interventions against all the diseases is appropriate.

Control of STH infections can play a major role in reducing the burden of communicable diseases among emergency-affected and displaced populations. Moreover,

given its simplicity, STH control can represent a starting point for the reconstruction of health-care systems in countries affected by emergencies.

A recommended treatment strategy is given in Table 23. Implementing and sustaining universal treatment twice annually is recommended for high-risk communities. Once-per-year treatment is sufficient in low-risk communities. Health education regarding safety of food and water, and proper sanitation is important.

Table 23. **Recommended treatment strategy for preventive chemotherapy[a] of soil-transmitted helminthiasis**

Community category	Prevalence of any STH infection among school-age children	Action to be taken[a]	
High-risk community	≥ 50%	Treat all school-age children (enrolled and not enrolled) twice each year[b]	Also treat: ■ preschool children ■ women of childbearing age, including pregnant women in the second & third trimesters and lactating mothers ■ adults at high risk in certain occupations (e.g. tea-pickers and miners)
Low-risk community	≥ 20% and < 50%	Treat all school-age children (enrolled and not enrolled) once each year	Also treat: ■ preschool children ■ women of childbearing age, including pregnant women in the second & third trimesters and lactating mothers ■ adults at high risk in certain occupations (e.g. tea-pickers and miners)

Source: *Preventive chemotherapy in human helminthiasis*. Geneva, World Health Organization, 2006 (http://www.who.int/neglected_diseases/preventive_chemotherapy/pct_manual/en/index.html; accessed August 2009).

[a] When prevalence of any soil-transmitted helminthiasis (STH) is less than 20%, large-scale preventive chemotherapy interventions are not recommended. Affected individuals should be dealt with on a case-by-case basis.

[b] If resources are available, a third drug-distribution intervention might be added. In this case, the appropriate frequency of treatment would be every 4 months.

Immunization

None available.

Epidemic control

STH infection is usually endemic, with little likelihood of rapid changes in incidence.

References

1. *Soil-transmitted helminths*. Geneva, World Health Organization (http://www.who.int/intestinal_worms/en/, accessed June 2009).

CD-WGE technical focal point: Department of Control of Neglected Tropical Diseases (NTD)

TETANUS

Description

Clinical description

Tetanus results from contamination of wounds with spore forming bacterium *Clostridium tetani*, which produces *tetanospasmin*, a neurotoxin leading to muscles spasm and contraction, with the characteristic fixed smile, locked jaw and arching back, and sudden generalized seizures.

Neonatal tetanus occurs when tetanus occurs in a neonate at 3–28 days after birth, usually when unclean instruments are used to cut the umbilical cord or, when contaminated materials are used to cover the umbilical stump. Maternal tetanus occurs during pregnancy or within 6 weeks post partum, usually as a consequence of unclean delivery or induced abortion. Overall case fatality is high (10–80%) without hospitalization and intensive care; fatality approaches 100% at extremes of age (infancy and elderly).

Infectious agent

Bacterium: *Clostridium tetani*

Case definition for neonatal tetanus

Suspected case:

Any neonatal death at age 3–28 days in which the cause of death is unknown; **or** any neonate reported as having suffered from neonatal tetanus at age 3–28 days and not investigated.

Confirmed case:

Any neonate with a normal ability to suck and cry during the first 2 days of life, **and** who, between the age of 3 and 28 days, cannot suck normally, **and** becomes stiff or has spasms (i.e. jerking of the muscles).

Note: The basis for case classification is purely clinical and does not depend on laboratory confirmation. Cases of neonatal tetanus reported by physicians are considered as confirmed (*1*).

Mode of transmission

Infection occurs when *C. tetani* spores, which occur worldwide as constituents of soil and in the gastrointestinal tracts of animals (including humans), are intro-

duced into the body through open wounds, puncture wounds or surgical sites (when surgical procedures are performed under unhygienic conditions). Cases have resulted from infection of wounds considered too trivial for medical attention.

Neonatal tetanus usually occurs through the introduction of spores via the umbilical cord (e.g. through the use of an unclean instrument to cut the cord during delivery, or through the application of contaminated materials on the umbilical stump after delivery).

Incubation period

Usually 3–21 days; 10 days on average (may range from 1 day to several months). Shorter incubation periods have been shown to be associated with heavily contaminated wounds, more severe disease and poor prognosis.

Period of communicability

No direct person-to-person transmission

Reservoirs

C. tetani is a harmless normal inhabitant of the intestines of horses, other animals and humans. Tetanus spores are ubiquitous in the environment and can enter the body through any type of wound via soil or objects contaminated with animal or human faeces.

Epidemiology

Disease burden

Despite being an easily preventable disease, tetanus, in particular neonatal and maternal tetanus, remains a major cause of mortality in developing countries. Côte d'Ivoire is one of 46 countries that, as of April 2009, have not eliminated neonatal tetanus.

In 1997, there were 241 reported cases of neonatal tetanus, dropping to 94 cases in 1998, and to 31 cases in 2007 (Table 24). However, only a fraction of all cases are reported. For example, 1513 cases and 1135 deaths (mortality rate of 2.09 per 1000 live births) from neonatal tetanus were estimated to have occurred in Côte d'Ivoire in 1999; only 83 cases were reported. The World Health Organization (WHO) and the United Nations Children's Fund (UNICEF) estimate that in 2007, 76% of newborns were protected against tetanus through maternal immunization (*2*).

Table 24. **Cases of neonatal tetanus and tetanus reported in Côte d'Ivoire, 2003–2007**

Year	Annual number of reported cases of neonatal tetanus	Total annual number of reported cases of tetanus
2007	31	Not available
2006	32	Not available
2005	2	2
2004	14	Not available
2003	12	12

Source: *Tetanus (neonatal) reported cases*. Geneva, World Health Organization, dated 16 December 2008 (*http://www.who.int/Immunization_monitoring/en/globalsummary/timeseries/tsincidencente.htm*, accessed May 2009).

WHO/UNICEF estimates for coverage of the third dose of the diphtheria-tetanus-pertussis vaccine (DTP3) in Côte d'Ivoire was 76% in 2007. Côte d'Ivoire has joined other countries in implementing supplementary immunization activity with tetanus toxoid targeting all women of childbearing age (usually aged between 15 and 45 years), living in high-risk areas, in order to achieve the goal of elimination of maternal and neonatal tetanus.

Geographical distribution

Worldwide, although it is more common in agricultural areas where contact with animal excreta is more frequent and immunization coverage is inadequate. Rural, impoverished and tropical areas bear the greatest burden of this disease.

Seasonality

Universal.

Risk factors for increased burden

Population movement

Mass population displacement may put the displaced population at risk of accidental injuries, while inadequate wound-care in such settings may increase the risk of tetanus infection.

Overcrowding

Not relevant

Poor access to health services

In settings where access to health services is poor, it is likely that there will be:

- Inadequate wound management and treatment;
- Poor provision of protection through immunization;
- Inadequate care during childbirth (thereby increasing the risk of neonatal and maternal tetanus).

Food shortages

Not relevant

Lack of safe water, poor hygienic practices and poor sanitation

Poor wound care allows tetanus spores to settle on open wounds. Poor sanitation practices allow for increased contamination of formites with spores.

Prevention and control measures

Case management

- Prompt treatment with tetanus immunoglobulins (if available), tetanus antitoxin (if immunoglobulins not available), tetanus toxoid and antibiotics;
- Wound care;
- Airway management;
- Intensive care can improve outcomes, but is rarely available.

Prevention

Immunization is the best means of prevention. Strategies for prevention include:

- Education of the public on the necessity of immunization;
- Universal active immunization with tetanus toxoid (or tetanus toxoid-containing vaccines);
- Tetanus prophylaxis as a part of wound management (for wounds that may be prone to infection with tetanus, e.g. punctures, burns, sepsis, wounds contami-

nated with soil/manure, or any wound more than 6 hours old) and provide a full primary course of tetanus toxoid plus tetanus immunoglobulin if available) (*3*) (see Annex 6: *WHO fact sheets and information sources: Wounds and injuries* for other reference documents).

Prevention of maternal and neonatal tetanus requires maternal immunization with tetanus toxoid and use of hygienic delivery practices (e.g. assistance by a trained attendant, delivery in a health facility, adaptation of traditional practices known to increase the risk of infection, etc).

Immunization

Neonatal tetanus can be prevented by vaccinating women of child-bearing age either during pregnancy or outside of pregnancy (3 doses of tetanus toxoid with an interval of at least 4 weeks between the first and second dose and at least 6 months interval between the second and third dose. These are minimum intervals. If more time lapses, the schedule should not be re-started, but the next due dose should be given).

For lifelong immunity, a person should receive three doses of tetanus toxoid-containing vaccine (DTP) in infancy, followed by a tetanus toxoid-containing booster at school entry (age 4–7 years), during adolescence (age 12–15 years) and during early adulthood, hence a total of six doses.

Importantly, recovery from clinical tetanus does not result in protection against the disease in the future and hence immunization is required.

Epidemic control

Outbreaks are rare, but when they occur, a thorough case-investigation and search for a common source (e.g. repeated use of needles for injections, unhygienic medical and/or delivery procedures, low vaccination coverage, contaminated drugs etc.) should be carried out.

References

1. *WHO-recommended standards for surveillance of selected vaccine preventable diseases*. Geneva, World Health Organization, 2003 (WHO/V&B/03.01) (http://www.who.int/immunization/documents/WHO_VB_03.01/en/index.html, accessed June 2009).
2. *WHO-UNICEF estimates of PAB coverage*. Geneva, World Health Organization, dated 16 December 2008 (http://www.who.int/Immunization_monitoring/en/globalsummary/timeseries/tswucoveragepab.htm, accessed May 2009).

3. *Prevention and management of wound infection. Guidance from WHO's Department of Violence and Injury Prevention and Disability and the Department of Essential Health Technologies*. Geneva, World Health Organization (http://www.who.int/hac/techguidance/tools/Prevention%20and%20management%20of%20wound%20infection.pdf, accessed June 2009).

CD-WGE technical focal point: Department of Immunization, Vaccines and Biologicals (IVB)

TUBERCULOSIS

Description

Clinical description

The most important symptom of pulmonary tuberculosis (TB) is a productive cough of long duration (> 2 weeks). Other symptoms include haemoptysis, significant weight loss, chest pain, breathlessness, fever, night sweats, tiredness, and loss of appetite. In camp settings, it is the priority of the health services to detect sources of infection by sputum microscopy, and to cure these people, as patients with smear-positive pulmonary TB are the main source of transmission. Extrapulmonary TB may involve bone, brain/meningitis, lymph nodes and any organ including kidney, liver and spleen.

Infectious agent

Bacterium: *Mycobacterium tuberculosis.* This complex of infectious agents includes *M. tuberculosis* and *M. africanum* primarily from humans, and *M. bovis* primarily from cattle. More recently, *M. canettii* and *M. microti* have been incorporated in this complex.

Case definition

Suspected case:

Any person with symptoms or signs suggestive of TB, in particular cough of long duration (> 2 weeks or in accordance with current National TB Control Programme recommendations).

Confirmed case:

A patient with bacteriologically-confirmed TB or who has been diagnosed by a clinician as having TB.

Note: Any person given anti-TB treatment should be recorded as a case. A "trial" TB treatment should not be used as a means of diagnosis.

Definite case of TB

A patient with culture-positive *M. tuberculosis* complex infection. (In countries where culture is not routinely available, a patient with at least one sputum smear that is positive for acid-fast bacilli (AFB) is also considered a "definite" case.)

Note: This new definition of sputum-smear TB case is recommended by WHO and was endorsed by the Strategic Technical and Advisory Group for TB in June 2007 (*1*).

Laboratory criteria for diagnosis

Each suspected case of TB should have two (formerly three) sputum samples examined by light microscopy for AFB by Ziel-Nielsen staining. The best sputum yield is obtained in the early morning, therefore at least one sample should be from an early-morning collection.

If at least one sputum smear is positive

Any TB suspect with at least one positive smear result is categorized as a smear-positive TB patient, who must then be registered and started on anti-TB treatment.

When the two sputum smears are negative

If the initial two smears give a negative result, but suspicion of TB remains high, give a short (at least 1 week) trial of antibiotics as treatment for acute respiratory infections (e.g. amoxicillin or co-trimoxazole; **do not use anti-TB drugs or any fluoroquinolone**). If there is no improvement, the patient's sputum must be re-examined for AFB, 2 weeks after the first sputum examination.

At least 65% of all cases of pulmonary TB are expected to be confirmed by positive sputum-smear examination. Chest X-ray lesions compatible with active TB should encourage further sputum examination if the two sputum-smear examinations were negative. X-ray itself is not a diagnostic tool for pulmonary TB.

In some 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 the two samples again give negative results 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 judgment alone will decide whether someone is categorized as having smear-negative pulmonary TB.

Screen close contacts (household or shelter members) with suggestive symptoms for TB suspicion (e.g. productive cough of long duration) using the same procedure, giving priority to children and people with underlying conditions such as HIV-infected persons. Isoniazid preventive therapy should be provided to TB contacts aged less than 5 years (5 mg/kg per day for 6 months) if they are screened negative for active TB.

TB in HIV-infected patients

HIV-infected patients with TB infection have a much higher risk of developing active TB. The clinical presentation of TB depends on the degree of immunosuppression, with pulmonary TB still the commonest form of TB in HIV-infected patients. The principles of TB control are the same.

It is recommended that TB patients are offered HIV testing as the prevalence of HIV in adults in Côte d'Ivoire is ≥ 1%.

HIV-positive patients should be assessed carefully for concurrent TB infection. HIV-positive TB patients should be provided with co-trimoxazole preventive therapy. Whenever possible, eligible HIV-positive TB patients should be provided with antiretroviral therapy though the timing of this in relation to commencement of TB therapy remains contentious. The patient should continue to be observed for the development of TB symptoms after commencement of antiretrovirals, as TB immune-restoration disease may occur.

Diagnostic criteria for classification of TB in adults

WHO has recently revised recommendations for diagnosis and treatment of TB in resource-constrained settings with a high prevalence of HIV.

Pulmonary TB

Pulmonary TB refers to disease involving the lung parenchyma. *Tuberculous intrathoracic lymphadenopathy (mediastinal and/or hilar) or tuberculous pleural effusion without lung involvement is a case of extrapulmonary TB.* A patient with both pulmonary and extrapulmonary TB should be classified as a case of pulmonary TB.

Smear-positive pulmonary TB:

The revised case definition of smear-positive pulmonary TB is the same for HIV-infected and non-HIV-infected patients, i.e. requiring at least one positive smear in settings with a functional system of external quality assurance for smear microscopy.

Smear-negative pulmonary TB:

The revised case definition of smear-negative pulmonary TB includes:

- At least two sputum specimens that are negative for AFB; **and**
- Radiographic abnormalities consistent with active TB; **and**
- Laboratory confirmation of HIV infection; or
- Strong clinical evidence of HIV infection; **and**
- Decision by a clinician to treat with a full course of anti-TB chemotherapy.

If there is no laboratory confirmation of HIV infection or if the patient has no strong clinical evidence of HIV infection, the following criteria should be used to establish the diagnosis of smear-negative pulmonary TB:

- At least two sputum specimens that are negative for AFB; **and**
- No clinical response to a course of broad-spectrum antibiotics; **and**
- Radiographic abnormalities consistent with active pulmonary TB; **and**
- Decision by a clinician to treat with a full course of anti-TB chemotherapy.

This group also includes cases without a smear result, which should be exceptional in adults but relatively more frequent in children.

A patient whose initial sputum smears were negative and whose subsequent sputum-culture result is positive is a smear-negative pulmonary TB case whether or not there is a laboratory confirmation of HIV and whether or not there is strong clinical evidence of HIV infection.

Extra-pulmonary TB

Extra-pulmonary TB refers to TB of organs other than the lungs, e.g. pleura, lymph nodes, abdomen, genitourinary tract, skin, joints, bones and meninges. Diagnosis should be based on culture-positive specimens, or histological or strong clinical evidence consistent with active extra-pulmonary TB, followed by a medical decision by a clinician to treat with a full course of anti-TB chemotherapy. Some cases will be easy to diagnose such as; peripheral lymphadenitis, with swelling of cervical or axial lymph nodes, chronic evolution and/or production of caseous discharge. Other cases such as TB of bone or joints, TB peritonitis, TB laryngitis, severe life-threatening forms (e.g. miliary TB, TB meningitis) usually require a referral to a hospital for assessment.

Diagnosis of TB in children

The diagnosis of TB in children is difficult. Children rarely have smear-positive pulmonary TB and often have extrapulmonary TB. They are rarely infectious. The diagnosis of TB should be considered in a child if there is:

- An illness lasting for more than 10 days;
- A history of close contact with a TB patient;
- A poor response to antibiotic therapy;
- Weight loss or abnormally slow growth;
- Loss of energy; or
- Increasing irritability and drowsiness.

A hospital referral for X-ray and special examinations (e.g. lumbar puncture in case of meningitis TB suspicion) is often required.

- Children with headache, change of temperament, recent squint or ocular muscle paralysis should be suspected of TB meningitis.
- Children with high fevers, dyspnoea, gastrointestinal symptoms, confusion should be suspected for acute miliary TB is suspected.
- Suspected TB of the bone, tuberculous arthritis or pleural effusions also require referral.
- Commoner forms of extrapulmonary disease (e.g. cervical or axillary lymphadenitis, peritonitis with ascites) can be diagnosed and treated in a camp situation.

Note: The same considerations explained above in adults apply in children for the diagnosis of TB in HIV-positive patients.

Mode of transmission

Exposure to tubercle bacilli in airborne-droplet nuclei produced by people with pulmonary or laryngeal TB during expiratory efforts, such as coughing and sneezing.

Bovine TB results from exposure to tuberculous cattle, usually by ingestion of unpasteurized milk or dairy products (rare).

Incubation period

About 2–10 weeks from infection to demonstrable primary lesion or significant tuberculin reaction. The subsequent risk of pulmonary or extrapulmonary TB is greatest in the first 1–2 years, although latent infection may persist for a lifetime.

Period of communicability

As long as viable tubercle bacilli are being discharged in the sputum. Effective treatment usually eliminates communicability within 2–4 weeks in household settings, although TB may still be found intermittently in expectorated sputum thereafter.

Reservoirs

Humans.

In some areas, diseased cattle are reservoirs.

Epidemiology

Disease burden

A global disease, TB has widespread implications beyond the individual and country. While many regions have reported a relatively stable number of TB notifications over the last decade, the WHO African Region has had a precipitous climb in TB notifications, contributing more than a quarter of new smear-positive cases globally. A total of more than 2.5 million persons were newly diagnosed with TB in Africa in 2005, with more than 1 million of these being smear-positive, giving a prevalence of 511 per 100 000 population (*2*). Mortality caused by TB in this region is the highest in the world with 74 deaths per 100 000 population. In contrast, the rate of mortality caused by TB is 5.5 per 100 000 in the Americas (*2*).

The impact of the HIV epidemic has forced renewed attention on TB. In the WHO African Region, TB is the leading cause of death among people living with HIV; of the estimated 1.37 million HIV positive TB patients globally in 2007, 80% live in sub-Saharan Africa. Ravaged by war, the long-term repercussions of a disrupted health system cannot be overstated and the impact is the greatest in those with co-existent complicated, high-fatality diseases such as these. The rise in multidrug resistant TB and extremely drug-resistant TB in and beyond the region underlines the pressing need for sustained coordinated clinical care and research.

Globally, Côte d'Ivoire is among the 15 countries with the highest incidence of TB. The impact of the HIV epidemic and years of civil war resulting in major disruptions in health services have severely worsened the disease burden. In 2006, the incidence of TB was 420 cases per 100 000 population per year, with a prevalence of 747 per 100 000 (Table 25), higher than the overall prevalence for the WHO African Region. The directly observed treatment, short-course (DOTS) notification rate of new and relapsed patients is 110 per 100 000 population and the DOTS treatment success rate is 76%. Less than three quarters present with smear-positive pulmonary disease. The rise in numbers presenting with extra-pulmonary TB and smear-negative TB (i.e. more difficult to diagnose) is concerning. The HIV prevalence in incident case of TB is 13.62% in Côte d'Ivoire, although this is probably an underestimate since the tracking of HIV among TB patients is relatively recent.

Table 25. **Morbidity caused by tuberculosis in Côte d'Ivoire**

Year	Annual incidence of TB per 100 000 population	Prevalence of TB per 100 000 population	TB detection rate under DOTS (%)	TB treatment success rate under DOTS (%)
2006	420	747	37	76
2000	364	651	32	No data
1990	168	333	50	68

Source: Indicator definitions and metadata, 2008. WHO Statistical Information System, 2008. (*http://www.who.int/whosis/indicators/compendium/2008/en/*, accessed May 2009).

DOTS, direct observation, short course; TB, tuberculosis.

Geographical distribution

Reported throughout the country with higher incidence around Abidjan (may be due to increased case ascertainment).

Seasonality

No specific seasonality.

Alert threshold

Not applicable.

Risk factors for increased burden

Population movement

Population displacement disrupts existing TB-control activities, resulting in an increased risk of transmission. Movement of untreated TB patients into new areas spreads the disease. Movement of susceptible persons (e.g. immunosuppressed, malnourished, HIV-infected persons) into TB-endemic areas or camps increases the risk of acquisition. Treatment interruptions, treatment failure, treatment relapse and non-adherence to combination therapy lead to persistent reservoirs of TB and increase the risk of multidrug resistant TB.

Overcrowding

Overcrowding and poor indoor ventilation contribute to increased risk of transmission.

Poor access to health services

People affected by TB who cannot access health services for diagnosis and treatment remain infectious, thereby increasing transmissibility. Directly-observed therapy is a key component to maintaining drug adherence. The case-fatality ratio (CFR) is high without proper treatment. Interruption of treatment is one of the most important causes of development of multidrug-resistant TB.

Food shortages

Malnourished populations, especially malnourished children of all ages, are considered to be at particular risk of developing severe active TB.

Lack of safe water, poor hygienic practices and poor sanitation

Not relevant.

Other

The spread of HIV infection has brought about a new urgency to TB management, with a rise in numbers of co-infected patients being associated with resistant TB and associated high rates of mortality.

Prevention and control measures

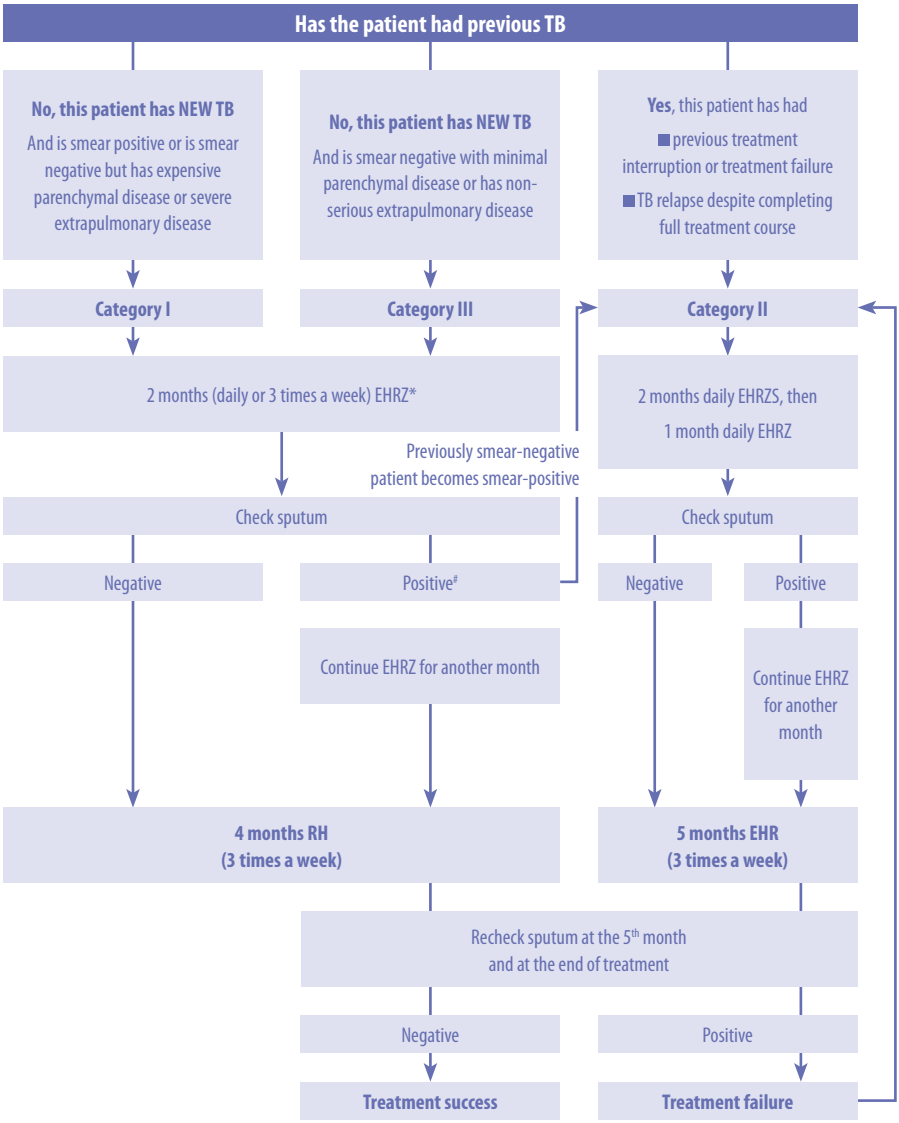
Case management

After the diagnosis of TB, 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:

- Site of disease;
- Severity of the disease;
- Bacteriological status (assessed by sputum microscopy);
- History of anti-TB treatment (new or previously treated).

The management approach is summarized in the flowchart, Figure 13

Fig. 13 **Flowchart for the management of tuberculosis (TB)**



* Ethambutol may be omitted in the initial phase of treatment in patients with limited parenchymal involvement, non cavitary smear-negative pulmonary TB who are known to be HIV-negative and in children with primary TB; #: a previously smear-negative patient who becomes smear-positive whilst on TB treatment should be re-registered as treatment failure and commenced on Category II treatment.

EHRZS, ethambutol, isoniasid, rifampicin, pyrazinamide, streptomycin

New case

A patient who has never had treatment for TB or who has taken anti-TB drugs for less than 4 weeks and has:

- Sputum smear-positive pulmonary TB; or
- Sputum smear-negative pulmonary TB, or extrapulmonary TB.

Previously-treated case

A patient who has at any time received anti-TB treatment for more than 1 month. This group of patients comprises:

- **Return after interruption:** patient who returns to treatment with positive bacteriology (e.g. sputum smear-positive microscopy), following interruption of treatment for 2 months or more. This could be common among recent refugees or internally displaced persons.
- **Failure:** a patient who remained, or returned to being, smear-positive, 5 months or later during treatment; also, a patient who was smear-negative before starting treatment and became smear-positive after the second month of treatment. Therefore, after having failed a previous treatment these patients are started on a re-treatment regimen.
- **Relapse:** a patient previously treated for TB who has been declared cured or treatment completed, and is diagnosed with bacteriologically confirmed (smear- or culture-positive) TB**.**
- **Chronic:** a patient who remained, or returned to being, smear-positive at the fifth month or the end of a fully supervised, standardized re-treatment regimen.

Good case management includes directly observed therapy during the intensive phase for all new sputum smear-positive cases, the continuation phase of rifampicin-containing regimens and the entire re-treatment regimen.

There are three main types of treatment regimens: **Category I** for new smear-positive pulmonary cases, and severely ill TB patients, **Category II** for re-treatment cases, and **Category III** for smear-negative pulmonary or extrapulmonary cases (not severely ill patients). See Treatment categories below.

The chemotherapeutic regimens are based on standardized combinations of five essential anti-TB drugs:

- rifampicin (R)
- isoniazid (H)
- pyrazinamide (Z)
- ethambutol (E), and
- streptomycin (S).

TB drugs should be given to TB patients in fixed-dose combination forms.

Each of the standardized chemotherapeutic regimens consists of two phases:

Initial (intensive) phase

- 2–3 months, with three to five drugs given daily under direct observation, for maximum reduction in the number of TB organisms.
- The number of drugs used relates to the risk of failure of treatment due to possible bacterial resistance.

Continuation phase

- 4–6 months, with two to three drugs (including rifampicin) given three times per week under direct observation or, in some cases (e.g. during repatriation of refugees), two drugs (ethambutol and isoniazid) for 6 months given daily unsupervised, but in a fixed-dose combination form.*
    - All doses of rifampicin-containing regimens should be given by staff.
    - Swallowing of medication must be confirmed by health worker or treatment supporter.
    - Hospitalized patients should be kept in a separate ward for the first 2 weeks of treatment.

** 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 doses per week written after the letter. If there is no number after the letter, a daily dosage is given. The symbol "/" separates the different phases of the therapy, e.g. 2RHZE/4H3R3 means that for the first 2 months of treatment, rifampicin, isoniazid, pyrazinamide and ethambutol are given daily. This is followed by 4 months of rifampicin and isoniazid given regularly but each given only three times per week.*

HIV-positive patients

Anti-TB drug treatment is the same for HIV-positive and HIV-negative patients. However:

- Thioacetazone should not be administered to HIV-positive TB patients as there is an increased risk of severe and sometimes fatal skin reactions; and

- HIV-positive TB patients should be prescribed a rifampicin containing regimen throughout the 6-month course of treatment.

Studies of controlled clinical trials 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).

The decision to use isoniazid preventive therapy must be carefully evaluated, and firstly requires exclusion of active TB in the patient.

To manage effectively the problem of co-infection with TB and HIV, TB and HIV programmes should coordinate activities through a TB/HIV coordinating body (*3*).

The guidelines of the national tuberculosis control programme in Côte D'Ivoire should also be consulted.[3]

Standardized short-course chemotherapy using regimens of 6–8 months

Category I: EHRZ/RH

These patients are:

- New smear-positive TB cases; and
- Severely ill patients with other forms of TB (new smear-negative pulmonary TB with extensive parenchymal involvement, and new cases of severe forms of extrapulmonary TB). This category includes patients with TB meningitis, disseminated TB, pericarditis, peritonitis, bilateral or extensive pleurisy, vertebral disease with neurological complications, and intestinal and genitourinary disease.

The recommended regimen is for 6 months. The initial (or intensive) phase of treatment lasts for 2 months, with rifampicin, isoniazid, pyrazinamide and ethambutol given daily or three times per week, under direct supervision.

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 three times per week, under direct supervision. Daily self-administered ethambutol and isoniazid may be used in the continuation phase for 6 months, so this treat-

3. To obtain these guidelines, please contact: Ministère de la Santé de l'Hygiène publique, Programme National de Lutte Contre la Tuberculose, Abidjan, Côte D'Ivoire; e-mail: pnlt-rci@aviso.ci.

ment regimen takes a total of 8 months. However, note that there is a higher rate of treatment failure and relapse associated with this regimen using ethambutol and isoniazid in the continuation phase.

If, the sputum-smear examination is positive at the end of the second month, the initial phase is prolonged for a third month. The patient then starts the continuation phase irrespective of the results of the sputum examination at the end of the third month.

In the continuation of the treatment, if the smears are still positive at the end of the fifth month or at the end of the treatment regimen, the patient is classified as a **treatment failure.** The patient is re-registered, and commences a full course of the re-treatment regimen as a category II patient.

Drug dose is adjusted for body-weight gain at the end of the initial phase (second or third month).

Category II: 2SHRZE/1HRZE/5HRE

This category should be used for patients who were previously treated and are now sputum smear-positive. This includes:

- Treatment after interruption;
- Treatment failure; and
- Relapse after treatment.

These patients should receive a standardized re-treatment regimen, fully supervised throughout both phases of treatment.

The initial phase of treatment lasts for 3 months, where isoniazid, rifampicin, pyrazinamide and ethambutol are given daily. This regimen is supplemented by streptomycin daily for the first 2 months.

The continuation phase of this regimen is followed by 5 months of isoniazid, rifampicin and ethambutol given three times per week.

Sputum-smear examination is performed at the end of the initial phase of treatment (at the end of the third month), 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 with isoniazid, rifampicin, pyrazinamide and ethambutol for one more month. Patients who are still positive at the end of the fourth month progress to the continuation phase, regardless of the results of the sputum examination. If patients are still or become smear-positive at the fifth

month or at the end of the category-II treatment, they should be considered as chronic TB cases and referred to health facilities where they can be appropriately managed.

Category III

The category III regimen is indicated in:

- Patients with smear-negative pulmonary TB (with limited parenchymal involvement)
- Adults and children with non-serious extrapulmonary TB (including symptomatic primary TB usually observed in children).

These patients receive the same treatment regimen as category I patients. However, ethambutol may be omitted in the initial phase of treatment in patients with limited parenchymal involvement, non cavitary smear-negative pulmonary TB who are known to be HIV-negative and in children with primary TB.

When the continuation phase cannot be carried out under direct observation, daily ethambutol and isoniazid should be used in the continuation phase for 6 months. All doses of rifampicin-containing regimens should be directly observed by staff or treatment supporter. Confirmation that the medication has been swallowed should be obtained.

Prevention

Detection and treatment of smear-positive (infectious) TB cases are the most effective interventions to prevent the transmission of TB.

Key elements of community health education emphasize:

- De-stigmatization of TB patients;
- Curability of TB disease;
- Early (self-) referral of suspected cases of TB;
- Importance of adherence to treatment;
- Investigation of contacts.

The most important messages to convey are:

- TB in adults should be suspected when the person has a productive cough of long duration (> 2 weeks) or in accordance with the directives of the NTCP, and/or blood in sputum, with significant weight loss.
- Cover the mouth whenever coughing or sneezing to prevent the spread of microorganisms.

- 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 a severe, even fatal, form of TB.
- Identification of TB and appropriate treatment constitute the best prevention.
- All TB patients must take the full course of treatment prescribed.
- Treatment makes patients non-infectious within 8 weeks, but cure takes 6–8 months.
- Treatment must be completed even though the patient may feel better sooner.
- Interruption of treatment may result in a recurrence of TB, which may be difficult or impossible to treat and spread TB bacilli to others, especially to 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 level of literacy should not be assumed. Cured patients are often helpful teachers and supporters of new patients.*

Good ventilation and reduction of overcrowding should be ensured in health clinics, and separation of hospitalized patients in a dedicated ward for at least the first 2 weeks of treatment.

Particular care should be used to separate infectious TB patients from HIV-positive individuals.

Vaccination with Bacille Calmette–Guérin (BCG) should be used to prevent severe forms of TB in children (see immunization section below).

Children (aged less than 5 years) who are close contacts of patients with smear-positive pulmonary TB and who, after investigation, do not have active TB, should receive isoniazid prophylaxis as follows: 5 mg/kg per day for 6 months with regular follow-up (e.g every 2 months). This will significantly reduce the likelihood of TB disease. Breastfeeding children of sputum smear-positive mothers are the most important group for isoniazid prophylaxis. If the child is well, BCG vaccination can be carried out after the course of isoniazid prophylaxis; 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). Children aged more than 5 years who are well do not require prophylaxis, but only clinical follow-up.

Immunization

BCG has been shown to be effective in preventing severe forms of disease such as TB meningitis and miliary TB in children. As overcrowding and malnutrition are common in many refugee and displaced populations, the risk of TB transmission to children is increased. 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 is part of the national immunization schedule. In 2006, the estimated coverage of BCG was 77% in Côte d'Ivoire. Re-vaccination with BCG is not recommended.

Epidemic control

Recognize and treat new and secondary cases.

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CD-WGE technical focal point: Stop TB (STB)

TYPHOID FEVER

Description

Clinical description

Typhoid fever is a systemic bacterial infection, characterized by insidious onset of sustained fever, severe headache, nausea, loss of appetite, malaise, constipation or sometimes diarrhoea. Clinical manifestations may vary from an inapparent or mild illness to severe clinical disease with multiple complications. Severe forms have been described in association with mental dullness and meningitis.

Case-fatality ratios (CFRs) of 10–20% among untreated patients can be reduced to less than 1% with prompt and appropriate antibiotic therapy. However, strains that are resistant to all three of the first-line antibiotics in use (chloramphenicol, ampicillin and co-trimoxazole) have become prevalent in several areas of the world; these strains are associated with more severe illness and higher rates of complications and death, especially in children aged less than 2 years.

The symptoms of paratyphoid fever are similar to those of typhoid fever, but tend to be milder, with a lower fatality rate.

Infectious agent

Typhoid fever: bacterium *Salmonella enterica* subsp. *enterica* serovar Typhi (commonly *S.* Typhi, the latter not italicized according to new nomenclature).

Paratyphoid fever: *Salmonella enterica* subsp. *enterica* serovar Paratyphi var. A & B (commonly *S.* Paratyphi A & B according to new nomenclature).

Case definition

Suspected case:

Clinical diagnosis is difficult. Without laboratory confirmation, any case with fever of at least 38 °C for 3 or more days is considered suspect if the epidemiological context is conducive.

Confirmed

A suspected case with isolation of *S.* Typhi from blood or stool cultures.

Carrier

S. Typhi organisms persisting in stools or urine for more than 1 year after onset of the disease.

Mode of transmission

Faecal–oral route, particularly ingestion of food and water contaminated by faeces and urine of patients and carriers. In addition, shellfish taken from sewage-contaminated beds, vegetables fertilized with night-soil and eaten raw; contaminated milk and milk products have also been shown to be sources of infection.

About 2% of infected adults are faecal carriers. Patients with concurrent *Schistosoma haematobium* infection are at a higher risk of becoming urinary carriers of *S.* Typhi.

Incubation period

Depends on inoculum size and host factors. Usually 8–14 days but may be from 3 days up to more than 2 months. For paratyphoid, 1–10 days.

Period of communicability

From the symptomatic period for 2 weeks, but people can transmit the disease as long as the bacteria remain in their body: 2–5% of infected cases remain carriers for several months or longer. Chronic asymptomatic carriers are greatly implicated in spread of the disease.

Reservoirs

Humans for both typhoid and paratyphoid. Rarely, domestic animals for paratyphoid.

Epidemiology

Disease burden

Typhoid fever occurs worldwide with an estimated 21 million cases and 216 000–600 000 deaths annually. The real impact of the disease is difficult to assess because the clinical picture is confused with other febrile illnesses and the laboratory diagnosis in developing countries is not standardized. Most of this burden occurs in Asia. In some regions, especially in Africa, estimates of the burden of typhoid fever are limited by insufficient data.

In the last outbreak in the Democratic Republic of the Congo, between 27 September 2004 and early January 2005, no less than 42 564 cases of typhoid fever were reported, including 214 deaths and 696 cases of peritonitis and intestinal perforations. In almost all endemic areas, the incidence of typhoid fever is highest in children of school age or younger.

S. Paratyphi is becoming predominant in some provinces in China and increasing numbers of cases are being reported from Pakistan.

Data on the burden of typhoid disease in Côte d'Ivoire are not readily available.

Geographical distribution

No specific data on the geographical distribution of typhoid within Côte d'Ivoire are available. All regions are considered to be endemic.

Seasonality

No specific data are available on seasonality in Côte d'Ivoire.

Alert threshold

Two or more linked cases must lead to an epidemiological investigation.

Epidemics

Although largely an endemic disease, *S.* Typhi has epidemic potential.

No epidemics have been documented in Côte d'Ivoire.

Risk factors for increased burden

Population movement

The disease can cause outbreaks, particularly in complex emergency settings with population movement, where there is a lack of safe food and water and access to adequate sanitation facilities. Dissemination of multidrug-resistant strains of *S.* Typhi as a result of population movement is an emerging issue.

Overcrowding

Increases contact with infected individuals and facilitates transmission.

Poor access to health services

Early detection and containment of cases are vital in reducing transmission. The CFR is high (10–20%) without appropriate treatment. Poor surveillance and monitoring are further obstacles to effective prevention and control of disease.

Food shortages

Not relevant.

Lack of safe water, poor hygienic practices and poor sanitation

In the general population, the risk is related to the availability of safe food and water and access to adequate sanitation facilities. Poor hygienic practices in food preparation and handling, washing, and after defecation are further contributors. Although largely considered to be an endemic disease, epidemics do occur, frequently as a result of breakdowns in water supplies and sanitation systems.

Others

Emergence of multidrug-resistant strains of *S.* Typhi including resistance to ciprofloxacin. Milk and dairy products are an important source of infection.

Prevention and control measures

Case management

Early antimicrobial treatment, selected according to the antimicrobial resistance pattern of the strain. Quinolones (e.g. ciprofloxacin), co-trimoxazole, chloramphenicol and ampicillin are usually used for typhoid fever. Dehydration prevention and case management using oral rehydration salts (ORS) therapy plays an important role (*1*).

Prevention

Prevention depends on the provision and use of safe water, adequate sanitation and health education for proper hygiene and food safety (see *Diarrhoeal diseases [others]: Prevention;* and Annex 4: *Safe water and sanitation*).

Immunization

Two new-generation typhoid vaccines – live, oral Ty21a and injectable Vi polysaccharide – have been shown to be safe and efficacious, and are internationally licensed for people aged more than 2 years. The single-dose injectable Vi vaccine provides about 70% protection, and protection lasts at least 3 years. The live attenuated Ty21a vaccine is available as capsules and as a liquid suspension, both administered orally. The liquid formulation is licensed for use in people aged more than 2 years; the capsules from age 5 years. The recommended three- to four-dose schedule (one dose every other day) of the liquid Ty21a vaccine provides 53–78% protection. Similar levels of protection are achieved with four doses (one dose every other day) of the Ty21a capsules. The need for revaccination is not well defined. However, in most endemic settings, one booster dose of the concerned vaccine 3–7 years after primary immunization seems to be appropriate.

In view of the continued high burden of typhoid fever and increasing antibiotic resistance, and given the safety, efficacy, feasibility and affordability of two licensed vaccines (Vi and Ty21a), countries should consider the programmatic use of typhoid vaccines for controlling endemic disease.

In most countries, the control of the disease will require vaccination only of high-risk groups and populations. Given the epidemic potential of typhoid fever, and observations on the effectiveness of vaccination in interrupting outbreaks, vaccination against typhoid fever is also recommended for outbreak control.

Immunization of school-age and/or pre-school-age children is recommended in areas where typhoid fever in these age groups is shown to be a significant public health problem, particularly where antibiotic-resistant *S.* Typhi is prevalent. Vaccination against typhoid fever may be offered to travellers to destinations where the risk of typhoid fever is high, especially to those staying in endemic areas for more than 1 month and/or in locations where antibiotic resistant strains of *S.* Typhi are prevalent.

All typhoid-fever vaccination programmes should be implemented in the context of other efforts to control the disease, including health education, water-quality and sanitation improvements, and training of health professionals in diagnosis and treatment (*2*).

Epidemic control

Epidemics often occur as point-source epidemics, from healthy carriers to food (including use of contaminated utensils). Outbreaks may occur through person-to-person contamination (faecal–oral transmission via contaminated hands or instruments). Direct faecal contamination of untreated water supplies may cause extensive outbreaks.

Investigations must pinpoint the source and mode of infection to identify control measures (chlorination/boiling of water, selective elimination of suspect food).

Inform the health authorities if one or more suspected cases are identified.

Confirm the outbreak, following WHO guidelines (see Annex 2).

Confirm the diagnosis and ensure prompt treatment.

References

1. *Background document: the diagnosis, treatment, and prevention of typhoid fever.* Geneva, World Health Organization, 2003 (WHO/V&B/03.07) (http://whqlibdoc.who.int/hq/2003/WHO_V&B_03.07.pdf, accessed June 2009).
2. Typhoid vaccines: WHO position paper. *Weekly Epidemiological Record* 2008, 83:49–60 (http://www.who.int/wer/2008/wer8306.pdf, accessed June 2009).

CD-WGE technical focal point: Department of Public Health and Environment (PHE)

YAWS (*Framboesia tropica*)

Description

Clinical description

After an incubation period of 9–90 days, a primary papular lesion forms which over several months may increase in size then heal spontaneously. Typically pruritic, further facilitating autoinoculation resulting in the development of tender regional lymphadenopathy. As the primary lesion heals, secondary lesions develop, which again usually heal spontaneously. After 5–10 years, 10% of untreated patients develop destructive lesions involving bone, cartilage, skin, and soft tissue, similar to those seen in tertiary syphilis. In contrast to venereal syphilis, cardiovascular and neurological abnormalities almost never occur in patients with yaws. There is no difference in susceptibility between males and females. Yaws predominantly affects children aged less than 15 years. Peak incidence occurs in children aged 6–10 years.

Infectious agent

Bacterium: *Treponema pallidum sp. pertenue*

Case definition

Clinical case: Any person who lives in an endemic area and presents with one or more of the following signs:

- Ulcer with scab
- Papillomas
- Palmar/plantar hyperkeratosis (thickening).

Yaws is classified into the following four stages:

- Primary stage: Initial yaws lesion develops at inoculation site;
- Secondary stage: Widespread dissemination of treponemes results in multiple skin lesions similar to primary yaws lesion;
- Latent stage: Usually, no symptoms are present, but skin lesions can relapse;
- Tertiary stage: Bone, joint, and soft tissue deformities may occur.

Mode of transmission

Primarily by direct contact (including autoinoculation), hence the disease predominates in children. There is little evidence of perinatal blood borne transmission.

Incubation period

9–90 days (mean, 21 days)

Period of communicability

Variable; may extend intermittently for several years when moist lesions are present. The infectious agent is not usually found in late destructive lesions.

Reservoirs

Occurs only in humans and possibly higher primates

Epidemiology

Disease burden

Yaws reemerged in western and central Africa in the 1970s following a successful control programme in the 1950s–1960s. It remains largely a neglected disease involving rural, isolated, silent communities and a renewed effort to understand its epidemiology and pathogenesis is necessary.

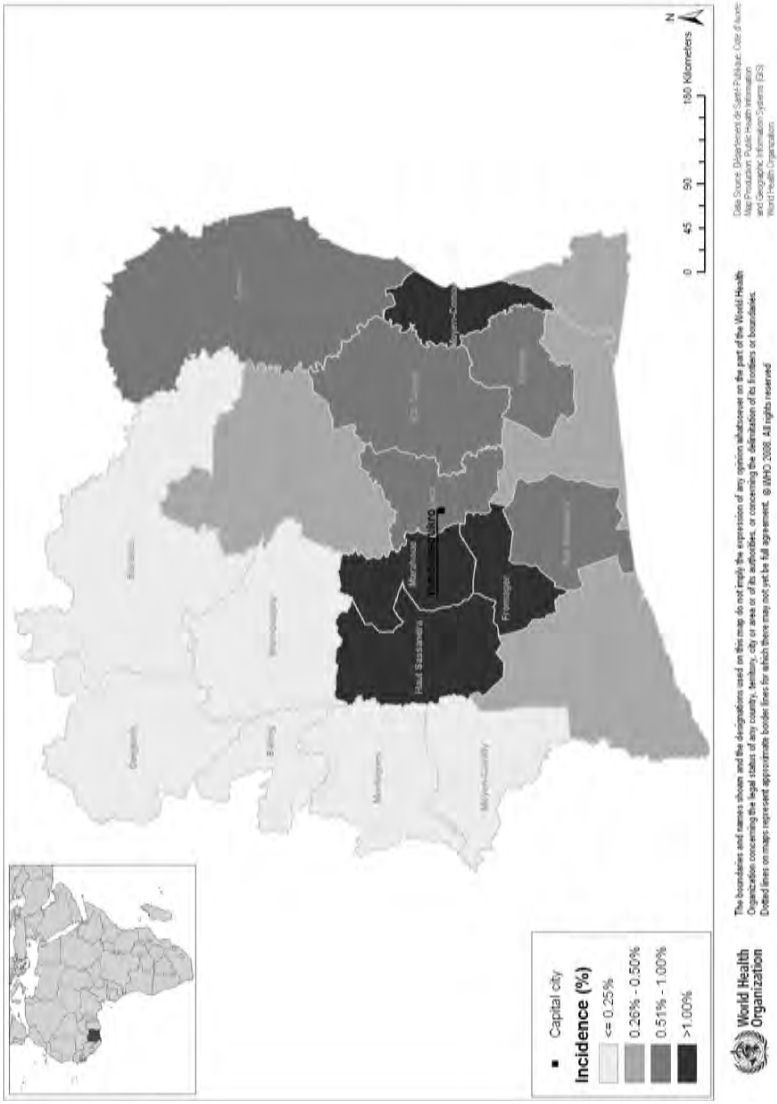
Little is known about the current exact number of cases of yaws in Côte d'Ivoire (or elsewhere) but yaws is generally regarded as endemic in the country. Data from the ministry of health suggest that the incidence was 0.6% in 2000, with 9212 new cases reported, falling from 1.5% in 1996. The estimates are likely to be an under-representation of the situation as most estimates are made by passive surveillance in rural areas (where the disease predominates), which have poorer reporting mechanisms.

The World Health Organization (WHO) and the United Nations Children's Fund (UNICEF) led a worldwide campaign to eradicate yaws in 1952–1964 by administering more than 50 million treatments in 46 countries, achieving a 95% reduction in prevalence. Unfortunately, yaws re-emerged in the 1970s in central and western Africa (notably Côte d'Ivoire, the Congo and Ghana), Haiti, India and Indonesia. WHO estimated there were 2.5 million endemic cases of yaws worldwide in 2000.

Geographical distribution

Previously distributed worldwide, yaws is now endemic in central and western Africa with scattered cases in Latin America, Caribbean islands, India, south-east Asia and some Pacific islands. All districts in Côte d'Ivoire are affected, the forest regions most severely (see Figure 14).

Fig. 14 **Incidence of yaws by region, Côte d'Ivoire, 2000**



Source: Toure B, Département de Santé Publique, Abidjan, Côte d'Ivoire.

Seasonality

Climate change may influence infectivity but little is known.

Risk factors for increased burden

Population movement

Movement of infected persons to new non-endemic areas will increase the geographical spread and burden of this disease.

Overcrowding

Transmission is facilitated by disruption of epithelial surfaces (lacerations, bites etc) and overcrowding leads to increased risk of spread.

Poor access to health services

A chronic infection, the period of transmissibility increases when access to health services is limited.

Food shortages

Not relevant.

Lack of safe water, poor hygienic practices and poor sanitation

Poor wound-care contributes to autoinoculation and transmission to others.

Prevention and control measures

Case management

Report to health authority. Avoid intimate contact and contamination of wound. Treat with a single intramuscular dose of 1.2 million units of benzathine penicillin for patients aged 10 years and older; 0.6 million units for those aged less than 10 years. For patients with penicillin hypersensitivity, use 15 days of oral tetracycline, erythromycin or doxycycline as alternatives. As the mode of transmission is by direct contact, contact tracing is important to break the chain of transmission and early detection of new cases.

Prevention

General health promotion, improving health services, including wound-care and improving sanitation habits.

Treatment of asymptomatic individuals is beneficial. WHO recommends treating the entire population when the prevalence is more than 10%. If prevalence is between 5% and 10%, treat patients, contacts and all children aged less than 15 years. If prevalence is less than 5%, treat active cases plus household and other contacts.

Immunization

None available.

CD-WGE technical focal point: Department of Control of Neglected Tropical Diseases (NTD)

YELLOW FEVER

Description

Clinical description

Disease severity ranges from an undifferentiated, self-limiting febrile illness to haemorrhagic fever that is fatal in 25–50% of cases. Some infected people do not show any symptoms.

There are two phases of disease associated with yellow fever:

Acute phase

Characterized by abrupt onset of fever, muscle pain (with prominent backache), headache, loss of appetite, nausea and/or vomiting with conjunctival injection, facial flushing and relative bradycardia. Most patients recover after 3–4 days and their symptoms disappear.

Toxic phase

In others, after a period of a few hours or few days where symptoms seemingly remit, profound symptoms recur with high fever, headache, lumbosacral pains, nausea, vomiting, abdominal pains and somnolence. The patient rapidly develops jaundice and 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 7–10 days after onset. The remaining patients recover without significant organ damage.

Infectious agent

The yellow fever virus belongs to the Flavivirus group.

Case definition

Suspected case: Acute fever followed by jaundice within 2 weeks of the onset of the first symptoms

Confirmed case: A suspected case that is laboratory confirmed (national reference laboratory) or epidemiologically linked to a confirmed case or outbreak.

Laboratory criteria for diagnosis:

- Isolation of yellow fever virus; **or**
- Presence of yellow fever-specific IgM or a fourfold or greater rise in serum IgG levels in paired sera (acute and convalescent) in the absence of vaccination against yellow fever; **or**
- Positive liver histopathology post mortem; **or**
- Detection of yellow fever antigen in tissues by immunohistochemistry; **or**
- Detection of genomic sequences from the yellow fever virus in blood or organs by polymerase chain reaction (PCR).

Mode of transmission

Bite of infective *Aedes* mosquitoes

The vectors of yellow fever in forest areas in west Africa are *Aedes furcifer-taylori and Aedes luteocephalus*. In urban areas, the vector is *Aedes aegypti* (all-day biting species).

There are three recognized types of transmission cycle for yellow fever and all types occur in Africa:

- Sylvatic yellow fever: generally results in sporadic cases most commonly among young men working in the forest;
- Intermediate yellow fever: results in small-scale epidemics and occurs in humid or semi-humid savannah, separate villages in an area suffer simultaneous cases;
- Urban yellow fever: results in large epidemics that tend to spread outwards from one source to cover a wide area.

In forested areas, where the yellow fever virus circulates between mosquitoes and monkeys, the disease is continuously present throughout the year. In field or savannah areas outside the forest areas, transovarian transmission (from one generation of mosquitoes to the next) has been documented but its contribution to the maintenance of infection is unknown.

Incubation period

3–6 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 sus-

ceptible people and abundant vector mosquitoes coexist. Once infected, mosquitoes remain so for life.

Reservoirs

Urban areas: humans and *Aedes* mosquitoes.

Forest areas: vertebrates other than humans, mainly non-human primates, and forest mosquitoes.

Epidemiology

Disease burden

Yellow fever is transmitted in sub-Saharan Africa and South America, but could, in principle, be seen in any *Aedes aegypti*-infested location. Globally, epidemics of yellow fever have been increasing over the last 20 years. Studies indicate that morbidity and mortality attributable to yellow fever are underestimated by a factor of 10–500. The precise extent of illness and death due to yellow fever is not known: cases of yellow fever go undetected because the signs and symptoms have a wide spectrum and overlap with those of many other diseases; mild cases may not seek care in a health facility; disease surveillance is not adequate to detect cases of sylvatic yellow fever that can occur in remote areas.

Mandatory mass vaccinations in the 1950s kept the incidence of yellow fever low in west Africa. However, as vaccine programmes and coverage waned, yellow fever re-emerged in the 1980s.

The annual rate of infection in west Africa is about 1%, but large epidemics have occurred with high rates of attack. Between 1986 and 1991, more than 20 000 cases and 5000 deaths were officially reported in Africa, but the true magnitude has been estimated to be 50 times greater.

Between 1983 and 1999, 12 cases were reported from Côte d'Ivoire: 11 in 1997, and 1 in 1999. Since 2000, there has been a sharp rise in the number of reported cases (Table 26). In 2001, a large outbreak originating in the west of the country spread to half of the country's districts, including Abidjan. In 2008, again, the Ministry of Health of Côte d'Ivoire declared an outbreak of yellow fever in Abidjan, which was laboratory-confirmed at the beginning of May.

Table 26. **The burden of yellow fever in Côte d'Ivoire, 1999–2008 (as of 31 August, 2008)**

Year	Number of reported cases	Number of reported deaths	EPI* vaccine coverage (%)
2008 (as of July 31)	13	1	Not available
2007	0	0	68
2006	16	3	67
2005	3	1	52
2004	92	4	47
2003	158	9	51
2002	156	23	51
2001	280	22	53
2000	31	6	65
1999	1	1	49

Sources: 1999–2004 http://www.who.int/csr/disease/yellowfev/trends/en/index.html#profile in French). 2005–2008: West Africa IST/ AFRO. For data up to and including 2004, as well as for 2006, annual reported cases include both suspected and confirmed cases. Annual cases for 2005 and 2007 include only laboratory confirmed cases. For 2008, annual reported cases include only cases confirmed by the regional reference laboratory (Institut Pasteur, Dakar).

*EPI, Expanded Programme on Immunization.

Geographical distribution

Increased circulation of the yellow fever virus in west Africa is linked to the existence of a high proportion of non-immunized subjects. The situation is aggravated by forced migrations of unprotected people to areas of risk and the decline of mass vaccination campaigns. Outbreaks were previously sporadic, but now are more widely distributed, with the potential for explosive urban events (Fig. 15).

Seasonality

Between late August and early March, mainly during the rainy season.

Yellow fever events

One confirmed case should be considered as a yellow fever event leading to an investigation and appropriate implementation of control measures. Recorded events in 2001–August 2008 are given in Table 27.

Fig. 15 **Geographical distribution of yellow fever events reported from Côte d'Ivoire, 1997–2007**

Table 27. **Yellow fever events, Côte d'Ivoire, 2001–2008**

Date	Event
As of 31 July 2008	Thirteen confirmed cases since May 2008. The Ivorian government conducted a mass-vaccination campaign covering 2.1 million people in Abidjan.
October 2006	Two confirmed cases in Korhogo and Ouragahio.
December 2005	Outbreak near Burkina Faso. Mass vaccination targeting subdistrict of Doropo in Bouna district. Achieved a vaccination coverage of 100% and resulted in immunization of 26 114 persons. Also planned to vaccinate another 290 000 persons in the remainder of Buona district and the surrounding district of Boundoukou.
September 2005	Outbreak in neighbouring Burkina Faso
March–October 2001	A large outbreak began in the west of the country, spreading to Abidjan and in the end affecting 31 of 62 districts, with 280 cases and 22 deaths (case-fatality ratio, 8%). Ten days after the last notification of cases in the city, a second peak occurred in the countryside with 8 suspected cases. WHO coordinated a large immunization campaign targeting 2.9 million persons.

Source: Disease outbreaks by country. Cote d'Ivoire. Geneva, World Health Organization (http://www.who.int/csr/don/archive/country/civ/en/, accessed May 2009).

Risk factors for increased burden

Population movement

Unvaccinated people moving to areas of endemicity are at risk. Threat of epidemic transmission when a person with a forest-acquired infection travels to an *A. aegypti*-infested location while viraemic. Changes in land use constitute a risk factor.

Overcrowding

Increased population density, as in urban settings, contributes to a favourable environment for the vector, *Aedes aegypti*. Living in temporary shelters exposes people to the increased risk of mosquito bites.

Poor access to health services

Collapse of vaccination programmes. Poor vector-control programmes contribute to increased burden.

Food shortages

Not relevant.

Lack of safe water, poor hygienic practices and poor sanitation

Poor environmental sanitation may promote vector breeding.

Prevention and control measures

Case management

No specific treatment for yellow fever is available. Dehydration and fever can be corrected with oral rehydration salts. Intensive supportive care may improve the outcome but is rarely available.

Prevention

- Preventive vaccination through routine childhood immunization and catch-up mass vaccination.
- Reactive mass vaccination to control outbreaks.
- Surveillance (case-reporting of yellow fever is universally required by the International Health Regulations, 2005).
- Vector control.

Immunization

In endemic areas, immunization should be provided routinely through incorporation of yellow fever vaccine in routine child immunization programmes and mass preventive campaigns. Yellow fever vaccine is included in the childhood vaccination programme in Côte d'Ivoire; vaccination coverage among the target population in 2006 was estimated at 67% according to official country estimates (*1*).

Vaccination is not recommended for symptomatic HIV-infected persons, other immunocompromised individuals or pregnant women.

The Global Alliance for Vaccines and Immunization (GAVI) agreed in 2000 to support yellow fever vaccine introduction in all GAVI-eligible countries at risk of yellow-fever outbreaks.

Epidemic control

An infected mosquito spreads yellow fever when it bites non-infected humans. When mosquitoes carry the virus from person to person, 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.

Under epidemic conditions, the following must be implemented:

- Mass vaccination with yellow-fever vaccine;
- Emergency mosquito-control measures:

- Eliminating potential mosquito breeding sites (the most important mosquito-control measure for yellow-fever control);
- Spraying to kill adult mosquitoes (less important because of small impact);
- Use of insecticide-treated bednets for hospitalized cases.

In sylvatic (jungle) yellow fever, nonimmunized persons should avoid tracts of jungle where infection has been localized.

References

1. *WHO vaccine-preventable diseases: monitoring system. 2007 Global summary*. Geneva, World Health Organization, 2007 (http://whqlibdoc.who.int/hq/2007/WHO_IVB_2007_eng.pdf, accessed June 2009).

CD-WGE technical focal point: Department of Global Alert and Response (GAR)

PART III

List of tables and figures

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PART IV

Annexes

ANNEX 1. KEY NATIONAL INDICATORS AND GENERAL INFORMATION FOR CÔTE D'IVOIRE

Key health indicators

Indicator	Data	Source (year)*
Last census	1998	UN (2008)
Estimated population	15 366 672	UN (2008)
Children under age 5 years who are underweight for age (%)	16.7	MICS (2006), converted to WHO standards
Children under age 5 years who are underweight for age, severe (%)	5.4	MICS (2006), converted to WHO standards
Life expectancy at birth, males/females (years)	42/47	WHO (2005)
Neonatal mortality rate per 1000 live births	65	UNICEF (2000)
Infant (age < 1 year) mortality rate per 1000 live births	118	WHO (2005)
Children under age 5 years, mortality per 1000 live births	196	WHO (2005)
Children under age 5 years, mortality ranking	26 of 189	UNICEF (2008)
Low-birth-weight infants (%)	17	UNICEF (1996–2005)
Children under age 5 years, wasting (%)	8.6	MICS (2006), converted to WHO standards
Children under age 5 years, wasting, severe (%)	2.9	MICS (2006), converted to WHO standards
Children under age 5 years, stunted for age (%)	40.1	MICS (2006), converted to WHO standards
Children under age 5 years, stunted for age, severe (%)	19.5	MICS (2006), converted to WHO standards
Births attended by skilled health staff (%)	62.5	WHO (2000)
Maternal mortality ratio (per 100 000 live births)	810	WHO (2006)
Population with access to improved water source (%)	81 (66 rural)	WHO/UNICEF JMP (2008; data for 2006)
Population with sustainable access to improved sanitation (%)	24 (12 rural)	WHO/UNICEF JMP (2008; data for 2006)
Physicians per 10 000 population	12	WHO (2004)
Nurses/midwives per 10 000 population	46/14	WHO (2004)
Human Development Index Ranking	166 of 177	HDR (2007/2008)
Adult literacy	92 of 108	HDR (2007/2008)
Population living on less than US$ 1 per day (%)	14.8	WHO (2002)
Human Poverty Index	92 of 108	HDR (2007/2008)

Sources:

Information was extracted from databases of the following organizations:

United Nations (UN): http://unstats.un.org/

Multiple indicator cluster surveys (MICS), converted to WHO standards: http://www.who.int/nutgrowthdb/database/countries/who_standards/civ.pdf

World Health Organization (WHO): http://www.who.int/hac/crises/civ/en/

United Nations Children's Fund (UNICEF): Information by country, *http://www.unicef.org/infobycountry/cotedivoire.html*

WHO/UNICEF Joint Monitoring Programme (JMP) for water supply and sanitation: http://www.wssinfo.org/

Human development reports (HDR) from the United Nations Development Programme: http://hdr.undp.org/en/statistics/

Progress towards the Millennium Development Goals

Millenium Development Goals	1990	1995	2000	2006
Goal 1: Eradicate extreme poverty and hunger				
Malnutrition prevalence, weight for age (% of children under age 5 years)	..	..	18	..
Goal 3: Promote gender equality and empower women				
Ratio of young literate females to males (% aged 15–24 years)	63	..	74	..
Goal 4: Reduce child mortality				
Immunization, measles (% of children aged 12–23 months)	56	57	73	73
Mortality rate, infant (per 1000 live births)	105	100	95	90
Mortality rate, children under age 5 years (per 1000)	153	144	136	127
Goal 5: Improve maternal health				
Births attended by skilled health staff (% of total)	..	45	63	57
Contraceptive prevalence (% of women aged 15–49 years)	..	11	15	13
Maternal mortality ratio (modeled estimate, per 100 000 live births)	..	..	..	810
Pregnant women receiving prenatal care (%)	..	83	88	85
Goal 6: Combat HIV/AIDS, malaria, and other diseases				
Children with fever receiving antimalarial drugs (% of children under age 5 years with fever)	..	..	58	36
Condom use, in female population aged 15–24 years (% of females aged 15–24 years)	..	11	..	..
Condom use, in male population aged 15–24 years (% of males aged 15–24 years)	..	41	..	..
Incidence of tuberculosis (per 100 000 population)	168	251	364	420
Prevalence of HIV, female (% aged 15-24 years)	..	..	..	5.1
Prevalence of HIV, total (% of population aged 15–49 years)	..	..	..	7
Tuberculosis cases detected under DOTS (%)	..	50	32	37

Goal 7: Ensure environmental sustainability				
Improved sanitation facilities (% of population with access)	21	27	33	37
Improved water source (% of population with access)	69	76	83	84
Other				
Life expectancy at birth, total (years)	53	50	47	48
Literacy rate, adult total (% of people aged 15 years and above)	34	..	49	..
Population, total (millions)	12.8	15	17	18.9

Source: World Bank. Global Data Monitoring Information System (http://ddp-ext.worldbank.org/ext/GMIS/gdmis.do?siteId=2&menuId=LNAV01HOME3, accessed 26 August 2008).

DOTS, directly observed treatment, short-course

Health systems structure

Côte d'Ivoire's health system has a pyramid-shaped structure with two branches (administrative and operational). The system of health-care administration is divided into three levels:

- The central level includes the Minister's Cabinet and the General and Central Departments. It is responsible for creating health-care policy and providing strategic direction at various levels of the health-care system.
- The regional level includes 19 regional departments. It is responsible for coordinating health-care activities at the district level. It also oversees district activities.
- The peripheral level is represented by 79 health-care districts, which are the primary operational units. They are run by the district management teams. The districts cover urban and rural areas throughout the country.

Operationally, the health-care system is also divided into three levels (Decree 96-876 of 25 November 1996):

- Primary level: rural health centres, urban health centres, urban health-training and community-based urban health-training centres, specialized urban health centres including school and university health centres and antituberculosis centres.
- Secondary level: general hospitals, regional general hospitals, specialized hospital centres and the Bingerville psychiatric hospital.
- Tertiary level:
  - Specialized institutes: Institut Raoul Follereau d'Adzopé (IRF), Institut National de la Santé Publique (INSP), Institut National d'Hygiène Publique (INHP), Laboratoire National de Santé Publique (LNSP), Institut de Cardiologie d'Abidjan (ICA), Public Health Pharmacy (PHP), le Service d'Aide Médicale d'Urgence (SAMU), National Blood Transfusion Centre (NBTC), Institut National de Formation des Agents de Santé (INFAS); and
  - University hospital centres in Abidjan (Treichville, Cocody and Yopougon), and Bouaké.

Private, faith-based and other associations are integrated into this health-care system, at the primary level with infirmaries and drug-purchasing sites and the secondary and tertiary levels with medical offices, clinics, private hospitals, pharmacies and laboratories.

Source: *Application of the CCM-Côte d'Ivoire to the Sixth Round of the Global Fund.* (http://www.theglobalfund.org/grantdocuments/6CIVM_1302_0_full.pdf; accessed August 2009).

See also annual Appeal documents available at Humanitarian Appeal (http://www.humanitarianappeal.net; accessed August 2009).

History of the humanitarian crisis

1893 Côte d'Ivoire made into a colony of France

1960 Independence declared under President Felix Houphouet-Boigny. He holds power until he dies in 1993.

1993 Henri Konan Bedie becomes president after the death of Houphouet-Boigny.

1999 Bedie overthrown in bloodless military coup led by Robert Guei. Bedie flees to France.

2000 Laurent Gbagbo is proclaimed president.

2002 Mutiny in Abidjan by soldiers unhappy at being demobilized grows into full-scale rebellion, with Ivory Coast Patriotic Movement rebels seizing control of the north.

2003 Power sharing agreement.

2004 Deadly clashes during crackdown on opposition rally against President Gbagbo in Abidjan. United Nations (UN) peacekeeping force deployed. Later that year, Ivorian air force attacks rebels; French forces enter the fray after nine of their soldiers are killed in an air strike. Violent anti-French protests ensue. UN imposes arms embargo.

2005 Planned elections are shelved as President Gbagbo invokes a law that he says allows him to stay in power. The UN extends his mandate for a further year. Charles Konan Banny is nominated as prime minister by mediators.

2007 After a number of failed attempts at peace-brokering, the Ouagadougou Peace Agreement (OPA) between the Ivorian Government and the Forces Nouvelles, mediated by Burkina Faso, is signed on 4 March 2007. Under the deal, New Forces leader Guillaume Soro is named as prime minister. In April, President Gbagbo declares "the war is over" between his government and northern rebels, as the two sides move to dismantle the military buffer zone. Within days, aid workers report an increase in violence. UN Security Council votes to maintain sanctions for another year; UN renews mandate of 8000 peacekeepers for 6 months to ensure polls are held by the middle of the year.

2008 April – President Gbagbo cancels custom duties after a second day of violent protests against rising food costs. Date of long-awaited presidential elections deferred from June to the end of November.

2008 May – Former rebels who still control the northern half of the country begin disarming.

Source: *Timeline: Ivory Coast*. BBC News. Dated 5 November 2008 (http://news.bbc.co.uk/2/hi/africa/1043106.stm, accessed May 2009).

Further reading

Republic of Côte d'Ivoire. Humanitarian country profile. IRIN (UN Office for the Coordination of Humanitarian Affairs). Dated February 2007 (http://www.irinnews.org/country.aspx?CountryCode=CI&RegionCode=WA, accessed May 2009).

Map resources

Internally displaced persons

Internal Displacement Monitoring Centre (IDMC):
http://www.internal-displacement.org/

Refugees

Office of the United Nations High Commissioner for Refugees (UNHCR):
http://www.unhcr.org/cgi-bin/texis/vtx/country?iso=civ

National immunization schedule and coverage

National immunization schedule

Vaccine	Age
BCG	Birth;
DTPHep	6, 10, 14 weeks;
DTPHibHep	6, 10, 14 weeks; [From July 2008]
Measles	9 months;
Oral polio vaccine	Birth; 6, 10, 14 weeks;
Tetanus toxoid	Pregnant women first contact; +1, +6 months; +1, +1 year;
Yellow fever	9 months

BCG, Bacille Calmette–Guérin; ; D, diphtheria; Hep, hepatitis B; Hib, *Haemophilus influenzae* B; P, pertussis (acellular or whole-cell); T, tetanus.

Cases reported to WHO in 2007

Communicable disease	Reported cases in 2007
Diptheria	Not available
Japanese encephalitis	–[a]
Measles	5
Mumps	–[a]
Pertussis	–[a]
Polio	0
Rubella	48
Rubella, congenital rubella syndrome	–[a]
Tetanus, neonatal	31
Tetanus, total	31
Yellow fever	0

Source: *WHO vaccine-preventable diseases: monitoring system. 2007 Global summary*. Geneva, World Health Organization, 2007 (http://whqlibdoc.who.int/hq/2007/WHO_IVB_2007_eng.pdf, accessed June 2009).

[a] Data not available.

Immunization coverage

Vaccine	Official country estimates (%), 2007	WHO/UNICEF estimates (%), 2006
BCG	94	77
DTP, first dose	93	95
DTP, third dose	76	77
Hepatitis B, first dose	–[a]	–[a]
Hepatitis B, third dose	76	77
Hib, third dose	–[a]	–[a]
Japanese encephalitis	–[a]	–[a]
Measles-containing vaccine	67	73
Measles-containing vaccine, second dose	–[a]	–[a]
Polio, third dose	75	76
Tetanus toxoid, second and subsequent dose	45	—[a]
Tetanus, protection at birth	–[a]	52
Vitamin A doses received by age 12 months	6	–[a]
Yellow fever	68	–[a]

Source: WHO vaccine-preventable diseases: monitoring system. 2007 Global summary. Geneva, World Health Organization, 2007 (http://whqlibdoc.who.int/hq/2007/WHO_IVB_2007_eng.pdf, accessed June 2009).

BCG, Bacille Calmette–Guérin; D, diphtheria; Hep, hepatitis B; Hib, *Haemophilus influenzae* B; P, pertussis (acellular or whole-cell); T, tetanus.

[a] Data not available.

ANNEX 2. STEPS IN OUTBREAK MANAGEMENT

Preparation

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

Detection

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

- Acute watery diarrhoea in children over the age of 5 years
- Bloody diarrhoea
- Suspected cholera
- Measles
- Meningitis
- Acute haemorrhagic fever syndrome
- Acute jaundice syndrome
- Suspected poliomyelitis (acute flaccid paralysis)
- Cluster of deaths of unknown origin.

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

[a] Diseases/syndromes in list to be modified according to country profile.

Response

Confirmation

The lead health agency should investigate reported cases to confirm the outbreak situation – number of cases higher than that expected for the same period of year and population. Clinical specimens will be sent for testing.

The lead health agency should activate an outbreak-control team with membership from relevant organizations: Ministry of Health, WHO and other United Nations organizations, nongovernmental organizations in the fields of health and water and sanitation, veterinary experts.

Investigation

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

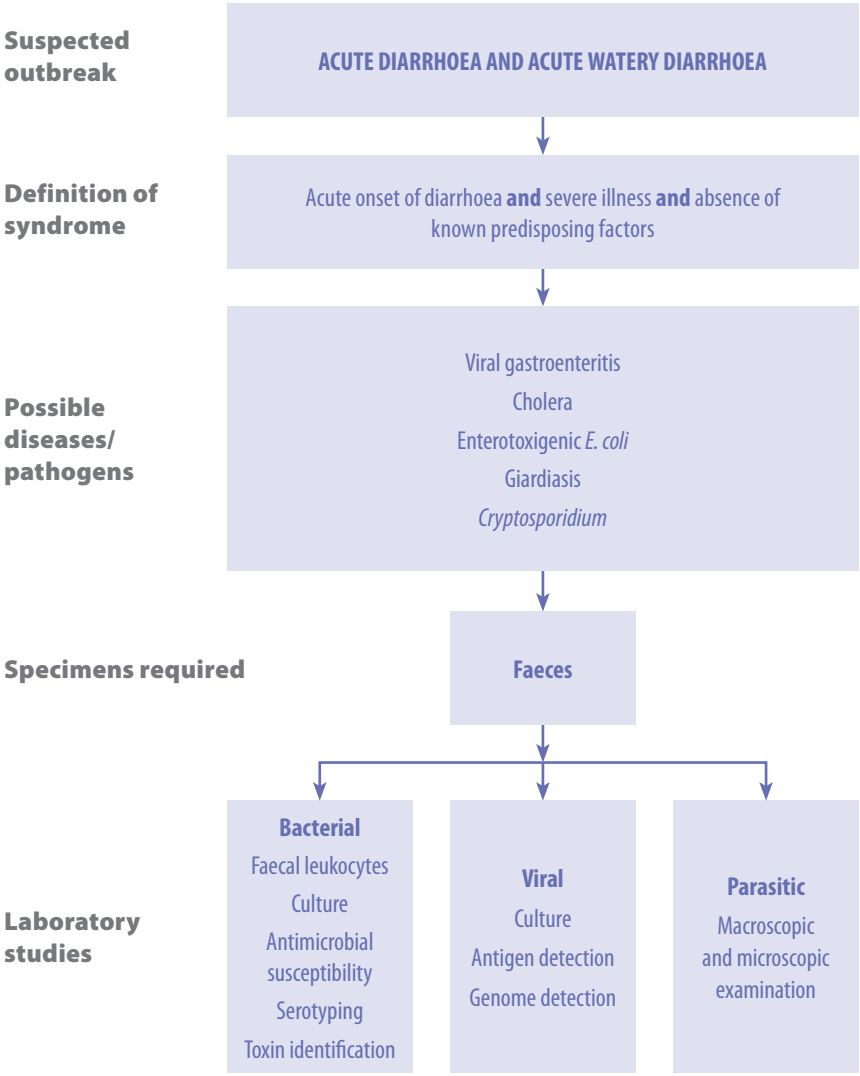
Control

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

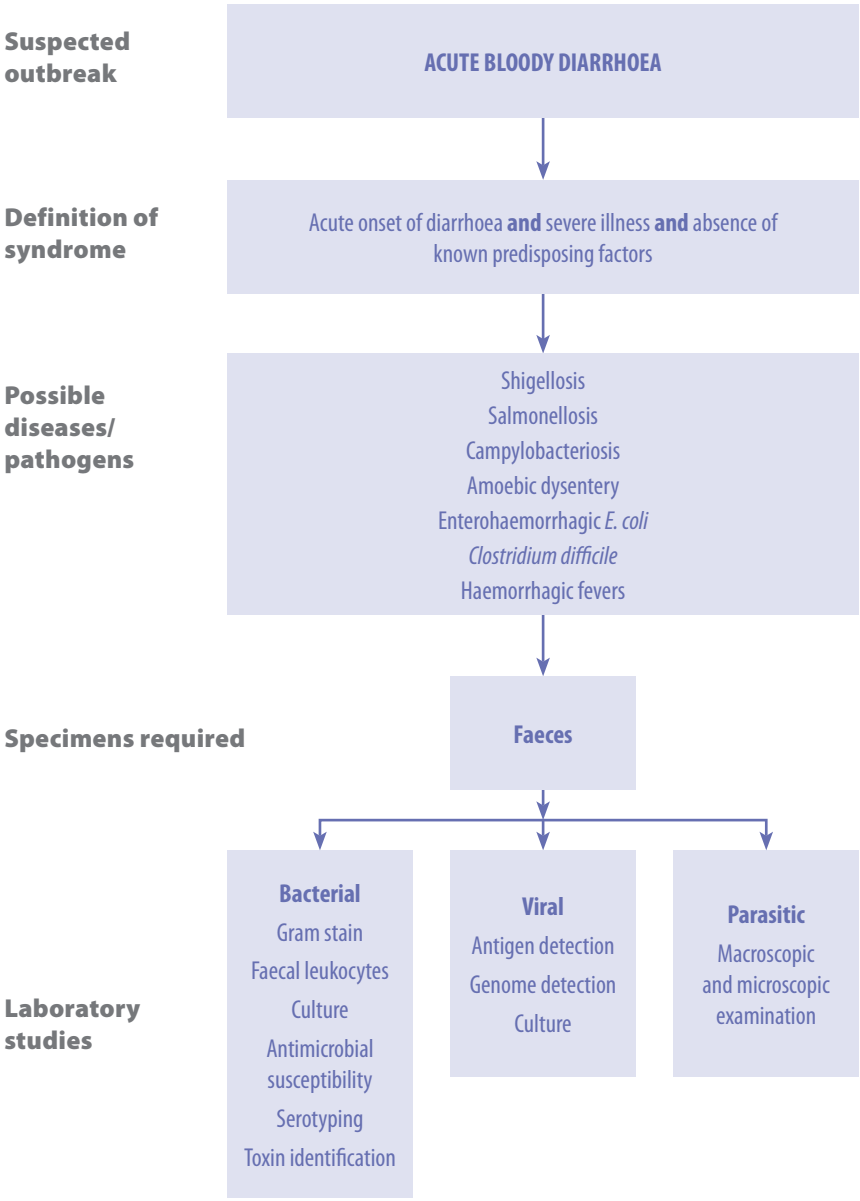
Evaluation

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

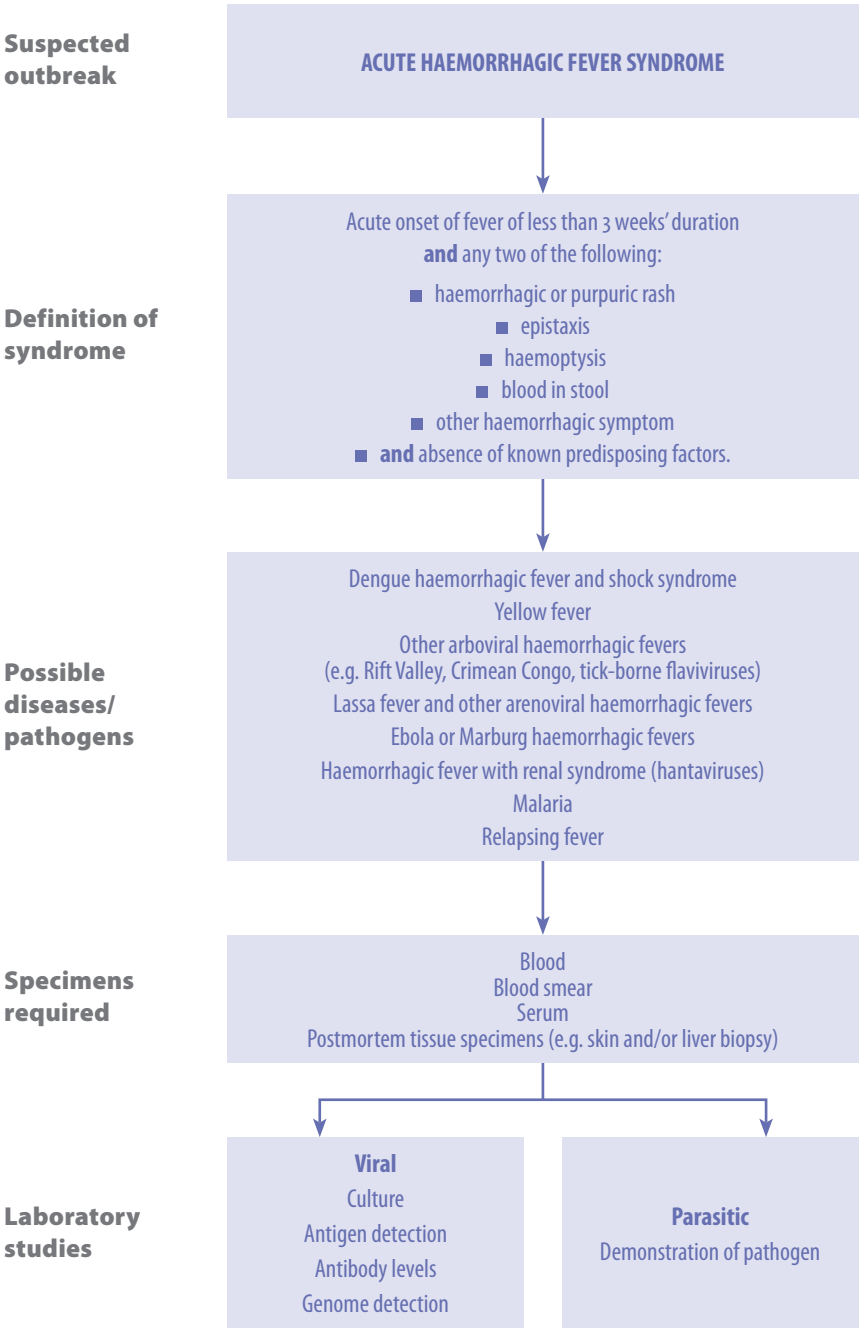
ANNEX 3. FLOWCHARTS FOR THE DIAGNOSIS OF COMMUNICABLE DISEASES

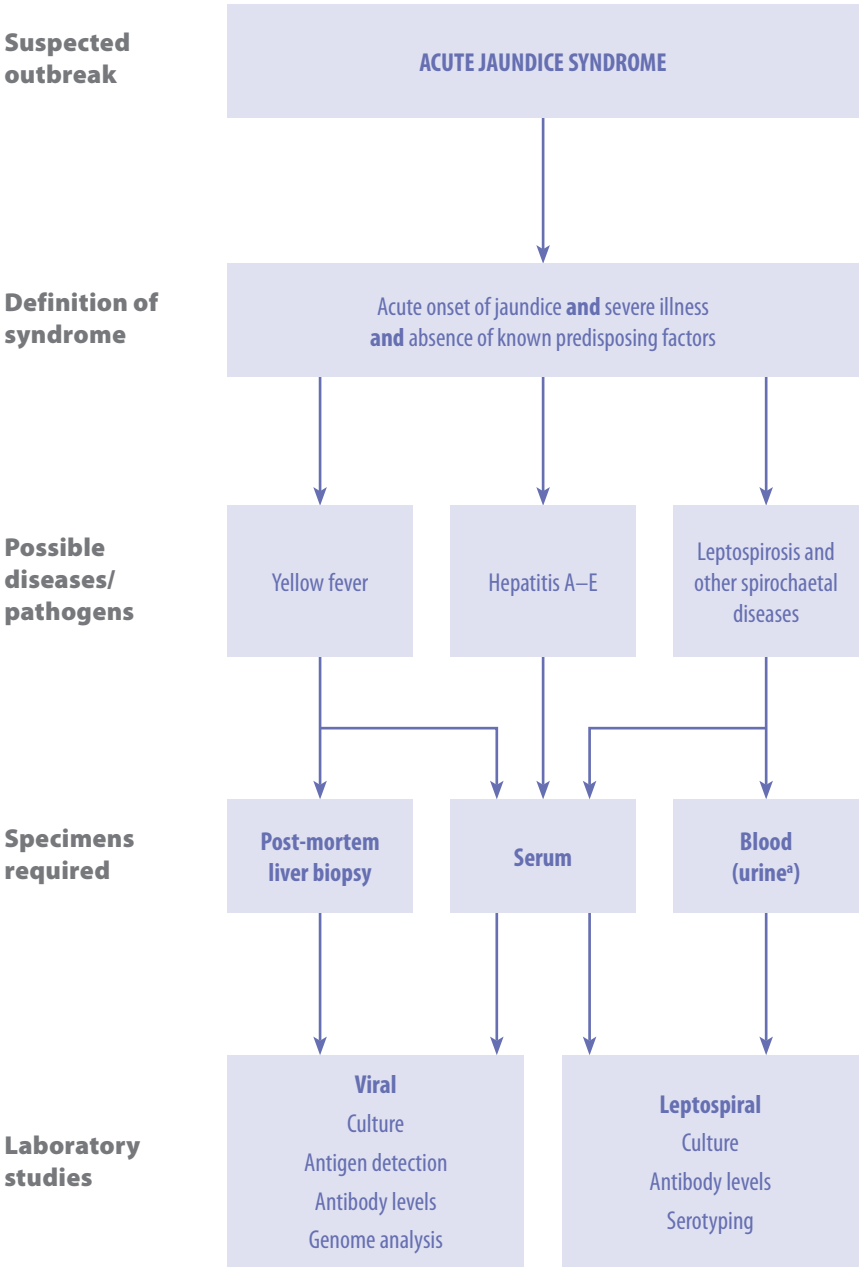


Ebola and other haemorrhagic fevers may initially present as bloody diarrhoea. If such an etiology is suspected, refer to "Acute haemorrhagic fever syndrome" for appropriate specimen-collection guidelines.

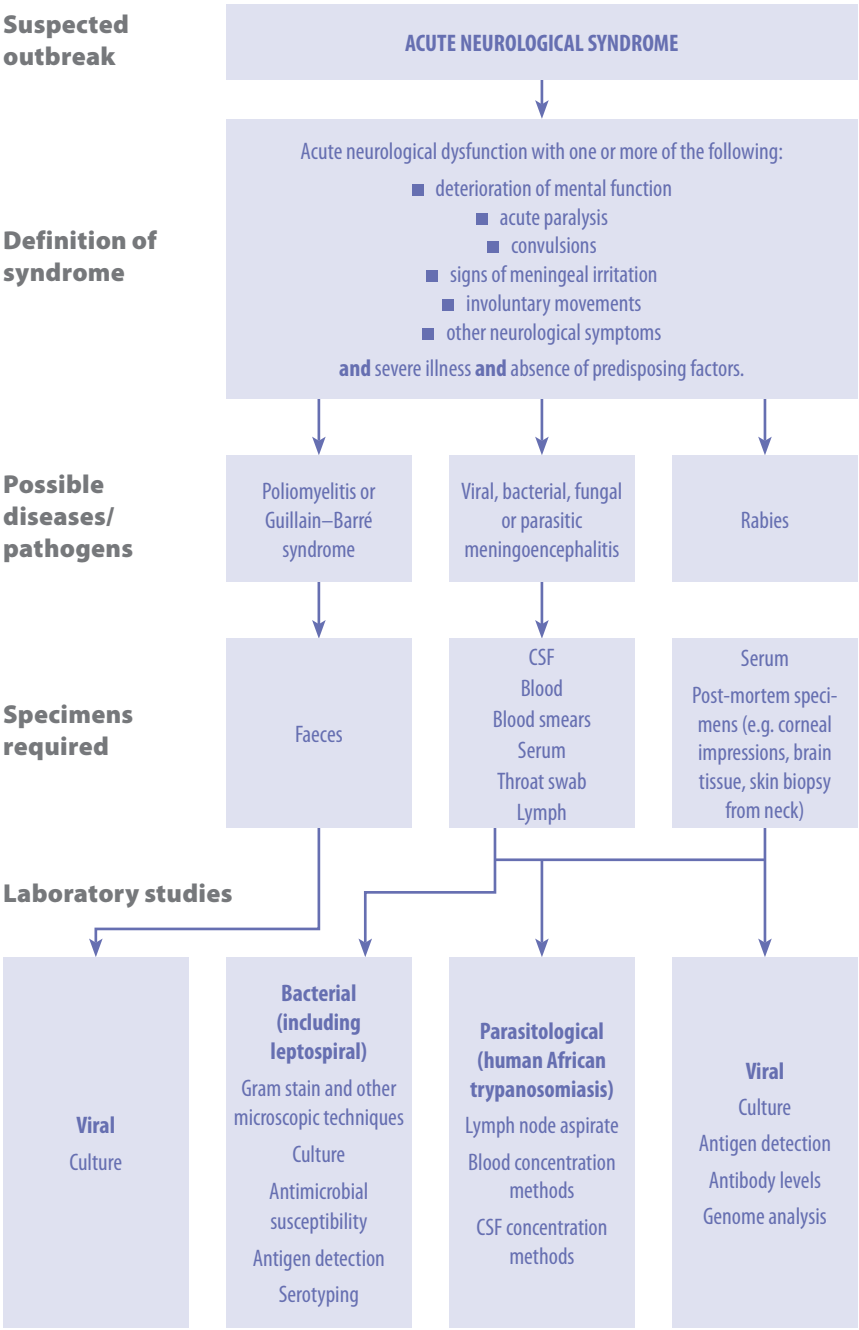


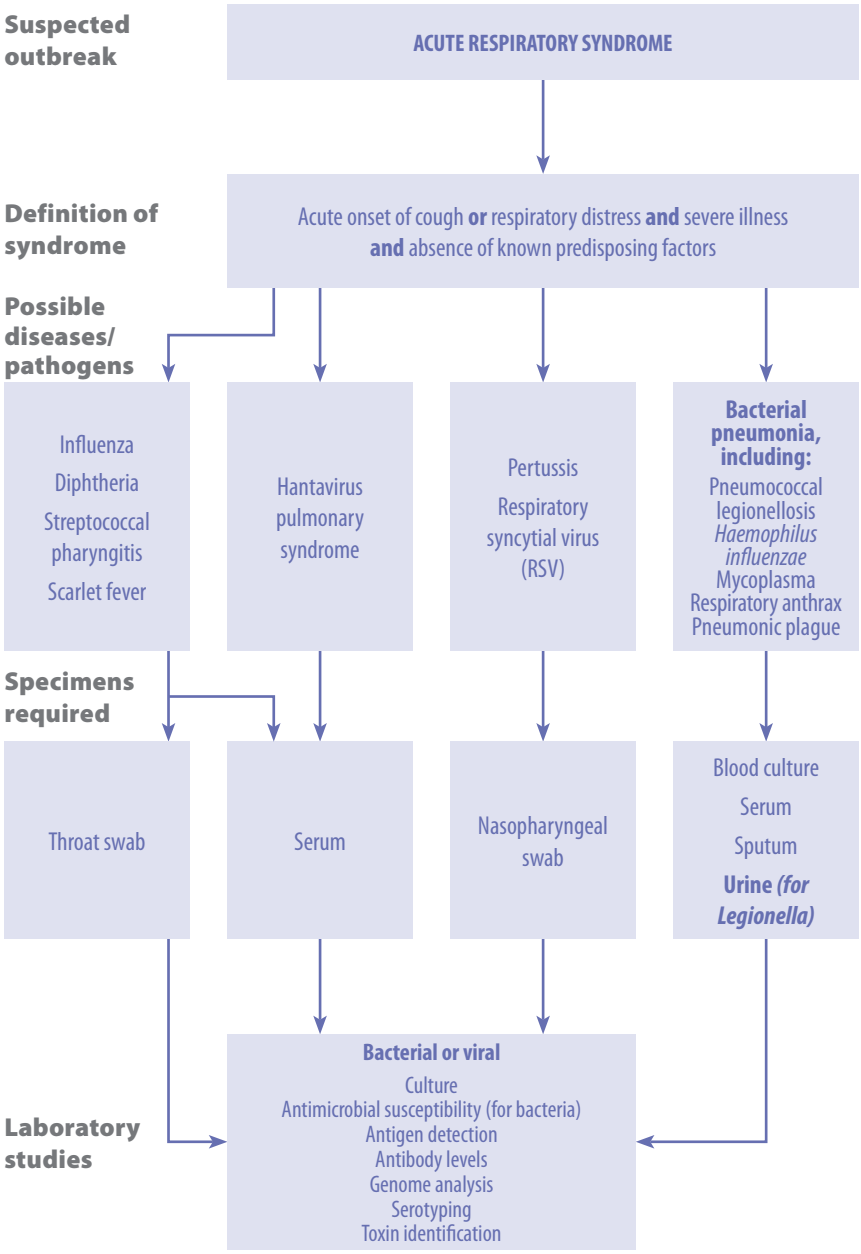
Ebola and other haemorrhagic fevers may initially present as bloody diarrhoea. If such an etiology is suspected, refer to "Acute haemorrhagic fever syndrome" for appropriate specimen-collection guidelines.


Suspected outbreak
ACUTE HAEMORRHAGIC FEVER SYNDROME
Definition of syndrome
Acute onset of fever of less than 3 weeks' duration
and any two of the following:
haemorrhagic or purpuric rash
epistaxis
haemoptysis
blood in stool
other haemorrhagic symptom
and absence of known predisposing factors.
Possible diseases/ pathogens
Dengue haemorrhagic fever and shock syndrome
Yellow fever
Other arboviral haemorrhagic fevers
(e.g. Rift Valley, Crimean Congo, tick-borne flaviviruses)
Lassa fever and other arenoviral haemorrhagic fevers
Ebola or Marburg haemorrhagic fevers
Haemorrhagic fever with renal syndrome (hantaviruses)
Malaria
Relapsing fever
Specimens required
Blood
Blood smear
Serum
Postmortem tissue specimens (e.g. skin and/or liver biopsy)
Laboratory studies
Viral
Culture
Antigen detection
Antibody levels
Genome detection
Parasitic
Demonstration of pathogen



[a] Requires specialized media and handling procedures.


Suspected outbreak
ACUTE NEUROLOGICAL SYNDROME
Definition of syndrome
Acute neurological dysfunction with one or more of the following:
deterioration of mental function
acute paralysis
convulsions
signs of meningeal irritation
involuntary movements
other neurological symptoms
and severe illness and absence of predisposing factors.
Possible diseases/ pathogens
Poliomyelitis or Guillain–Barré syndrome
Viral, bacterial, fungal or parasitic meningoencephalitis
Rabies
Specimens required
Faeces
CSF
Blood
Blood smears
Serum
Throat swab
Lymph
Serum
Post-mortem specimens (e.g. corneal impressions, brain tissue, skin biopsy from neck)
Laboratory studies
Viral
Culture
Bacterial (including leptospiral)
Gram stain and other microscopic techniques
Culture
Antimicrobial susceptibility
Antigen detection
Serotyping
Parasitological (human African trypanosomiasis)
Lymph node aspirate
Blood concentration methods
CSF concentration methods
Viral
Culture
Antigen detection
Antibody levels
Genome analysis



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

ANNEX 4. SAFE WATER AND SANITATION

Safe water

The minimum emergency requirement is 20 litres/person per day. The following are effective methods of obtaining safe water.

Household filtration

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

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, which can be done by adding one of the following products to 1 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%)	110 ml

The stock solution must be stored in a closed container, in a cool dark place and used within 1 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. The free residual chlorine level after 30 minutes should be between

0.2 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 appropriately.

If the water is cloudy, it must be either filtered before chlorination or boiled vigorously. Chlorination of turbid water may not make it safe.

Boiling

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

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 disease transmission. Sanitary systems that are appropriate for the local conditions should be constructed with the cooperation of the community. The minimum emergency standard is one latrine for every 20 people.

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 handwashing with soap or ash after any contact with excreta. The disposal of children's excreta in latrines needs to be emphasized.

Further reading

Franceys R, Pickford J, Reed R. *A guide to the development of on-site sanitation*. Geneva, World Health Organization, 1992.

Wisner B, Adams J, eds. *Environmental health in emergencies and disasters: a practical guide*. Geneva, World Health Organization, 2002 (http://www.who.int/water_sanitation_health/hygiene/emergencies/emergencies 2002/en/, accessed June 2009).

Fact sheets on environmental sanitation. Geneva, World Health Organization (http://www.who.int/water_sanitation_health/hygiene/emergencies/envsanfactsheets/en/, accessed June 2009).

ANNEX 5. INJECTION SAFETY

Analysis of data collected as part of the Comparative Risk Assessment component of the Global Burden of Disease study (*1*) suggests that the WHO African Region 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 viral hepatitis B, hepatitis C and HIV that are attributable to unsafe injection practices is 10.9%, 16.4% and 2.5%, respectively. Thus, in all relief efforts to assist the population and the displaced populations 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;
- Demand 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; discard them immediately in a sharps box to prevent needle-stick injury;
- Dispose of full sharps boxes by open-air incineration and burial.

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 indicator for quality of 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; and
  - 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; or
  - used syringes and needles that are not disposed of properly.

References

1. *The global burden of disease: 2004 update.* Geneva, World Health Organization, 2008 (http://www.who.int/healthinfo/global_burden_disease/2004_report_update/en/index.html; accessed August 2009).

ANNEX 6. WHO FACT SHEETS AND INFORMATION SOURCES

WHO Fact Sheets

Title	Publication No./Date
African trypanosomiasis (sleeping sickness)	Fact Sheet No. 259 revised August 2006 http://www.who.int/mediacentre/factsheets/fs259/en/
Anthrax	Fact sheet No. 264 October 2001 http://www.who.int/mediacentre/factsheets/fs264/en/
Cholera	Fact sheet No. 107 revised November 2008 http://www.who.int/mediacentre/factsheets/fs107/en
Dengue	Fact sheet No. 117 revised March 2009 http://www.who.int/mediacentre/factsheets/fs117/en/
Diphtheria	Fact sheet No. 89 revised December 2000 http://www.who.int/mediacentre/factsheets/fs089/en/
Ebola	Fact sheet No. 103 revised December 2008 http://www.who.int/mediacentre/factsheets/fs103/en/
Food safety and food-borne illness	Fact sheet No. 237 revised March 2007 http://www.who.int/mediacentre/factsheets/fs237/en/
Food safety in emergencies	January 2005 http://www.who.int/foodsafety/foodborne_disease/emergency/en/
Hepatitis B	Fact sheet No. 204 revised August 2008; no date on current link http://www.who.int/mediacentre/factsheets/fs204/en/
Hepatitis C	Fact sheet No. 164 (being updated) http://www.who.int/mediacentre/factsheets/fs164/en/
Hepatitis E	Fact sheet No. 280 revised January 2005 http://www.who.int/mediacentre/factsheets/fs280/en/index.html
Influenza	Fact sheet No. 211 April 2009 http://www.who.int/mediacentre/factsheets/fs211/en/
Injection safety	Fact sheet No. 231 revised October 2006 http://www.who.int/mediacentre/factsheets/fs231/en/
Lassa fever	Fact sheet No. 179 revised April 2005 http://www.who.int/mediacentre/factsheets/fs179/en/index.html
Leprosy	Fact sheet No. 101 revised February 2010 http://www.who.int/mediacentre/factsheets/fs101/en/index.html

Lymphatic filiariasis	Fact sheet No. 102 Revised September 2000 http://www.who.int/mediacentre/factsheets/fs102/en/index.html
Malaria	Fact sheet No. 94 January 2009 http://www.who.int/mediacentre/factsheets/fs094/en/
Marburg haemorrhagic fever	Fact sheet July 2008 http://www.who.int/mediacentre/factsheets/fs_marburg/en/index.html
Measles	Fact sheet No. 286 revised December 2009 http://www.who.int/mediacentre/factsheets/fs286/en/
Meningitis	Fact sheet No. 141 revised February 2010 http://www.who.int/mediacentre/factsheets/fs141/en/
Plague	Fact sheet No. 267 revised February 2005 http://www.who.int/mediacentre/factsheets/fs267/en/
Poliomyelitis	Fact sheet No. 114 updated January 2008 http://www.who.int/mediacentre/factsheets/fs114/en/
Rabies	Fact sheet No. 99 revised December 2008 http://www.who.int/mediacentre/factsheets/fs099/en/
Salmonella - drug resistant	Fact sheet No. 139 revised April 2005 http://www.who.int/mediacentre/factsheets/fs139/en/index.html
Schistosomiasis	Fact sheet No. 115 revised February 2010 http://www.who.int/mediacentre/factsheets/fs115/en/
Smallpox	Smallpox http://www.who.int/mediacentre/factsheets/smallpox/en/
Tuberculosis	Fact sheet No. 104 revised March 2007 http://www.who.int/mediacentre/factsheets/fs104/en/ Stop TB Fact sheet http://www.who.int/tb/publications/2008/factsheet_april08.pdf
Typhoid fever and paratyphoid fever	Water-related diseases http://www.who.int/water_sanitation_health/diseases/typhoid/en/
Water, sanitation and health	Introduction to fact sheets on water sanitation, and health http://www.who.int/water_sanitation_health/hygiene/emergencies/envsanfactsheets/en/print.html
World Health Organization	About WHO http://www.who.int/about/en/
Yellow fever	Fact sheet No. 100 revised December 2009 http://www.who.int/mediacentre/factsheets/fs100/en/

WHO Information Sources

Acute lower respiratory tract infections	Acute respiratory tract infections in children http://www.who.int/fch/depts/cah/resp_infections/en/
African trypanosomiasis	Human African trypanosomiasis (sleeping sickness) http://www.who.int/trypanosomiasis_african/en/
Bacillary dysentery (Shigellosis) (see also diarrhoeal diseases)	Guidelines for the control of epidemics due to Shigella dysenteriae type 1 http://whqlibdoc.who.int/publications/2005/9241592330.pdf
Child health in emergencies	Acute respiratory tract infections in children http://www.who.int/fch/depts/cah/resp_infections/en/ Emergencies documents http://www.who.int/child_adolescent_health/documents/emergencies/en/index.html Home treatment for children with severe pneumonia just as effective as hospital http://www.who.int/child_adolescent_health/news/2008/09_01/en/index.html IMCI chart booklet (WHO/UNICEF, 2008) http://whqlibdoc.who.int/publications/2008/ 9789241597289_eng.pdf IMCI for high HIV settings (WHO, 2006) http://whqlibdoc.who.int/publications/2006/ 9789241594370.cb_eng.pdf Operational guidance on infant feeding in emergencies (IFE, 2007) http://www.ennonline.net/ife/view.aspx?resid=6 Paediatric HIV and treatment of children living with HIV http://www.who.int/hiv/topics/paediatric/en/index.html Pocket book of hospital care for children: guidelines for the management of common illnesses with limited resources (WHO,2005) http://www.who.int/child_adolescent_health/documents/9241546700/en/index.html Technical updates of the guidelines on IMCI (WHO, 2005) http://www.who.int/child_adolescent_health/documents/9241593482/en/index.html The treatment of diarrhoea: A manual for physicians and other senior health workers (WHO, 2005) http://www.who.int/entity/child_adolescent_health/documents/9241593180/en/
Cholera (see also Diarrhoeal diseases)	Acute diarrhoeal diseases in complex emergencies: critical steps. http://www.who.int/cholera/publications/critical_steps/ Cholera and other epidemic diarrhoeal diseases control. Technical cards on environmental sanitation (WHO, 1997) http://www.who.int/csr/resources/publications/cholera/WHO_EMC_DIS_97_6/en/ Cholera outbreak: assessing the outbreak response and improving preparedness http://www.who.int/cholera/publications/cholera_outbreak/ Cholera: prevention and control http://www.who.int/topics/cholera/control/en/index.html http://www.who.int/cholera/technical/DiarrhoealDiseaseKits/ en/index.html First steps for managing an outbreak of acute diarrhoea. http://www.who.int/cholera/publications/first_steps/

	Joint WHO/UNICEF statement for cholera vaccine use in tsunami-affected areas http://www.who.int/cholera/tsunami_choleravaccine/en/index.html Laboratory methods for the diagnosis of epidemic dysentery and cholera (CDC, 1999) http://www.cdc.gov/ncidod/dbmd/diseaseinfo/cholera/top.pdf Oral cholera vaccine use in complex emergencies: What next? Report of a WHO meeting. Cairo, Egypt, 14–16 December 2005. (WHO, 2005) http://www.who.int/cholera/publications/cholera_vaccines_emergencies_2005.pdf
Communicable disease control in emergencies	Communicable disease control in emergencies: a field manual (WHO, 2005) http://whqlibdoc.who.int/publications/2005/9241546166_eng.pdf
Dengue	Guidelines for conducting a review of a national dengue prevention and control programme (WHO 2005) WHO/CDS/CPE/PVC/2005.13 Parks W, Lloyd LS. Planning social mobilization and communication for dengue fever prevention and control: a step-by-step guide (WHO 2005) http://www.who.int/tdr/publications/publications/pdf/planning_dengue.pdf Report of the Scientific Working Group on Dengue (WHO 2006) http://www.who.int/tdr/publications/publications/swg_dengue_2.htm Space spray application of insecticides for vector and pest control: a practitioner's guide (WHO 2003) http://whqlibdoc.who.int/hq/2003/WHO_CDS_WHOPES_GCDPP_2003.5.pdf Global Strategic Framework for Integrated Vector Management (WHO 2004) http://whqlibdoc.who.int/hq/2004/WHO_CDS_CPE_PVC_2004_10.pdf Dengue haemorrhagic fever: diagnosis, treatment, prevention and control, 2nd ed. (WHO 1997) http://www.who.int/csr/resources/publications/dengue Denguepublication/en/print.html [forthcoming - Dengue: Guidelines for diagnosis, treatment, prevention and control. Third edition (WHO 2009)] Equipment for vector control: Specification guidelines. (WHO 2006) http://whqlibdoc.who.int/hq/2006/WHO_CDS_NTD_WHOPES_2006.5_eng.pdf Decision-making for the judicious use of insecticides (WHO 2004) http://whqlibdoc.who.int/hq/2004/WHO_CDS_WHOPES_2004.9b.pdf
Diarrhoeal diseases (see also cholera)	Acute diarrhoeal diseases in complex emergencies: critical steps. http://www.who.int/cholera/publications/critical_steps/ First steps for managing an outbreak of acute diarrhoea. http://www.who.int/cholera/publications/first_steps/ Interagency diarrhoeal disease kits - information note. (WHO, 2006) http://www.who.int/topics/cholera/materials/en/index.html
Diphtheria	WHO Diphtheria vaccine position paper http://www.who.int/immunisation/wer8103Diphtheria_Jan06_position_paper.pdf
Dracunculiasis (Guinea worm)	WHO Weblink http://www.who.int/dracunculiasis/en/
Drug donations	Guidelines for drug donations (WHO, 1999) http://whqlibdoc.who.int/hq/1999/WHO_EDM_PAR_99.4.pdf

Food safety, food-borne disease outbreaks	Ensuring food safety in the aftermath of natural disasters http://www.who.int/foodsafety/foodborne_disease/emergency/en/ Five keys to safer food : simple advice to consumers and food handlers http://www.who.int/foodsafety/consumer/5keys/en/index.html Foodborne disease outbreaks: guidelines for investigation and control http://www.who.int/foodsafety/publications/foodborne_disease/fdbmanual/en/ Guideline for the safe preparation, storage and handling of powdered infant formula (WHO, 2007) http://www.who.int/foodsafety/publications/micro/pif2007/en/index.html
Gender and gender-based violence	Interagency Standing Committee (IASC) Guidelines for gender-based violence interventions in humanitarian settings (IASC, 2005) http://www.humanitarianinfo.org/iasc/content/products/docs/tfgender_GBV Guidelines2005.pdf Interagency Standing Committee (IASC) Gender handbook in humanitarian action women, girls, boys and men different needs – equal opportunities (IASC, 2006) http://www.humanitarianinfo.org/iasc/content/documents/subsidi/tf_gender/IASC%20Gender%20Handbook%20(Feb%202007).pdf UNHCR/WHO Clinical management of rape survivors: Developing protocols for use with refugees and internally displaced persons, revised edition (UNHCR.WHO 2004) http://www.who.int/reproductive-health/publications/clinical_mngt_rapesurvivors/
Hepatitis	Hepatitis A http://www.who.int/csr/disease/hepatitis/whocdscsredc2007/en/ Hepatitis E http://www.who.int/csr/disease/hepatitis/whocdscsredc200112/en/ http://www.who.int/mediacentre/factsheets/fs280/en/
HIV/AIDS	Antiretroviral drugs for treating pregnant women and preventing HIV infection in infants in resource-limited settings towards universal access recommendations for a public health approach (WHO, 2006) http://www.who.int/hiv/pub/guidelines/WHOPMTCT.pdf Essential prevention and care interventions for adults and adolescents living in resource-poor settings (WHO, 2008) http://www.who.int/hiv/pub/guidelines/EP/en/index.html HIV and infant feeding: a framework for priority action (WHO, 2003) http://www.who.int/hiv/pub/mtct/en/HIVandInfantFeeding.pdf Interagency Standing Committee (IASC) Guidelines for HIV/AIDS interventions in emergency settings (IASC, 2004) http://www.who.int/3by5/publications/documents/iasc/en/ IMCI for high HIV settings (WHO, 2006) http://whqlibdoc.who.int/publications/2006/ 9789241594370.cb_eng.pdf Rapid HIV tests: guidelines for use in HIV testing and counselling services in resource-constrained settings (WHO, 2004) http://www.who.int/hiv/pub/vct/en/rapidhivtestsen.pdf

Influenza	Avian influenza http://www.who.int/topics/avian_influenza/en/ Pandemic influenza preparedness and mitigation in refugee and displaced populations: WHO guidelines for humanitarian agencies(WHO, 2008) http://www.who.int/diseasecontrol_emergencies/HSE_EPR_DCE_2008_3rweb.pdf Pandemic influenza preparedness and mitigation in refugee and displaced populations: WHO training modules for humanitarian agencies (WHO, 2006) http://www.who.int/diseasecontrol_emergencies/training/influenza/en/index.html Global influenza preparedness plan (WHO, 2005) http://www.who.int/csr/resources/publications/influenza/WHO_CDS_CSR_GIP_2005_5.pdf WHO latest guidance on Influenza H1N1 http://www.who.int/csr/disease/swineflu/guidance/health_professionals/en/index.html
Laboratory specimen collection	Guidelines for the collection of clinical specimens during field investigation of outbreaks (WHO, 2002) http://www.who.int/csr/resources/publications/surveillance/WHO_CDS_CSR_EDC_2000_4/en/
Leishmaniasis	Leishmaniasis: the disease and its epidemiology http://www.who.int/leishmaniasis/disease_epidemiology/en/index.html http://www.who.int/leishmaniasis/en/
Malaria	Guidelines for the treatment of malaria (WHO, 2006) http://www.who.int/malaria/docs/TreatmentGuidelines2006.pdf Malaria control in complex emergencies. An inter-agency field handbook (WHO, 2005) http://www.who.int/malaria/docs/ce_interagencyfhbook.pdf
Malnutrition	Communicable diseases and severe food shortage situations (WHO, 2005) http://www.who.int/diseasecontrol_emergencies/guidelines/Severe_food_shortages.pdf Community-based management of severe malnutrition http://www.who.int/nutrition/topics/comm_based_malnutrition/en/index.html Guidelines for the inpatient treatment of severely malnourished children (WHO, 2003) http://www.who.int/nutrition/publications/guide_inpatient_text.pdf Guiding principles for feeding infants and young children during emergencies (WHO, 2004) http://www.who.int/nutrition/publications/guiding_principles_feedchildren_emergencies.pdf Infant and Young Child Feeding in Emergencies. Operational guidance for emergency relief staff and programme managers (IFE, 2007) http://www.ennonline.net/pool/files/ife/ops-guidance-2-1-english-010307.pdf Nutrition in emergencies publications http://www.who.int/nutrition/publications/nut_emergencies/en/ Infant feeding in emergencies – guidance for relief workers in Myanmar and China http://www.who.int/child_adolescent_health/news/2008/13_05/en/index.html

	Management of the child with a serious infection or severe malnutrition: guidelines at first referral level in developing countries (WHO, 2000) http://www.who.int/child_adolescent_health/documents/fch_cah_00_1/en/index.html The management of nutrition in major emergencies (WHO,2000) http://whqlibdoc.who.int/publications/2000/9241545208.pdf
Management of dead bodies	Management of dead bodies after disasters: a field manual for first responders (PAHO, 2006) http://www.paho.org/english/dd/ped/DeadBodiesFieldManual.pdf Management of dead bodies in disaster situations (WHO, 2004) http://www.paho.org/english/DD/PED/DeadBodiesBook.pdf
Measles	WHO/UNICEF Joint statement on reducing measles mortality in emergencies http://whqlibdoc.who.int/hq/2004/WHO_V&B_04.03.pdf WHO measles information http://www.who.int/immunisation/topics/measles/en/index.html WHO Measles Vaccine Position paper http://www.who.int/immunization/wer7914measles_April2004_position_paper.pdf
Medical waste in emergencies	Four steps for the sound management of health-care waste in emergencies (WHO, 2005) http://www.healthcarewaste.org/en/documents.html?id=184&suivant=8 Guidelines for Safe Disposal of Unwanted Pharmaceuticals in and after Emergencies (WHO, 1999) http://www.healthcarewaste.org/en/documents.html?id=15&suivant=16 Water sanitation and health http://www.who.int/water_sanitation_health/medicalwaste/emergmedwaste/en/
Meningitis	Control of epidemic meningococcal disease. WHO practical guidelines, 2nd ed., (WHO, 1998) http://www.who.int/csr/resources/publications/meningitis/whoemcbac983.pdf
Mental health in emergencies	Mental health in emergencies http://www.who.int/mental_health/resources/emergencies/en/index.html Interagency Standing Committee (IASC) Guidelines on mental health and psychosocial support in emergency settings (IASC, 2007) http://www.humanitarianinfo.org/iasc/content/products/docs/Guidelines%20IASC%20Mental%20Health%20Psychosocial.pdf
Outbreak communications	WHO Outbreak communication guidelines http://www.who.int/csr/resources/publications/WHO_CDS_2005_28/en/index.html
Poliomyelitis	WHO-recommended surveillance standard of poliomyelitis http://www.who.int/immunization_monitoring/diseases/poliomyelitis_surveillance/en/index.html WHO Polio vaccine position paper http://www.who.int/immunization/wer7828polio_Jul03_position_paper.pdf

Pertussis	WHO Pertussis Vaccine Position paper http://www.who.int/immunization/topics/wer8004pertussis_Jan_2005.pdf
Soil transmitted helminths	Preventive chemotherapy in human helminthiasis http://whqlibdoc.who.int/publications/2006/9241547103_eng.pdf
Surgery - emergency surgical care	Integrated Management of Essential and Emergency Surgical Care (IMEESC) tool kit http://www.who.int/surgery/publications/imeesc/en/index.html
Surveillance	Protocol for the assessment of national communicable disease surveillance and response systems: guidelines for assessment teams, 2001 http://www.who.int/csr/resources/publications/surveillance/whocdscsrisr20012.pdf WHO report on global surveillance of epidemic-prone infectious diseases, 2000 http://www.who.int/csr/resources/publications/surveillance/WHO_CDS_CSR_ISR_2000_1/en/
Tetanus; maternal and neonatal tetanus	Maternal and neonatal tetanus elimination http://www.who.int/immunization_monitoring/diseases/MNTE_initiative/en/index.html Immunological basis of immunisation – tetanus http://www.who.int/immunization/documents/ISBN9789241595551/en/index.html WHO Position Paper on Tetanus Immunisation http://www.who.int/immunization/wer8120tetanus_May06_position_paper.pdf Maternal and neonatal tetanus – published in The Lancet, December 2007 http://www.who.int/hpvcentre/Maternal_and_neonatal_tetanus_Seminar.pdf
Travel advice	Guide on safe food for travellers http://www.who.int/foodsafety/publications/consumer/travellers/en/index.html International travel and health (2009) http://www.who.int/ith/en/
Typhoid (see also diarrhoeal diseases)	Background document: the diagnosis, treatment, and prevention of typhoid fever (WHO, 2003) [pdf-230kb] http://whqlibdoc.who.int/hq/2003/WHO_V&B_03.07.pdf
Tuberculosis	Tuberculosis care and control in refugee and displaced populations: An interagency field manual (UNHCR/WHO, 2007) http://whqlibdoc.who.int/publications/2007/9789241595421_eng.pdf
Vaccines	WHO Vaccines and biologicals http://www.who.int/immunization/en/ WHO vaccine-preventable diseases: monitoring system. 2007 Global summary http://www.who.int/immunization/documents/WHO_IVB_2007/en/index.html Immunization against diseases of public health importance http://www.who.int/immunization_delivery/en/index.html Linking vaccines with other interventions http://www.who.int/immunization_delivery/interventions/en/index.html Standards for surveillance of selected vaccine-preventable diseases (WHO/V&B/0 WHO Position Papers on Vaccines http://www.who.int/immunization/documents/positionpapers/en/index.html

	WHO Documents on vaccines and immunization: http://www.who.int/immunization/documents/en/ http://www.who.int/vaccines-documents/ WHO Immunological basis of immunization series http://www.who.int/immunization/documents/general/en/index.html WHO-recommended standards for surveillance of selected vaccine preventable diseases WHO/V&B/03.01 http://www.who.int/immunization/documents/WHO_VB_03.01/en/index.html
Vector control	Integrated vector management http://www.who.int/malaria/integratedvectormanagement.html Malaria vector control http://www.who.int/malaria/vectorcontrol.html Pesticides and their application for the control of vectors and pests of public health importance (WHO 2006) http://whqlibdoc.who.int/hq/2006/WHO_CDS_NTD_WHOPES_GCDPP_2006.1_eng.pdf Sound management of pesticides and diagnosis and treatment of pesticide poisoning: a resource tool. http://www.who.int/whopes/recommendations/IPCSPesticide_ok.pdf
Water, sanitation and health	Guidelines for drinking-water quality, third edition, incorporating first addendum http://www.who.int/water_sanitation_health/dwq/gdwq3rev/en/index.html Environmental health in emergencies and disasters: a practical guide http://www.who.int/water_sanitation_health/emergencies/emergencies2002/en/index.html WHO Technical notes for emergencies http://www.who.int/water_sanitation_health/hygiene/envsan/technotes/en/index.html Frequently asked questions in case of emergencies http://www.who.int/water_sanitation_health/emergencies/qa/en/index.html Four steps for the sound management of health-care waste in emergencies http://www.healthcarewaste.org/en/documents.html?id=184&suivant=25
Wounds and injuries	Best Practice Guidelines on Emergency Surgical Care in Disaster Situations http://www.who.int/surgery/publications/BestPracticeGuidelinesonESCinDisasters.pdf Integrated Management of Essential and Emergency Surgical Care (IMEESC) tool kit http://www.who.int/surgery/publications/imeesc/en/index.html Prevention and management of wound infection http://www.who.int/hac/techguidance/tools/Prevention%20and%20management%20of%20wound%20infection.pdf WHO generic essential emergency equipment list (WHO, 2006) http://www.who.int/surgery/publications/EEEGenericListFormatted%2006.pdf
Yaws	Yaws: the disease and its treatment http://www.searo.who.int/en/Section10/Section2134_10824.htm

Yellow fever	Yellow fever disease http://www.who.int/vaccines-documents/DocsPDF/www9842.pdf District guidelines for yellow fever surveillance http://www.who.int/csr/resources/publications/yellowfev/whoepigen9809.pdf Manual for the monitoring of yellow fever virus infection http://whqlibdoc.who.int/hq/2004/WHO_IVB_04.08.pdf
Zoonosis	Zoonoses and veterinary public health http://www.who.int/zoonoses/resources/en/

Web sites

WHO Headquarters	http://www.who.int/
WHO Regional Office for Africa (AFRO)	http://www.afro.who.int/
WHO AFRO Division of Communicable Disease Prevention and Control (DDC)	http://afro.who.int/ddc/index.html
WHO Disease Control in Humanitarian Emergencies	http://www.who.int/diseasecontrol_emergencies/en/
WHO Cholera	http://www.who.int/topics/cholera/en/index.html
WHO Dengue	http://www.who.int/topics/dengue/en/
WHO Epidemic and Pandemic Alert and Response	http://www.who.int/csr/en/
WHO Global Malaria Programme	http://www.who.int/malaria/
WHO Global Malaria Programme – Epidemics and Emergencies	http://www.who.int/malaria/epidemicsandemergencies.html
WHO Health Action in Crises (HAC)	http://www.who.int/hac/en/
WHO Stop TB	http://www.stoptb.org/
WHO Vector control	http://www.who.int/neglected_diseases/vector_ecology/en/
WHO Water and Sanitation	http://www.who.int/water_sanitation_health/en/

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