World Health Organization

COMMUNICABLE DISEASE EPIDEMIOLOGICAL PROFILE

Sri Lanka

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 World Health Organization team for Disease Control in Humanitarian Emergencies (DCE). The aim is 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 is designed for use in developing public health strategy, and in prioritizing and coordinating communicable disease control activities between all agencies working in such countries.

Diseases have been included in this document because of their high burden or epidemic potential for Sri Lanka, or because they are (re)emerging diseases; that is, important but neglected tropical diseases subject to global elimination or eradication.

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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 GAR, Food Safety, Zoonoses and Foodborne Diseases (FOS), Public Health and Environment (PHE) in the 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 and Services (HSS) cluster; Security and Staff Services (SES) in the General Management (GMG) cluster; and the cluster of Health Action in Crises (HAC) and the Polio Eradication Initiative (POL).

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Acronyms

ADL	acute adenolymphangitis
AES	acute encephalitis syndrome
AFB	acid-fast bacillus
AFP	acute flaccid paralysis
AIDS	acquired immunodeficiency syndrome
ALRI	acute lower respiratory infections
ART	antiretroviral therapy
BCG	bacille Calmette-Guérin
CFR	case-fatality ratio
CSF	cerebrospinal fluid
CXR	chest X-Ray
DALY	disability-adjusted life year
DEC	diethylcarbamazine citrate
DHF	dengue haemorrhagic fever
DOTS	directly observed treatment, short-course
DSS	dengue shock syndrome
DT	diphtheria–tetanus
DTP3	third dose of diphtheria–tetanus–pertussis vaccine
EIA	enzyme immunoassay
ELISA	enzyme-linked immunosorbent assay
EPTB	extrapulmonary tuberculosis
FA	fluorescent antibody
HAV	*Hepatitis A virus*
HEV	*Hepatitis E virus*
HI	haemagglutination inhibition
Hib	*Haemophilus influenzae* type B
HIV	human immunodeficiency virus
IDU	injecting drug user
IM	intramuscular
IMCI	Integrated Management of Childhood Illness
IV	intravenous
JE	Japanese encephalitis

LCL	localized cutaneous leishmaniasis
LF	lymphatic filariasis
LLIN	long-lasting insecticidal net
MAT	microscopic agglutination test
MDA	mass drug administration
MDR	multidrug resistant
MDT	multidrug therapy
MOH	ministry of health
NID	national immunization day
NSAID	non-steroidal anti-inflammatory drug
OC	oily chloramphenicol
OCV	oral cholera vaccine
OPV	oral poliovirus vaccine
ORS	oral rehydration salts
PCP	*Pneumocystic (carinii) jiroveci* pneumonia
PCR	polymerase chain reaction
PEP	post-exposure prophylaxis
RDT	rapid diagnostic testing
RNA	ribonucleic acid
RSV	respiratory syncytial virus
Sd1	*Shigella dysenteriae* serotype 1
SNID	subnational immunization day
STH	soil-transmitted helminth
SPR	slide positivity rate
STI	sexually transmitted infection
TB	tuberculosis
Td	tetanus toxoid with reduced diphtheria content
TT	tetanus toxoid
UNICEF	United Nations Children's Fund
WHO	World Health Organization
wP	whole-cell pertussis vaccine

PART I

Introduction

Purpose

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

Publications in this series are primarily intended to guide public health actions. Although they contain clinical information, they are not designed for clinical practice. Clinical decisions should not be based solely on information contained within this document.

Target audience

This publication is aimed at public health managers and public health professionals working for populations living in the Democratic Socialist Republic of Sri Lanka (referred to hereafter as Sri Lanka).

Rationale

Diseases have been included in this document because of their high burden or epidemic potential for Sri Lanka, or because they are (re)emerging diseases; that is, important but neglected tropical diseases subject to global elimination or eradication. Each chapter describes a specific disease. The annexes provide sources to guide the formulation of public health action.

Production process

This document is the product of collaboration between the Communicable Diseases Working Group on Emergencies (CD-WGE), appropriate WHO region and country offices, and DCE, with technical coordination by DCE. Each disease-specific section was drafted by DCE, and then submitted to the appropriate WHO focal point of the CD-WGE and the WHO regional and country offices, to ensure technical accuracy and inclusion of country-specific information. The quantity and quality of epidemiological data from Sri Lanka have been compromised by the country's humanitarian crisis, which has disrupted health and surveillance systems for many years.

Background to humanitarian crisis and its impact on health in Sri Lanka

The Democratic Socialist Republic of Sri Lanka, an island in the Indian Ocean, has a population of approximately 19 million people. Based on a 1981 census, the majority of the population (69%) are Buddhist, with the remainder being Hindu (15%), Muslim (8%) or Christian (8%). Ethnically, most are Sinhalese (74%). The largest minority group is Tamil (18%) (*1*). The longstanding civil war between the government and the Liberation Tigers of Tamil Eelam (LTTE) in the north and east of the country concluded in May 2009. During the 25-year conflict, more than 70 000 people died and more than one million were internally displaced.

With the cessation of conflict, efforts are being made to shift from relief to recovery. The conflict has caused damage to infrastructure and homes; restricted mobility; disruption of local economies; disorder in community and institutional networks, educational, water and sanitation facilities; deterioration of health services (through loss of staff, damage to equipment and scarcity of medicines); and psychological trauma. (Annex 1 provides a map and a timeline of the crisis).

Sri Lanka is also prone to natural disasters. For example, there were landslides and floods in 2003, droughts in 2002–04, and the tsunami in December 2004, which devastated much of the coastal belt.

Nationally, Sri Lanka has achieved impressive levels of male and female literacy, school enrolment (with a male:female ratio close to one throughout the education system), and low under-five mortality rates, despite its low level of per-capita income. The nation has a countrywide, comprehensive network of health centres, hospitals and other medical institutions, and a large workforce engaged in curative and public health activities. Health service provisions in the public sector are mainly free to consumers. Management of all health-care institutions, other than teaching hospitals and field services, is decentralised to the provincial councils. However, malnutrition is prevalent in most of Sri Lanka's districts (and is worst in those areas which have been affected by conflict). More than 50% of the population consumes less than the minimum dietary level required for energy. (Annex 1 includes key national indicators, although it lacks data from many of the areas that have been affected by conflict.)

Major communicable disease threats

Communicable disease risk in natural disasters and conflict situations are determined by (*2*):

- population displacement
- baseline health status (including prevalence of malnutrition)
- lack of access to adequate shelter, food, water, and sanitation
- overcrowding in temporary settlements and camps
- lack of access to basic health services.

Death rates can be extremely high, particularly in areas of acute conflict, and mainly result from communicable diseases and malnutrition. Dead bodies do not usually pose an outbreak risk or require disinfection (except in cases of cholera, shigellosis and haemorrhagic fever).

For Sri Lanka, the following diseases, are considered major communicable disease risks:

Water-related diseases:

- bacillary dysentery (shigellosis)
- cholera
- other diarrhoeal diseases
- hepatitis A and E
- typhoid fever.

Diseases associated with overcrowding:

- measles
- acute lower respiratory tract infections
- meningococcal disease
- tuberculosis
- vector-borne diseases
  - dengue
  - Japanese encephalitis.

A coordinated approach to public health measures is required. This involves:

- preparedness – contingency planning and stockpiling of supplies
- rapid assessment of communicable disease threats – risks, capacities
- coordination and identification of a lead agency

- prevention of communicable disease threats – ensuring provision of adequate shelter, water, sanitation; simple messages on hand hygiene, food and health services; vaccination and vector control programmes; and basic laboratory facilities
- control of patients and contacts – establishing treatment protocols, training of health workers, drug and equipment supplies, and referral planning
- surveillance and early warning of the most important communicable disease threats – establishing alert, monitoring and feedback systems
- epidemic response – ensuring adequate supplies, standard operating procedures for investigation and response, training of epidemic response teams and appropriate risk communication.

The annexes provide background information on:

- key national indicators and general background (Annex 1)
- steps in managing an outbreak (Annex 2)
- the diagnosis of communicable diseases (Annex 3)
- safe water, sanitation and hygiene (Annex 4)
- injection safety (Annex 5)
- WHO fact sheets and information sources (Annex 6)
- WHO contacts (Annex 7).

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PART II

Country-specific disease information

ACUTE LOWER RESPIRATORY TRACT INFECTIONS

Description

Clinical description

Acute lower respiratory tract infections (ALRI) include bronchitis, bronchiolitis, and both bronchopneumonia and lobar pneumonia. If inappropriately treated, severe pneumonia is fatal in 10–20% of people. Prompt recognition and treatment are life saving.

Infectious agents

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

Other causes of pneumonia include *Staphylococcus aureus, Mycoplasma pneumoniae* and Gram-negative organisms. Gram-negative organisms are common, particularly in hospital-acquired pneumonia and in immunosuppressed individuals. *Mycobacterium tuberculosis* is an often unrecognized cause of acute respiratory infections. In addition, patients infected with human immunodeficiency virus (HIV) are particularly susceptible to *Pneumocystic (carinii) jiroveci* pneumonia (PCP).

Case classification of pneumonia

Bacterial pneumonia remains a major cause of death in children under 5 years of age; therefore, empirical treatment with antibiotics is recommended when signs of pneumonia are present. In children, the Integrated Management of Childhood Illness (IMCI) provides clear case classification for use in initiating treatment (*1*). If a child is wheezing, a trial of up to three cycles of a rapid-acting, inhaled bronchodilator should be given before classifying the condition as pneumonia.

Non-severe pneumonia (age 2–59 months)

Symptoms are:

- cough or difficult breathing
- fast breathing (after trial of bronchodilators, if wheezing is present, 50 breaths per minute or more for infants aged 2–11 months; 40 breaths per minute or more for a child aged 12–59 months)

- lack of general danger signs (i.e. no indication of inability to drink or breast feed, vomiting all ingested food, convulsions, lethargy, unconsciousness, chest indrawing or stridor).

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

Symptoms are:

- cough or difficult breathing
- any general danger sign in a typically calm child (inability to drink or breast feed, vomiting all ingested food, convulsions, lethargy, unconsciousness, chest indrawing or stridor).

Very severe respiratory disease (age < 2 months)

Clinically, it is difficult to distinguish between pneumonia and other causes of very severe respiratory disease in a young infant. An infant is classified as having severe respiratory disease if one or more of the following signs are present:

- not feeding well
- convulsions or fast breathing (60 breaths/minute or more)
- severe chest indrawing
- fever (axillary 37.5 °C or above)
- low body temperature (less than 35.5 °C axillary)
- movement only when stimulated
- no movement at all.

Mode of transmission

Transmission is through droplets (i.e. airborne), oral contact, or body contact with items freshly soiled with respiratory secretions.

Incubation period

Incubation varies depending on infective agent; the incubation period is usually 2–5 days.

Period of communicability

The period of communicability depends on the infective agent. The disease is usually communicable during the symptomatic phase.

Reservoirs

Humans are the primary reservoir for organisms that cause respiratory disease, including pneumonia.

Epidemiology

Disease burden

ALRI continue to be the leading cause of acute illnesses worldwide. Pneumonia in particular remains the most important cause of infant and young child mortality, accounting for about two million deaths each year and ranking first among causes of disability-adjusted life years (DALYs) lost in developing countries (94.6 million, 6.3% of total). Of every 1000 children born alive, 12–20 die from pneumonia before their fifth birthday.

In Sri Lanka, ALRI are leading causes of childhood mortality and morbidity, being responsible for 9% of deaths of children under 5 years of age (Ministry of Healthcare and Nutrition, Sri Lanka[1]). An estimated 58% of children with pneumonia are taken to an appropriate health-care provider. Populations most at risk for developing a fatal respiratory disease are the very young, the elderly and the immunocompromised. Sri Lanka is part of the South Asian Pneumococcal Alliance (for pneumococcal surveillance).[2]

Geographical distribution

There are no data available on geographic distribution.

Seasonality

Epidemics and outbreaks of influenza occur in different seasonal patterns. In tropical zones, seasonal patterns are less pronounced, and the virus can be isolated throughout the year.

Alert threshold

The alert threshold is an increase in the number of cases above the expected number for that time of the year in a defined area.

1 http://www.health.gov.lk

2 http://www.indiaclen.org/sapna/home/sapna3.html

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 human disease vulnerability.

Overcrowding and poor ventilation

Overcrowding and poor ventilation increase the risk of infection.

Poor access to health services

Prompt identification and treatment of cases are the most important control measures. Reduced access to effective health services can delay or prevent adequate treatment, resulting in high case-fatality ratios (≥ 20% in emergency situations).

Food shortages

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

Lack of safe water, poor hygiene practices and poor sanitation

Inadequate safe water, poor personal hygiene, hand-washing and ventilation increase spread of respiratory infections.

Others

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

Prevention and control measures

Case management

Treatment guidelines are given below for the most common bacterial causes of respiratory disease (Table 1). Patients who are slow to respond 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 (TB) and PCP.

In children, early recognition and appropriate treatment of cases are most important. Providers trained in IMCI should use the most recent guidelines (*1*).

For management of HIV-infected children, IMCI should be used in high HIV settings (*2*).

Supportive measures are part of integrated case management; such measures include continued feeding to avoid malnutrition (which increases risk of death), vitamin A if indicated, antipyretics to reduce high fever and protection from cold temperatures (especially keeping young infants warm). Prevention of low blood glucose (through breastfeeding or use of sugar water) is necessary for severe cases. Careful counselling on home-based care, including adherence to antibiotic treatment, should be given to care-givers in non-severe cases.

In adults, treatment options include 7–10 days of oral amoxicillin (e.g. 1 g three times a day) or erythromycin (500 mg every 6 hours). Severe disease should be referred for inpatient treatment.

Table 1. **General guidelines for first-line empirical treatment of acute lower respiratory infection in children (3, 4)**

Classification	Treatment
Non-severe pneumonia (age 2–59 months)	Five days of antibiotic therapy (oral amoxicillin at 25 mg/kg twice daily, or cotrimoxazole). If patient wheezes, add inhaled or oral bronchodilator. For persistent cough for > 3 weeks, refer for TB assessment.
Severe pneumonia/very severe respiratory disease (age 2–59 months)	A first dose of 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 a day) for 5 days; then, if the child responds well, complete treatment at home or in hospital with oral amoxicillin (25 mg/kg twice daily) plus IM gentamicin once daily for a further 5 days. Where referral is not possible or injection not available, treat with oral amoxicillin for 5 days (25 mg/kg twice daily).
Very severe respiratory disease (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/dose) every 12 hours and gentamicin (5 mg/kg/day; one dose) for up to 10 days.
Very severe respiratory disease (ages 2–8 weeks)	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 IM) or penicillin (50 000 units/kg/dose) every 8 hours and gentamicin (7.5 mg/kg/day; one dose) for up to 10 days.

IM, intramuscular; TB, tuberculosis.

Prevention

Efforts must be made to increase early diagnosis and treatment with efficacious antibiotics. This can be achieved through raising community awareness, health education about early danger signs, mobile clinic development and health-care worker training. Adequate nutrition is important. Cotrimoxazole prophylaxis should be used to prevent HIV-related infections in adults and children (*5*). Also, adequate hygiene, hand-washing and household ventilation should be promoted.

Immunization

Immunization against *H. influenzae* type B (Hib) vaccine, measles, pertussis and pneumococcal disease reduces the impact of pneumonia. Importantly, it also reduces the severity of virus-associated pneumonias, reducing bacterial superinfection.

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Further reading

- World Health Organization Statistical Information System [WHOSIS], *Mortality profiles*. Geneva, WHO, 2009 (http://www.who.int/whosis/mort/profiles/en, accessed 10 August 2010).

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

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 mucus 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-limiting and resolves in 4–7 days. Complications include intestinal perforation, toxic megacolon, rectal prolapse, haemolytic uraemic syndrome and convulsions (in young children). Case-fatality ratios are low in those not requiring hospital treatment, but can be as high as 15% in severe cases.

Infectious agent

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

S. flexneri is the main cause of endemic shigellosis in developing countries. However, *S. dysenteriae* serotype 1 (Sd1) causes the most severe disease and is usually responsible for epidemics, which are often large and may even be regional. *S. sonnei* and *S. boydii* usually cause relatively mild illness.

S. sonnei is the most common cause of shigellosis in Sri Lanka. Amoebic dysentery also occurs in Sri Lanka, but is less common.

Case classification

Suspected

Shigella is suspected when a person has diarrhoea with visible blood in the stools.

Confirmed

A suspected case is confirmed when *Shigella* is isolated from stools.

Mode of transmission

Shigella is transmitted orally and through faeces, particularly through contaminated water and food. Street food stalls are a common source of contaminated food. Flies may also transmit the organism.

Incubation period

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

Period of communicability

The period of communicability is during acute infection, and up to 4 weeks after illness without treatment, or 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; 99% of cases occur in developing countries. Also, 70% of cases and 60% of deaths occur in children under 5 years of age. Illness in infants under 6 months is unusual. In endemic areas, the disease is more severe in young children than in adults, in whom infection is often asymptomatic.

In Sri Lanka, all dysentery cases are reported collectively, irrespective of etiology. Data include shigellosis as well as other causes of dysentery (e.g. amoebiasis). The surveillance-case definition does not require laboratory confirmation. The number of notified cases dropped below 7000 in 2008 (Table 1).

Table 1. **Number of notified cases of dysentery by year in Sri Lanka, 2003–08 (*1*)**

Year	Notified cases of dysentery in Sri Lanka
2008 (until 26 Dec)	6 379
2007	7 297
2006	7 967
2005	7 987
2004	10 194
2003	8 073

Geographical distribution

Shigellosis occurs throughout Sri Lanka. In 2006, the highest rates of cases were recorded in the districts of Ampara, Badulla, Moneragala and Vavinya.

Seasonality

In 2006, the highest number of case notifications were received in November and December (about 1000/month) and the lowest in March to May (about 300–400/month) (*1*).

Alert threshold

Health authorities should be notified if one or more suspected cases of shigellosis are identified. 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
- five or more linked cases of bloody diarrhoea are reported.

Any of the above should lead to laboratory testing for *Shigella*.

Outbreaks

Sd1 can cause epidemics with high morbidity and mortality. Susceptibility is general, and secondary attack rates in households can be as high as 40%.

The Karuwalagaswewa area of Sri Lanka experienced an outbreak of dysentery in November–December 2003, with 120 cases reported in November and 45 cases in December (*1*).

Risk factors for increased burden

Population movement

Classic settings for explosive dysentery outbreaks include complex emergencies and natural disasters, such as flooding, that lead to large population movements and overcrowded refugee camps.

Overcrowding

Overcrowding increases the risk of infection. The risk of epidemics of Sd1 is high in camp settings; in these situations, up to one third of the population may be at risk.

Poor access to health services

Poor access to health services increases the risk of transmission, delays detection and containment, and reduces treatment.

Food shortages

Malnourished people of all ages are susceptible to severe disease and death; infants are particularly at risk if they are not breastfed.

Lack of safe water, poor hygiene practices and poor sanitation

Lack of safe water, poor sanitation and poor hygiene (e.g. due to lack of soap and safe water) are all important risk factors, and can lead to contaminated food items, especially from street food stalls.

Other

During an outbreak, it is important to monitor antibiotic sensitivity, because resistance may develop. To date, there have been no reports of *Shigella* resistance to ciprofloxacin (*1*).

Prevention and control measures

Case management

Early and appropriate therapy is important in the prevention and control of dysentery. Treatment with an effective antimicrobial agent can reduce both the severity and duration of the disease. The particular antibiotic selected will depend on resistance patterns of the bacteria and drug availability.

It is important to confirm the susceptibility of *S. dysenteriae* to antibiotics in the early stages of an outbreak and during the course of the outbreak. Consequently regular stool sampling is required, as resistance patterns may vary over time. Ciprofloxacin is the current first-line antibiotic of choice recommended for treatment of Sd1.

Supportive treatment includes using oral rehydration salts (ORS), continued feeding (frequent small meals) and antipyretics to reduce high fever. Breastfeeding should continue in infants and young children.

If antibiotics are in short supply, they should be reserved for high-risk groups (young children, and those who are elderly or malnourished).

Prevention

Prevention of shigellosis in any community relies mainly on adequate clean water, sanitation and hygiene; this involves:

Safe drinking-water

- *Provision of adequate quantities of safe drinking-water.* The World Health Organization (WHO) minimum emergency, standard provision is 20 l per person per day.

Safe disposal of human waste

- *Provision of adequate facilities for disposal of human waste*. The minimum emergency standard is one latrine for every 20 people.

Hygiene

- *Provision of sufficient, clean, quantities of water and soap and provision of adequate supplies for hand-washing, bathing and laundry needs*. The minimum emergency standard is 250–500 g of soap per person per month.
- *Promotion of hygiene* – including use of latrines and prevention of human defecation on the ground, or in or near water; disposal of children's excreta in latrines; thorough hand-washing before eating, after defecation, before food preparation, and after cleaning children or changing their diapers.
- *Promotion of food safety* – through
  - provision of clean, appropriately cooled and spatially adequate food storage facilities (for both uncooked and cooked food, and clean cooking utensils), and adequate quantities of water and fuel for cooking and re-heating
  - promotion of "five keys to safer food" – keep clean, separate raw and cooked food, cook food thoroughly, keep food at safe temperatures, use clean water and cooking tools.
- *Promotion of breastfeeding* – by providing information on the importance and protective qualities of breastfeeding for all infants and young children, and especially for those who are ill.

Immunization

Although several vaccine candidates are under development, none are yet available.

Epidemic control

Inform the health authorities if one or more suspected cases of shigellosis are identified. Early detection and notification of epidemic dysentery, especially among

adults, allows timely mobilization of the resources needed for appropriate case management and control.

WHO guidelines on confirming an outbreak should be followed (Annex 2). Control involves collecting rectal swabs from suspected cases and transporting them to a laboratory for culture to confirm the diagnosis of Sd1. Samples should be transported immediately in an appropriate medium (e.g. Cary-Blair), maintaining the cold chain (2–8 °C). Ideally, the specimen should be analysed within 2 hours, because the viability of the bacteria is very variable in this medium when refrigerated for 1–3 days, and laboratory findings become unreliable. Therefore, 10–20 samples should be used to confirm the outbreak, the pathogen strain and the antibiotic susceptibility.

If Cary-Blair medium is not available, then fresh stool samples can be sent to the laboratory, 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 undertaken at regular intervals, especially during outbreaks, 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.

Treatment and control activities should start immediately; they should not be delayed by waiting for laboratory results.

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

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Further reading

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- Prevention of foodborne disease: five keys to safer food [various resources]. Geneva, WHO, 2009 (http://www.who.int/foodsafety/consumer/5keys/en/index.html, accessed 10 August 2010).

- *Operational guidance for emergency relief staff and programme managers*. Infant Feeding in Emergencies Core Group, UNICEF et al., 2007 (http://www.who.int/child_adolescent_health/news/2008/13_05/en/index.html)
- Technical notes for emergencies [various topics]. Geneva, WHO, 2005 (http://www.who.int/water_sanitation_health/hygiene/envsan/technotes/en/index.html, accessed 10 August 2010).
- *The treatment of diarrhoea: a manual for physicians and other senior health workers*. Geneva, WHO, 2005 (WHO/FCH/CAH/05.1) (http://www.who.int/entity/child_adolescent_health/documents/9241593180/en, accessed 10 August 2010).
- *Acute diarrhoeal diseases in complex emergencies: critical steps*. Geneva, WHO, 2004 (http://www.who.int/topics/cholera/publications/critical_steps/en/index.html, accessed 10 August 2010).
- *First steps for managing an outbreak of acute diarrhoea*. Geneva, WHO, 2003 (WHO_CDS_CSR_NCS_2003.7) (http://www.who.int/cholera/publications/first_steps/en/index.html, accessed 10 August 2010).

Annexes:

- Annex 4: Safe water, sanitation and hygiene
- Annex 6: WHO fact sheets and information sources (cholera; child health in emergencies; diarrhoeal diseases; food safety; water, sanitation and health).

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

CHIKUNGUNYA FEVER

Description

Clinical description

Chikungunya fever is a mosquito-borne viral disease that causes an acute debilitating illness, characterized by a sudden onset of fever, and frequently accompanied by severe joint pain. Other common signs and symptoms include muscle pain, headache, nausea, fatigue and rash. The joint pain affects mainly the extremities (ankles, wrists and fingers), but also the large joints; this pain is often quite debilitating, but usually ends within a few days or weeks. Although most patients recover fully, in some cases, joint pain may persist for several months or even years. Occasional cases of eye, neurological and heart complications have been reported, as well as gastrointestinal complaints.

The disease shares some clinical signs with dengue, and can be misdiagnosed in areas where dengue is common. Haemorrhagic phenomena are relatively uncommon, as are serious complications; however, in older people with underlying conditions, the disease can contribute to the cause of death. Often, symptoms in infected individuals are mild and the infection may go unrecognized.

Infectious agent

Chikungunya virus is a member of the genus *Alphavirus,* of the family *Togaviridae*.

Case classification

Suspected case

A case of *Chikungunya virus* should be suspected when a person has sudden fever onset with severe and sometimes persistent joint pain. These symptoms may be accompanied by myalgia, headache, nausea or rash.

Probable case

A case of *Chikungunya virus* is probable when it is a suspected case with an epidemiological link to a confirmed chikungunya outbreak in the same place at the same time.

Confirmed case

A confirmed case is a patient with one of the following findings, irrespective of clinical presentation:

- detection of anti-chikungunya IgM enzyme-linked immunosorbent assay (ELISA) antibody titre of 40 units or more in a single serum sample
- a four-fold rise in anti-chikungunya IgG antibody titre between acute and convalescent serum samples
- detection by reverse transcriptase polymerase chain reaction (RT-PCR) of *Chikungunya virus* nucleic acids in acute serum
- isolation of *Chikungunya virus*.

Diagnostic tests in Sri Lanka are performed at the Medical Research Institute (MRI) and Molecular Medicine Unit of the Department of Microbiology at University of Kelaniya.

Mode of transmission

Aedes aegypti and *A. albopictus* mosquitoes have both been implicated as vectors in large outbreaks of chikungunya. Both of these mosquitoes also transmit dengue, although *A. aegypti* is the main vector of that disease.

Whereas *A. aegypti* is confined to the tropics and subtropics, *A. albopictus* also occurs in temperate and even cold–temperate regions.

A. aegypti breeds primarily in water in natural and artificial containers closely associated with human habitation, including earthenware jars, flower vases, metal drums and concrete cisterns used for domestic water storage. It also breeds in artificial outdoor habitats such as used automobile tyres, small discarded plastic food containers and other items that collect rainwater.

A. albopictus has a wide geographical distribution, is particularly resilient, and can survive in both rural and urban environments. Further, the mosquito's eggs can remain viable throughout the dry season, giving rise to larvae and adults the following rainy season. It is a tree-hole mosquito in natural areas, and breeds around garden bush vegetation in urban areas. Larval habitats are mostly in small containers. The ecological flexibility of *A. albopictus* allows colonization in coconut husks, cocoa pods, bamboo stumps, bromeliads, tree holes and rock pools, in addition to the same artificial outdoor habitats as *A. aegypti*.

These mosquitoes can bite throughout daylight hours, although there may be peaks of activity in the early morning and late afternoon. Both species will bite outdoors, but *A. aegypti* will also readily feed indoors.

Incubation period

After a person is bitten by an infected mosquito, onset of illness usually occurs between 3 and 7 days (range 2–12 days). "Silent" or asymptomatic infections are thought to occur.

Period of communicability

The duration of viraemia ranges from the onset of illness to 7 days, but is usually 3–5 days after onset. There is no evidence of direct human-to-human transmission. Chikungunya infection (both clinical and silent) is thought to confer life-long immunity.

Reservoirs

Human beings serve as the *Chikungunya virus* reservoir during epidemic periods. Outside these periods, the main reservoirs are monkeys, rodents, birds and other unidentified vertebrates. Outbreaks can occur in monkeys when herd immunity is low. Monkeys develop viraemia but no pronounced physical manifestations.

Epidemiology

Disease burden

The first outbreak of chikungunya fever reported in Sri Lanka occurred in 1965. It originated in Colombo and spread along the south-western coastal belt from Negombo to Matara from May to June of that year.

Chikungunya re-emerged in Sri Lanka in 2006–07 in an epidemic with more than 40 000 suspected cases. It affected districts along the coastal belt, including Batticaloa, Colombo, Jaffna, Kalmunai, Mannar, Puttalam and Trincomalee. The districts of Kandy, Kurunegala and Matale were less affected.

More than 100 000 cases have been reported in Sri Lanka since chikungunya resurged in 2006. High attack rates and high morbidity have been reported, as in outbreaks in India and the western Indian Ocean coastal areas.

Geographical distribution

In the 2006–07 outbreak, urban coastal districts were affected. In contrast, the 2008–09 outbreak (discussed below) affected mainly semiurban and rural areas, and *A. albopictus* was the predominant vector. This situation presents additional public health challenges, with vector control and source reduction needed as primary prevention measures.

Seasonality

Chikungunya occurs year round. As with other diseases caused by arboviruses (arthropod-borne viruses), outbreaks of chikungunya fever begin during the rainy season, when vector density peaks.

Alert thresholds

There are no defined alert thresholds for chikungunya.

Risk factors for increased burden

Population movement

The risk of transmission is increased by movement of viraemic populations into nonendemic areas where the vector is present, and movement of susceptible individuals into endemic areas. The creation of temporary shelters where rainwater is harvested and stored in household containers also increases the risk of transmission.

Overcrowding

In emergency situations, proximity of human habitation to water storage containers and accumulation of water (e.g. in discarded containers and temporary building materials such as used tyres) promote increased breeding sites for *A. aegypti* and increase contact between humans and the vector.

Poor access to health services

Poor access to health services can increase the risk of transmission of chikungunya, and of the disease being mistaken for dengue.

Food shortages

Food shortages are not thought to be associated with an increased risk of chikungunya.

Lack of safe water and poor environmental hygiene practices

Accumulation of water in discarded solid waste, plastic containers and other vessels may provide vector breeding sites, increasing the risk of transmission of the disease.

Others

Possible breeding sites for the vectors include abandoned gem-mining pits, coconut shells used for rubber tapping, remains of mortar shells in military camps, and large areas of plants that collect water on their leaf axils (e.g. habarala and bamboo). High rainfall also contributes to vector breeding.

Prevention and control measures

Case management

Chikungunya fever is not a life-threatening infection, and treatment is based on symptoms. Rest, fluids and paracetamol (acetaminophen) may relieve symptoms of fever and joint pain. Aspirin should be avoided during the acute stages of the illness. To avoid further spread of this disease, infected patients should limit their exposure to mosquitoes by staying indoors or under a mosquito net for at least 7 days after developing symptoms. Convalescence can be prolonged (lasting months or even years), and persistent joint pain may require analgesic and long-term anti-inflammatory therapy. No vaccine or specific antiviral treatment is currently available.

Prevention

Preventative efforts mirror those used for dengue. The most effective, economic and safe method of prevention is integrated vector control. This includes 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.

Biological and chemical control measures are used against both mosquito larvae and adults. The most effective ways of reducing larvae, and therefore the *Aedes* mosquito density in the community, are continued and sustained reduction of containers that may collect water, and covering and frequent draining of containers. Larvicides are also highly effective, but should be long lasting and preferably safe for use in drinking-water; for example, slow-release formulations of temephos, which is approved by the World Health Organization (WHO) Pesticide Evaluation Scheme (WHOPES). Insect growth regulators (e.g. pyriproxyfen) are also available, and are safe and long lasting. Biological control includes slow-release *Bacillus thuringiensis* H-14, the crustacean *Mesocyclop* and several species of larviforous fish. Reducing the adult mosquito density by spraying with insecticide from trucks and aircraft, or from portable backpack units, can be effective if conducted regularly and efficiently, although such operations are difficult to sustain.

Where insecticides or larvicides are used, regular monitoring of vector susceptibility is necessary to ensure the appropriate choice of nonresistant chemical. All mosquito control efforts should be accompanied by active monitoring and surveillance of the natural mosquito population, to determine the impact of the programme. Piped water supplies, well-maintained drainage and efficient systems for removal of domestic waste can also have a large positive impact. *Aedes* mosquitoes mainly bite during the day, particularly after sunrise and before sunset.

At household level, mosquito bites can be avoided when people:

- wear full-sleeve clothes and long dresses or trousers to cover their limbs
- use repellents (taking care when using them on small children and the elderly)
- use mosquito coils, repellents and electric vapour mats during the daytime
- use mosquito nets to protect babies, older people and others who may rest during the day; such nets are more effective when treated with insecticides
- hang insecticide-treated curtains at windows or doorways to repel or kill mosquitoes
- use fly screens on doors and windows.

Health centres are an essential fever-alert network for early detection, diagnosis, and symptomatic treatment of suspected cases.

Immunization

Vaccines are not available, but work is in progress.

Epidemic control

Control involves vector surveillance, including detection and isolation of the vectors. Measures include:

- identifying vector breeding places and encouraging their control by helping people to choose the most appropriate ways to reduce larvae and pupae populations
- promoting the use of mosquito repellents and other mosquito-avoidance measures for individuals exposed to bites (e.g. through their occupation)
- identifying and reducing infection in vertebrate animal reservoirs, where possible.

Further reading

- WHOPES website, 2010 (http://www.who.int/whopes/en, accessed 5 October 2010).
- *Guidelines on clinical management of chikungunya fever*. Geneva, WHO/SEARO, 2008 (http://www.searo.who.int/LinkFiles/Publication_guidelines_on_cli_mgmt_chikungunya_fvr-(cd-180).pdf, accessed 5 October 2010).
- *Guidelines for integrated vector management*. Harare, WHO/AFRO, 2003 CD-WGE technical focal point: Department of Global Alert and Response (GAR)

CHOLERA

Description

Clinical description

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

Infectious agent

Cholera is caused by the bacterium *Vibrio cholerae* O1 (classical and El Tor) and O139.

Case classification

Suspected case

Cholera is suspected if a person:

- over 5 years of age develops severe dehydration or dies from acute watery diarrhoea (clinical case)
- has a sudden increase in the daily number of acute watery diarrhoea, especially passing the "rice-water stools" typical of cholera.

Confirmed case

Cholera is confirmed if *V. cholerae* O1 or O139 is isolated from stools in any patient with diarrhoea. A new and rapid diagnostic test is being evaluated.

Mode of transmission

Cholera is transmitted mainly by the faecal–oral route. Transmission can occur through:

- ingesting contaminated water, including accidental ingestion of contaminated surface water
- eating contaminated food – especially fruits and vegetables contaminated through water or soil, or during preparation (e.g. rice, millet and food from street vendors) – and contaminated seafood

- person-to-person transmission; for example, when caring for cholera patients or through direct contact with bodies of deceased cholera patients (e.g. washing the body for funeral ceremonies)
- indirect contamination (of hands) through poor hygiene practices and lack of soap; and through wound infections arising from environmental exposure, especially brackish water from occupational accidents among fishermen.

Incubation period

The incubation period for cholera can be anything from a few hours to 5 days, but is usually 2–3 days.

Period of communicability

Cholera is communicable during the symptomatic phase and up to 2–3 days after recovery. In rare cases, the disease is communicable for months following recovery. Asymptomatic carriers are common.

Reservoirs

Humans are the main reservoir for cholera bacteria, although environmental reservoirs have recently been found in association with copepods or other zooplankton in brackish water or estuaries.

Epidemiology

Disease burden

In 2007, the World Health Organization (WHO) recorded 177 963 global cases (4031 deaths, case-fatality ratio [CFR] 2.3%). That year, officially reported cases from Asia increased 4.5-fold over previous years. India accounted for 23% of all cases notified from Asia. In countries such as Bangladesh, where cholera epidemics are common, incidence of the disease peaked during the floods that affected parts of South-East Asia in the monsoon season (*1*).

In Sri Lanka, the last case of cholera was reported in January 2003, in Jaffna (*2*). However, the proximity of the country to India, where cholera is endemic, places Sri Lanka at continued risk.

Geographical distribution

All areas in the country are considered to be at risk of transmission.

Seasonality

Generally, cases occur mainly during the wet seasons (starting in May–June and November–December in Sri Lanka, corresponding to monsoons). Climate fluctuations related to warming of oceans, such as El Niño, can be associated with an increase in cholera cases.

Alert threshold

Any suspected case of cholera must be investigated.

Outbreaks

The last outbreaks of cholera in Sri Lanka occurred from September 1997 to July 2000, and July 2002 to January 2003. Recent, notable outbreaks have been reported in Angola, Iraq, Sudan and Zimbabwe.

Risk factors for increased burden

Population movement

Population migration increases transmission of the infectious agent for cholera.

Overcrowding

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

Poor access to health services

Lack of health care and proper case management increase the risk of case fatality, because early detection and containment of cases (isolation facilities) are needed to reduce transmission. Lack of outbreak reporting, as well as poor surveillance and monitoring, are further obstacles to effective disease prevention and control.

Food shortages

Malnutrition increases the risk of severe diarrhoeal illness, with marked fluid and electrolyte disturbances, leading to further malnutrition. Prolonged diarrhoea in malnourished patients may increase the need for fluids, electrolytes and nursing.

Lack of safe water, poor hygiene practices and poor sanitation

Lack of safe water, poor hygiene practices and poor sanitation are the most important risk factors. Cultural practices may also be a risk factor; for example, funeral

practices in which meals are prepared by those who have handled the body without stringent hygiene are particularly hazardous.

Prevention and control measures

Case management

About 20% of those who are infected with cholera develop acute, watery diarrhoea, and 10–20% of these individuals also develop vomiting. If these patients are not treated promptly and adequately, 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 by rehydration; if this is applied appropriately, CFRs should remain below 1%. The mainstay of the case management of cholera is dehydration treatment using oral replacement salts (ORS) or intravenous (IV) fluids such as Ringer's lactate. Large volumes of ORS can be required, and IV rehydration is used for severe cases only.

Use of antibiotics (e.g. doxycycline or tetracycline) is not essential for disease treatment, but may be used with rehydration in severe cases to reduce the volume of diarrhoea, the duration of symptoms, the period of *V. cholerae* excretion, and the time spent in cholera treatment centres. Antimicrobial susceptibility should be assessed to select the appropriate antibiotic. Resistance can develop during an outbreak, so susceptibility should be reassessed periodically.

Measures that are effective in preventing the spread of cholera include strict handwashing practices, proper disinfection of articles and linen used by cholera-infected patients, and adequate hygiene during funeral preparations.

Prevention

Safe water, adequate sanitation and health education for proper hygiene and food safety are important prevention measures. Infants are best protected by exclusive breastfeeding for 6 months and continued breastfeeding for 2 years or beyond, with safe and adequate complementary foods introduced at 6 months. Infants who are artificially fed need specific support and close monitoring.

Safe drinking-water

Prevention of cholera in any community relies mainly on adequate water, sanitation and hygiene; this requires the following:

- *Provision of safe drinking-water.* The WHO minimum emergency, standard provision, is 20 l per person per day.

- *Provision of adequate facilities for disposal of human waste*. The minimum emergency standard is one latrine for every 20 people.
- *Provision of adequate supplies for hand-washing, bathing and laundry needs*. The minimum emergency standard is 250–500 g of soap per person per month.
- *Promotion of hygiene* – including use of latrines and prevention of human defecation on the ground, or in or near water; disposal of children's waste in latrines; thorough hand-washing before eating, after defecation, before food preparation, and after cleaning children or changing their diapers.
- *Promotion of food safety* – through:
  - provision of clean, appropriately cooled and spatially adequate food storage facilities (for both uncooked and cooked food, and clean cooking utensils), and adequate quantities of water and fuel for cooking and re-heating
  - promotion of "five keys to safer food" – keep clean, separate raw and cooked food, cook food thoroughly, keep food at safe temperatures, use clean water and cooking tools.
- *Promotion of breastfeeding* – by providing information on the importance and protective qualities of breastfeeding for all infants and young children, and especially for those who are ill.

Immunization

Oral cholera vaccine (OCV) should be used as an addition to the normal recommended cholera prevention and control measures. The vaccine can be given pre-emptively if the population at risk can be accurately identified. Currently, killed whole-cell *V. cholerae* O1 recombinant B-subunit of cholera toxoid (WC/rBS) is the only WHO pre-qualified OCV. WC/rBS is available for use in public health.

The relevance of OCVs should be assessed on a case-by-case basis, using the WHO decision-making tool (*3*).

Old, parenteral cholera vaccine should not be used; it has never been recommended by the WHO.

For more specific information on cholera vaccines and their use, contact the Global Task Force on Cholera Control at WHO headquarters (HQ) (e-mail: cholera@who.int).

Epidemic control

Because the risk of cholera outbreaks is high in overcrowded settings, it is important to be prepared. Disease surveillance systems must be sensitive enough to

detect major outbreaks. Ideally, cholera treatment units should be prepared before any outbreak emerges in high-risk settings.

Health authorities should be informed immediately if one or more suspected cases are identified. The outbreak should then be confirmed, following WHO guidelines (*4*).

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. At least 10 cases are required to confirm a cholera outbreak and to identify antibiotic susceptibility. Once the outbreak has been confirmed, it is not necessary to obtain laboratory confirmation for every patient.

Case-management and control activities should start immediately; they should not be delayed by waiting for laboratory results. It is important to:

- ensure prompt case management and confirm the cholera diagnosis
- isolate severe cases in cholera treatment centres
- find and treat the infection source as soon as possible
- provide adequate community involvement, health education and active case-finding
- set up ORS corners 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.

Interagency diarrhoeal disease kits (containing four separate modules) are available for preparedness or response (*5*). Complete kits are best for preparedness, but each module may be ordered separately, according to local availability of components. The minimum shelf-life of all kit components is 3 years, and no cold chain is required.

Agencies outside WHO may purchase kits or modules through The Medical Export Group BV (MEG), Gorinchem, The Netherlands (e-mail: info@meg.nl or see http://www.meg.nl). Within the WHO, they can be purchased through a GSM requisition (see "kits" in the catalogue on the WHO intranet).

References

1. Cholera, 2007. *Weekly Epidemiological Record*, 1 August 2008, Vol. 83, 31:269–284. Geneva, World Health Organization (WHO) (http://www.who.int/wer/2008/wer8331.pdf, accessed 9 August 2010).
2. *Quarterly Epidemiological Bulletin, Epidemiology Unit*. Sri Lanka, Ministry of Health, Nutrition & Welfare (http://www.epid.gov.lk/Epidemiological%20Bulletin.htm, accessed 9 August 2010).
3. *Oral cholera vaccine use in complex emergencies: what next? Report of a WHO meeting, Cairo, 14–16 December 2005*. Geneva, WHO, 2006 (http://www.who.int/topics/cholera/publications/en, accessed 9 August 2010).
4. *Cholera outbreak: assessing the outbreak response and improving preparedness*. Geneva, WHO, 2004 (http://www.who.int/cholera/publications/cholera_outbreak/en/index.html, accessed 9 August 2010).
5. *Interagency diarrhoeal disease kits — information note*. Geneva, WHO, February 2009 (http://www.who.int/topics/cholera/materials/en, accessed 9 August 2010).

Further reading

- Prevention of foodborne disease: five keys to safer food [various resources]. Geneva, WHO, 2009 (http://www.who.int/foodsafety/consumer/5keys/en/index.html, accessed 10 August 2010).
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CD-WGE technical focal point: Department of Public Health and Environment (PHE)

DENGUE

Description

Clinical description

Dengue is a mosquito-borne viral disease with a wide clinical spectrum that includes severe and non-severe clinical manifestations. After the incubation period, the illness begins abruptly with three phases: febrile, critical and recovery. A revised clinical classification according to levels of severity (dengue fever with or without warning signs, and severe dengue) is proposed to replace dengue fever, dengue haemorrhagic fever (DHF) and dengue shock syndrome (DSS), as shown in Figure 1.

Infectious agent

The *Dengue virus* complex comprises four antigenically related but distinct viruses, designated *Dengue virus* serotypes 1–4. Serotypes 2 and 3 are currently the most common in Sri Lanka. Recovery from infection by one of these serotypes confers lifelong immunity against that serotype, and partial or transient immunity to the other serotypes. However, the risk of developing severe dengue increases with sequential infection with different serotypes, through a process called antibody-dependent enhancement.

Case definition

Suspected

A suspected case of dengue is one that is compatible with the clinical description.

Probable

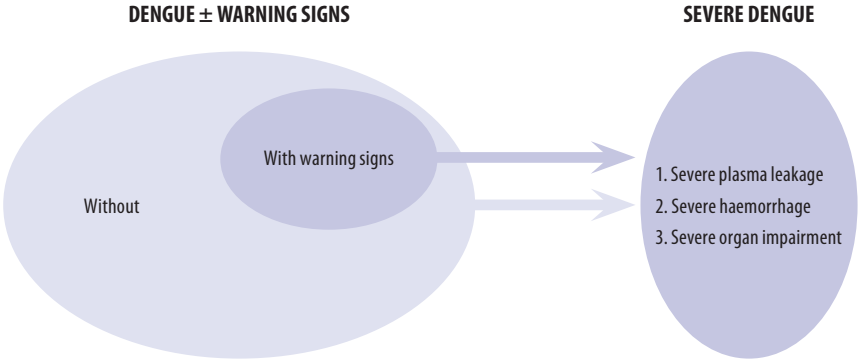
A probable case of dengue is one that is compatible with the clinical description, plus:

- supportive serology (a reciprocal haemagglutination-inhibition antibody titre of ≥ 1280, a comparable IgG enzyme immunoassay [EIA] titre, or a positive IgM antibody test in a late acute or convalescent-phase serum specimen)
- occurrence at the same location and time as other confirmed cases of dengue fever.

Confirmed

A confirmed case of dengue is compatible with the clinical description and laboratory confirmation through at least one of the following:

Figure 1. **Suggested dengue case classification and levels of severity (*1*)**



CRITERIA FOR DENGUE ± WARNING SIGNS

Probable dengue
Live in/travel to dengue-endemic area.
Fever and two 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[a]

- 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

[a] 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

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

Source: Reproduced from *Dengue: Guidelines for diagnosis, treatment, prevention and control.* New edition 2009. Geneva, WHO, 2009. Please see also page 41 of *Communicable disease epidemiological profile of Côte d'Ivoire* (http://www.who.int/diseasecontrol_emergencies/publications/cotedivoire/en/index.html, accessed 2 September 2010).

- isolation of the *Dengue virus* from serum, plasma, leukocytes or autopsy samples
- expression of at least a fourfold rise or fall in reciprocal IgG or IgM antibody titres to one or more *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
- detection of viral genomic sequences in autopsy tissue, serum or cerebrospinal fluid (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* (*Stegomyia*), principally *A. aegypti.* Vector surveillance demonstrates that *A. aegypti* is more common than *A. albopictus* in Sri Lanka. A monkey to mosquito cycle has been shown to occur in forests of South-East Asia and western Africa, but monkeys have not been shown to be an important reservoir for transmission to humans.

Incubation

The incubation period for dengue is commonly 4–7 days (range 3–14 days).

Period of communicability

There is no direct person-to-person spread of dengue. Patients are infective for mosquitoes from shortly before the onset of fevers 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

Globally, 2.5 billion people live in at-risk regions. There are an estimated 50 million cases of dengue fever each year, with an annual estimate of 500 000 DHF cases that require hospitalization, and about 2.5% of these patients die (*2*). Dengue is endemic in Sri Lanka; case-fatality ratios (CFRs) remain below 1% and there are increasing numbers of adult cases (Table 1). The World Health Organization (WHO) encourages enhanced global surveillance through participation in DengueNet (*3*).

Table 1. **Reported dengue cases and deaths by year, Sri Lanka, 2002–06 (*2*)**

Year	Reported cases	Reported deaths	Case-fatality ratio (%)
2006	5 646	46	0.81
2005	5 950	27	0.45
2004	15 408	88	0.57
2003	4 749	32	0.67
2002	8 931	64	0.72

Geographical distribution

Cases of dengue are reported from all over Sri Lanka, but the western part of the country is most affected. Sri Lanka's Ministry of Healthcare and Nutrition identified the following areas as being at high risk in 2009:

- Anuradhapura
- Batticaloa (Batticaloa, Eravur)
- Colombo (Boralesgamuwa, Dehiwala, Kaduwela, Kolonnawa, Maharagama, Nugegoda)
- Gampaha (Biyagama, Gampaha, Mahara, Wattala)
- Hambantota (Hambantota, Katuwana)
- Jaffna (Kopay, Tellippilai)
- Kalutara (Horana, Panadura)
- Kegalle (Kegalle, Mawanella, Yatiyantota)
- Kurunegala (Kurunegala, Polgahawela)
- Matara
- Puttlam (Chilaw, Dankotuwa, Mundel, Puttlam)
- Trincomalee (Trincomalee).

Seasonality

Generally, one peak DHF period, in June and July, is associated with the south-western monsoon that starts in late April and results in maximum annual rainfall. Another peak is reached at the end of the year, associated with the north-eastern monsoon rains that prevail from October to December.

Alert threshold

The alert threshold for dengue is more cases than are usually reported for the time of year.

Outbreaks

Large epidemics have occurred globally. An unprecedented pandemic occurred in 1998, when 56 countries reported 1.2 million cases of dengue and DHF (*4*). Recent outbreaks in Sri Lanka are shown in Table 2.

Table 2. **Recent outbreaks of dengue in Sri Lanka**

Year	Details of outbreak
1965–68	Of 51 cases of DHF, there were 15 deaths. Most towns throughout the country were affected during this outbreak, but the greatest impact was felt in the western coastal belt.
1989	Of 203 clinically diagnosed cases, 20 deaths occurred (CFR = 9.8%).
1990	Of 1350 cases, 363 were serologically confirmed.
1996	There were 289 cases from the Kurunegala district, and focal outbreaks occurred in the provincial towns of Batticaloa, Galle and Kandy.
2004	Of 15 408 cases, 88 deaths occurred (CFR = 0.57%). Cases were reported from 25 districts, with 72% of cases and 78% of deaths from five cities (Colombo, Kandy, Gampaha, Kalutara and Kurunegala) (CFR = 0.4–1.1%).
2009	In early 2009, nearly 600 cases of dengue, including 5 deaths, were notified over a 3-week period, with most cases arising from the areas of Colombo, Embilipitiya, Gampaha, Kandy, Matale and Ratnapura.

CFR, case-fatality ratio; DHF, dengue haemorrhagic fever.

Risk factors for increased burden

Population movement

Dengue transmission is increased by movement of viraemic populations into nonendemic areas where the vector is present, movement into endemic areas, establishment of new human settlements, and the creation of temporary shelters where drinking-water is obtained from outside sources or from rainwater harvesting and storage in household containers.

Overcrowding

In emergency situations, 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

Without proper treatment, CFRs of severe dengue can exceed 20%.

Food shortages

Food shortages do not affect the risk of infection with dengue.

Lack of safe water, poor hygiene practices and poor sanitation

Lack of safe water, poor hygiene practices and poor sanitation are risk factors. Accumulation of water in discarded vessels and debris may become vector breeding sites.

Other

Breakdown of vector control activities increases risk of the disease.

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 the disease. Dengue 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). Patients should be reviewed daily, and instructed to return to the health-care facility urgently if warning signs emerge. Non-steroidal anti-inflammatory drugs (NSAIDs) such as aspirin are contraindicated. Local breeding sites should be investigated and destroyed.

Patients who have dengue fever with warning signs, or factors such as pregnancy, extremes of age, or other illness, should be managed as inpatients, with careful intravenous (IV) rehydration with isotonic solution as required (it is important not to overhydrate).

Severe dengue fever should be referred as a medical emergency for intensive inpatient treatment.

Patients should be protected from day-biting mosquitoes by screens or insecticide-treated nets, and the facility should be sprayed with residual insecticide to reduce onward transmission.

Prevention

As with chikungunya, the most effective, economical and safe way to prevent dengue is integrated vector control. This includes 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.

Biological and chemical control measures against both larvae and adults are needed. In urban areas, *Aedes* mosquitoes breed in artificial containers such as plastic cups, used tyres, broken bottles and flowerpots. The most effective ways to reduce larvae (and therefore the *Aedes* mosquito density) in the community are continued and sustained reduction of containers that may collect water, and covering and frequent draining of containers. Larvicides are also highly effective, but should be long lasting and preferably safe for use in drinking-water; for example, slow-release formulations of temephos, which is approved by the WHO Pesticide Evaluation Scheme (WHOPES). Insect growth regulators (e.g. pyriproxyfen) are also available, and are safe and long lasting. Biological control includes slow-release *Bacillus thuringiensis* H-14, the crustacean *Mesocyclop* and several species of larviforous fish. Reducing the adult mosquito density by spraying with insecticide from trucks and aircraft or from portable backpack units can be effective if completed regularly and efficiently, although such operations are difficult to sustain.

Where insecticides or larvicides are used, regular monitoring of vector susceptibility is necessary to ensure the appropriate choice of nonresistant chemical. All mosquito control efforts should be accompanied by active monitoring and surveillance of the natural mosquito population, to determine the impact of the programme. Piped water supplies, well-maintained drainage and efficient systems for removal of domestic waste can also have a large impact. *Aedes* mosquitoes mainly bite during the day, particularly after sunrise and before sunset.

At household level, mosquito bites can be avoided when people:

- wear full-sleeve clothes and long dresses or trousers to cover their limbs
- use repellents (taking care when using them on small children and the elderly)
- use mosquito coils, repellents and electric vapour mats during the daytime
- use mosquito nets to protect babies, older people and others who may rest during the day; such nets are more effective when treated with insecticides
- hang insecticide-treated curtains at windows or doorways to repel or kill mosquitoes
- use fly screens on doors and windows.

Health centres are essential as an alert network, and for early diagnosis and treatment of suspected cases.

Immunization

Vaccines are not available, but several candidate vaccines are in development.

Epidemic control

The mainstay of epidemic control is vector control. Larval habitats of *Aedes* mosquitoes should be eliminated in urban or periurban areas, and larvicide applied to potential habitats. Stored water containers should always be kept covered. Water should be emptied from containers such as coolers, tanks, barrels, drums and buckets when these are not in use. Every other day, water should be removed from sites where it unavoidably accumulates and cannot be covered (e.g. refrigerator drip pans).

Spraying of insecticide from trucks or aeroplanes to kill adult mosquitoes should be 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, together with case definition, clinical protocols, staff information and education, and sufficient supplies. Hospital beds should be screened with insecticide-treated nets, and areas inside the hospital should be sprayed with insecticide.

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CD-WGE technical focal points: Departments of Global Alert and Response (GAR), Control of Neglected Tropical Diseases (NTD) and the Special Programme for Research and Training in Tropical Diseases (TDR)

DIARRHOEAL DISEASES

Description

Clinical description

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

- acute, watery diarrhoea
- acute, bloody diarrhoea (dysentery)
- persistent diarrhoea, lasting 14 days or more.

Infants under five years of age are particularly vulnerable.

Infectious agent

Bacteria

The bacteria that cause the most severe outbreaks of diarrhoeal illness are *Shigella dysenteriae* type 1 (Sd1, see Bacillary dysentery section) and *Vibrio cholerae* (see Cholera section). In addition, some *Salmonella* species (see typhoid fever for *S.* Typhi and *S.* Paratyphi) and a few strains of *Escherichia coli* may cause disease.

Protozoa

The protozoa that cause diarrhoeal disease include *Entamoeba histolytica, Giardia lamblia,* and *Cryptosporidium parvum*.

Viruses

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

Case classifications

Acute, watery diarrhoea in childhood

Clinical case definition

Acute, watery diarrhoea is passage of three or more loose or watery stools in 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, bloody diarrhoea is diarrhoea with blood visible in the stool.

Laboratory criteria for diagnosis

Laboratory culture of stools may be used to confirm possible outbreaks of specific diarrhoea, such as Sd1, 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, where the patient continues to excrete the organism despite being asymptomatic, leading to further spread of disease.

Incubation period

Salmonella generally requires an incubation period of 8–48 hours, whereas the incubation period of *E. coli* is typically longer at 2–8 days. The disease duration 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 under 5 years of age, and symptoms may last for up to 1 week. The incubation period of *Campylobacter* is usually 2–5 days (range 1–10 days).

Period of communicability

Diarrhoea is communicable during the acute stage of the disease and for the duration of diarrhoeal faecal excretion. Chronic carriers can continue to excrete *Salmonella* for several months.

Reservoirs

Humans are the typical reservoirs for diarrhoea-causing organisms. Additionally, certain organisms are carried and transmitted by cattle (*C. parvum*, *Salmonella* spp. and some *E. coli*) and domestic animals (*C. parvum* and *Salmonella* spp.).

Epidemiology

Disease burden

Diarrhoeal diseases are an important public health problem in Sri Lanka. Although there has been a dramatic reduction in diarrhoea-specific mortality and case-fatality ratio (CFR) in Sri Lanka over the past two decades, the diarrhoea-specific morbidity ratio has remained high and static. Admission rates to hospitals were 676.1–961.3 per 100 000 admissions in 2005, making diarrhoeal disease the sixth leading cause of hospitalisation in Sri Lanka (*1*).

Diarrhoeal disease is still common in children in Sri Lanka. According to a demographic and health (DHS) survey in 2000, 7% of children under 5 years of age had suffered from at least one attack of diarrhoea in a two-week reference period. Prevalence is highest (13%) among children in the 6–11-month age group, due to the introduction of solid foods into the diet (*2*).

Seasonality

Numbers of hospital admissions due to diarrhoea increase from January to March. Surveillance data from Lady Ridgeway Hospital (Colombo) indicate a similar seasonal pattern in hospital admissions due to *Rotavirus*; this pattern is not seen in temperate countries (*1*).

Alert threshold

The alert threshold for diarrhoeal diseases is an increase in the number of cases above the expected number compared with the same period in previous years in a defined area.

Outbreaks

A food-poisoning outbreak was registered in the Maskeliya-Dickoya Ministry of Health (MOH) area in the Nuwara Eliya district on 18 February 2007. Approximately 300 people with nausea, vomiting and diarrhoea were treated at the Dickoya District Hospital (*3*).

Risk factors for increased burden

Population movement

In acute, complex emergencies that result in large population movements, diarrhoeal diseases can account for 25–40% of deaths, with 80% of them among children

under two years of age. There was an increase in reported cases of diarrhoea in camps for internally displaced people after the Asian tsunami (2004).

Overcrowding

Overcrowding facilitates transmission. Unplanned urbanisation that results in poor sanitary conditions is a challenging factor for diarrhoeal control in Sri Lanka.

Poor access to health services

Lack of health care and lack of proper case management lead to an increased CFR.

Food shortages

Malnutrition increases susceptibility to and severity of diarrhoeal disease, which in turn contributes to further malnutrition. A major risk factor for infant diarrhoea is lack of exclusive breastfeeding for the first 6 months of life and lack of continued breastfeeding (supplemented by safe and adequate complementary foods) for two years or more.

Lack of safe water, poor hygiene practices and poor sanitation

Lack of safe water, poor hygiene practices and poor sanitation are risk factors for diarrhoeal epidemics. In 2004, the percentage of the Sri Lankan population using improved drinking-water sources was 79% (98% in urban regions and 74% in rural areas), and 91% of the population was using adequate sanitation facilities (*4*). Common sources of infection in emergency situations are:

- contaminated water sources (e.g. due to 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

Diarrhoeal fatalities can be reduced through effective management of dehydration, particularly in children; this involves:

- preventing dehydration by giving the recommended home fluid and oral rehydration salts (ORS)
- treating dehydration with ORS for mild to moderate dehydration, or with intravenous (IV) fluids (Ringer's lactate) for severe dehydration
- administering antibiotics appropriate for the infectious agent.

Normal feeding can resume when vomiting has stopped. Breastfeeding should continue in infants and young children. Zinc supplementation for children can decrease the severity and duration of illness (recommended doses of zinc are 20 mg/day for 14 days for children up to 12 years of age; 10 mg/day for 14 days for infants under 6 months).

Prevention

Overall, prevention of diarrhoeal diseases depends on use of safe water, adequate sanitation and health education for hygiene and food safety. Infants are best protected from diarrhoeal disease by exclusive breastfeeding for 6 months and continued breastfeeding for 2 years or beyond, with safe and adequate complementary foods introduced at 6 months. Infants who are artificially fed need specific support and close monitoring.

Prevention of cholera in any community relies mainly on adequate water, sanitation and hygiene; this requires the following:

- *Provision of safe drinking-water*. The World Health Organization (WHO) minimum emergency, standard provision is 20 l per person per day.
- *Provision of adequate facilities for disposal of human waste*. The minimum emergency standard is one latrine for every 20 people.
- *Provision of adequate supplies for hand-washing, bathing and laundry needs*. The minimum emergency standard is 250–500 g of soap per person per month.
- *Promotion of hygiene* – including use of latrines and prevention of human defecation on the ground, or in or near water; disposal of children's excreta in latrines; thorough hand-washing before eating, after defecation, before food preparation, and after cleaning children or changing their diapers.
- *Promotion of food safety* – through:
  - provision of clean, appropriately cooled and spatially adequate food storage facilities (for both uncooked and cooked food, and clean cooking utensils), and adequate quantities of water and fuel for cooking and re-heating
  - promotion of "five keys to safer food" – keep clean, separate raw and cooked food, cook food thoroughly, keep food at safe temperatures, use clean water and cooking tools.
- *Promotion of breastfeeding* – by providing information on the importance and protective qualities of breastfeeding for all infants and young children, and especially for those who are ill.

Early detection and containment of cases are vital in reducing transmission.

Immunization

Immunization against *Rotavirus* is being considered in Sri Lanka, pending more data on disease burden and cost effectiveness.

Epidemic control

The health authorities should be informed immediately if an increase in the number of cases above the expected number is identified. The outbreak should be confirmed, following WHO guidelines (Annex 2). Proper case management and epidemic control activities should be implemented.

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Annexes:

- Annex 3: Flowcharts for the diagnosis of communicable diseases
- Annex 4: Safe water, sanitation and hygiene
- Annex 6: WHO fact sheets and information sources (cholera; child health in emergencies; diarrhoeal diseases; food safety; water, sanitation and health).

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 mucous membranes of the respiratory tract (respiratory diphtheria), 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 and 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 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

C. diphtheriae is the bacterial agent that causes diphtheria.

Case classification

Suspected case

Not applicable.

Probable case

A case is probable if one or more of the following is observed: laryngitis, pharyngitis or tonsillitis; an adherent membrane of the tonsils, pharynx or nose.

Confirmed case

A confirmed case is a probable case that is laboratory-confirmed, with isolation of *C. diphtheriae* from a clinical specimen; a more than fourfold rise in serum

antibody levels (but only if both serum samples were obtained before administration of diphtheria toxoid or antitoxin); or an epidemiological link to a laboratory-confirmed case.

Carrier

A carrier is confirmed by the presence in the person of *C. diphtheriae* in the nasopharynx, without symptoms. Individuals who are asymptomatic carriers (i.e. are culture-positive for *C. diphtheriae*, but do not meet the clinical description) should not be reported as probable or confirmed cases of diphtheria.

Mode of transmission

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

Incubation period

The incubation period is usually 2–5 days, but can be longer.

Period of communicability

The disease is communicable until viable *C. diphtheriae* have disappeared from discharges and lesions. This usually takes 2 weeks or less, and rarely more than 4 weeks. Chronic carriers are rare, but can shed *C. diphtheriae* for 6 months or more. The disease is usually not contagious within 48 hours of commencement of treatment with antibiotics.

Reservoirs

Humans are the reservoirs for *C. diphtheriae.*

Epidemiology

Disease burden

No cases have been reported in Sri Lanka since 2001, when there were 15 cases (previous to that time, 1 case was reported in 1996 and 3 cases in 1997) (*1*). Coverage with the full three doses of the diphtheria–tetanus–pertussis vaccine (DTP3) has been high (≥ 97%) for over a decade.

Geographical distribution

Globally, 4190 cases of diphtheria were reported in 2007, and there were an estimated 5000 deaths from the disease in 2004 (*2*, *3*). Areas of particular concern are Afghanistan, Eastern Europe (previous Soviet Union states) and the Pacific.

Seasonality

There is no seasonality; however, transmission generally occurs throughout the year in warm climates, and increases in the colder months in temperate climates.

Alert threshold

The alert threshold for diphtheria is that any suspected or probable case must be investigated.

Epidemics

Devastating diphtheria epidemics affecting mainly children have been described from many countries throughout history. In countries endemic for diphtheria, the disease occurs mostly as sporadic cases or in small outbreaks. No recent outbreaks or epidemics have been reported from Sri Lanka.

Risk factors for increased burden

Population movement

Large movements of nonimmunized 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 of diphtheria.

Poor access to health services

Poor access to immunization services will lead to low vaccination coverage (defined as < 80%) for diseases that are included in the routine Expanded Programme on Immunization, thereby increasing the proportion of susceptible individuals and the risk of outbreaks.

Food shortages

Food shortages are not relevant to the risk of infection with diphtheria.

Lack of safe water, poor hygiene practices and poor sanitation

Outbreaks of toxigenic, cutaneous diphtheria have been described; lack of access to clean water and poor hygiene (coupled with overcrowding) may be risk factors.

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 cornerstones of therapy for diphtheria. The antitoxin neutralizes the diphtheria toxin before its entry into cells. Therefore, diphtheria antitoxin should be administered as soon as a presumptive diagnosis has been made.

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

- termination of toxin production
- decreased local infection
- prevention of spread of the organism to uninfected people.

Treatment and control activities should start immediately; they should not be delayed by waiting for laboratory results.

Patients

Diphtheria antitoxin (20 000–100 000 units) should be given in a single intramuscular (IM) or intravenous (IV) dose immediately after throat swabs have been taken and sensitivity-testing has been performed. The dose administered depends on the extent of disease; guidelines should be followed (*2*). Where feasible, the IV route is preferred, particularly for delayed treatment and extensive or severe disease. This is because availability of the antitoxin in the circulation is markedly delayed with IM administration, and, depending on the manufacturer, the volume of IM injection required can be large.

In addition, patients should receive procaine penicillin IM (25 000–50 000 units/kg per day for children; 1.2 million units per day 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.

Patients should then be given 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, to complete a total course of 14 days of antibiotic treatment.

Isolation

For pharyngeal diphtheria, strict isolation of patients is necessary.

For cutaneous diphtheria, strict isolation of patients is not necessary. However, barrier precautions must be observed, to prevent contact with cutaneous lesions.

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 should be instigated for anyone who has come into 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 patient, as well as health-care staff exposed to oral or respiratory secretions of a patient.

All close contacts must receive either a single IM dose of benzathine penicillin G (600 000 units for children under 6 years of age; 1.2 million units for persons over 6 years of age) or a 7-day course of oral erythromycin. If the nasal or throat culture is positive for toxigenic *C. diphtheriae*, antibiotics should be given, as described above under "Patients". Close contacts should also receive a dose of age-appropriate diphtheria toxoid-containing vaccine, unless a dose has been received within the previous 12 months.

Prevention

Prevention consists of:

- 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

The current routine, national immunization schedule in Sri Lanka includes four doses of DTP-containing vaccines given at 2, 4, 6 and 18 months; followed by diphtheria–tetanus (DT) vaccine at 5 years; and a combination of diphtheria and tetanus toxoid with reduced diphtheria content (Td) at 10–14 years. In nonepidemic situations, the approach to a previously unvaccinated population is to provide a full 3-dose primary series (the first two doses separated by 1–2 months, and the

third given 6–12 months after the second). For this vaccination, DTP should be administered to children under 7 years of age and Td to children over 7 years of age.

Immunization in epidemic situations is discussed below under "Epidemic control".

Epidemic control

Control of an epidemic involves:

- informing health authorities if one or more suspected cases are identified
- confirming the suspected outbreak, following World Health Organization (WHO) guidelines (Annex 2)
- investigating any probable case; checking whether it fulfils the case definition and recording the date of onset, age and vaccination status
- confirming the diagnosis by collecting both nasal and pharyngeal swabs for culture and swabs from any wounds or skin lesions (if appropriate facilities are available, the biotype and toxigenicity of *C. diphtheriae* should be determined)
- identifying close contacts and defining population groups at high risk; adult contacts must avoid contact with children and must not be allowed to undertake food handling until proven not to be carriers
- implementing outbreak response measures; priority should be given to case management and immunization of population in areas not yet affected, but to which the outbreak is likely to spread
- immunizing the population at risk as soon as possible, as outlined below.

In an outbreak involving adults, immunization of adults should take priority; however, good child immunity is important because children (especially those who are underimmunized) may act as transmission agents. Immunization procedures should be repeated after 1–2 months to provide at least two doses of diphtheria toxoid-containing vaccine (Td for children aged 7 years or more and for adults; DT for children younger than 7 years; if Td is unavailable, DT can be used for epidemic control). A third dose should be provided after 6–12 months for individuals without a known history of previous primary immunization.

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

HEPATITIS A AND E

Description

Hepatitis is a broad term for inflammation of the liver; it has a number of infectious and noninfectious causes. Two of the viruses that cause infectious hepatitis – *Hepatitis A virus* (HAV) and *Hepatitis E virus* (HEV) – are more prevalent in developing countries, and can be transmitted through water and food.

Clinical description

Clinical description is similar for both HAV and HEV. Infection occurs in childhood asymptomatically. Onset of illness in adults is usually abrupt, with fever, malaise, anorexia, nausea and abdominal discomfort, followed within a few days by jaundice. The disease varies in clinical severity from a mild illness lasting 1–2 weeks to a severely disabling disease lasting several months. Prolonged, relapsing hepatitis for up to 1 year occurs in 15% of cases. In general, severity increases with age.

Occasionally, extensive necrosis of the liver occurs during the first 6–8 weeks of illness. In such cases, high fever, marked abdominal pain, vomiting, jaundice and hepatic encephalopathy (with coma and seizures) are the signs of fulminant hepatitis, leading to death in 70–90% of patients.

A person with pre-existing chronic liver disease is at greater risk of fulminant HAV.

The case-fatality ratio (CFR) is low (0.1–0.3%); however, in pregnant women it can rise to 20% for those infected with HEV during the third trimester of pregnancy.

Infectious agent

HAV is a positive-stranded ribonucleic acid (RNA) picornavirus and is a member of the *Picornaviridae* family.

HEV is a spherical single-stranded RNA hepevirus and is a member of the *Hepeviridae* family.

Case classification

Specific serologic testing is required to distinguish the two cases. Hepatitis E should be suspected in outbreaks of waterborne hepatitis occurring in developing countries, especially if the disease is more severe in pregnant women, or if hepatitis A has been excluded. If laboratory tests are not available, epidemiologic

evidence can help in establishing a diagnosis. HEV infection tends to be more severe than HAV infection.

Suspected case

A suspected case of hepatitis is one that is compatible with the clinical description.

Confirmed case

A confirmed case of hepatitis is one that is laboratory-confirmed. The laboratory conducts a positive serology test for IgM anti-HAV/HEV. A rapid antigen-detection test to confirm HEV is under development.

Mode of transmission

HAV and HEV are both transmitted by the faecal–oral route. Faecally contaminated water is the most common vehicle of transmission.

Hepatitis A

Hepatitis A is transmitted:

- from person to person, via the faecal–oral route
- by food contaminated by infected food handlers or transmitted by undercooked or raw food, including molluscs harvested from contaminated waters
- through injecting and non-injecting drug use, and occasionally also through sexual contact (anal–oral)
- (rarely) through transfusion of blood and clotting factors from viraemic donors.

Hepatitis E

Hepatitis E is transmitted:

- primarily through faecally contaminated drinking-water (epidemic disease)
- from person to person probably through the faecal–oral route; secondary cases within the household of an infected person are uncommon during outbreaks
- through raw or uncooked shellfish (endemic disease)
- as a zoonotic infection, since several nonhuman primates, as well as pigs, cows, sheep, goats and rodents are susceptible to this virus.

Incubation period

The incubation period for hepatitis is shown in Table 1.

Table 1. **Incubation period for hepatitis**

	HAV	HEV
Range	15–50 days	15–64 days
Average	28–30 days	26–42 days

Period of communicability

Hepatitis A

- Maximum infectivity occurs in the latter half of incubation and a few days after the onset of jaundice.
- Prolonged viral excretion in infants and children has been documented for up to 6 months.

Hepatitis E

- The infectivity is unknown.
- The virus has been detected in stools 14 days after the onset of jaundice and approximately 4 weeks after ingestion of contaminated food or water, where it persists for about 2 weeks.

Reservoirs

Humans are the natural hosts, although HEV infection (and rarely HAV) occurs in some nonhuman primates. HEV infection has also been described in cows, sheep and goats, and there is a high prevalence of anti-HEV in some populations, making them sources of zoonotic infections in humans.

Epidemiology

Disease burden

There is no information on the national burden of hepatitis in Sri Lanka.

Hepatitis A

- Worldwide, HAV can be characterized by high, medium or low levels of endemicity. Levels of endemicity correlate with hygienic and sanitary conditions of each geographic area.

- In areas of high endemicity, adults are usually immune and epidemics are uncommon.
- Infection is common and occurs at a young age when sanitation is poor in the environment. The disease is most common among children and young adults.
- Most infections occur in childhood and are asymptomatic or unrecognized; therefore this group is important in transmission of the infection.

Hepatitis E

- HEV is the major cause of non-A non-B enterally transmitted hepatitis worldwide.
- In endemic areas, clinically evident disease occurs in young to middle-age groups (15–40 years). Lower rates in younger age groups may be due to sub-clinical or anicteric infection (i.e. with no jaundice).
- HEV infection tends to be more severe than HAV infection.

Geographical distribution

Epidemic and sporadic cases of hepatitis occur over a wide geographical area, particularly where inadequate environmental sanitation prevails.

- Outbreaks of hepatitis A tend to be cyclical.
- Outbreaks of HEV are more common in parts of the world with hot climates and are rare in temperate climates.
- HEV infections account for about 50% of acute sporadic cases in some highly endemic areas, and HEV is the single most important cause of acute clinical hepatitis among adults throughout central and South-East Asia.
- Viral hepatitis has been reported from all districts in Sri Lanka (*1*).

Seasonality

Hepatitis is 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 or jaundice syndrome.

Outbreaks

In 2007, an epidemic of hepatitis A broke out in the Gampola area in the Kandy district of Sri Lanka. About 200 people were admitted to a hospital in Gampola. Their cases were thought to result from consumption of contaminated water. People in and around Gampola, Pussellawa, Tholuwa and Tundeniya do not have access to safe drinking-water. Cases peaked in the second and third quarter of 2007 (Table 2).

Table 2. **Number of reported suspected viral hepatitis cases by quarter (Q) 2007, 2008, Sri Lanka (*1*)**

	1st Q 2007	2nd Q 2007	3rd Q 2007	4th Q 2007	1st Q 2008	2nd Q 2008	3rd Q 2008
Suspected viral hepatitis cases	750	2247	2019	868	635	500	413

Risk factors for increased burden

Population movement

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

Overcrowding

Overcrowding facilitates transmission of hepatitis.

Poor access to health services

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

Food shortages

Malnutrition increases gastrointestinal tract susceptibility to the invasiveness of the virus and of disease severity.

Lack of safe water, poor hygiene practices, and poor sanitation

Lack of safe water, poor hygiene and inadequate sanitation greatly increase the risk of infection.

Prevention and control measures

Case management

Case management should be supportive, treat the symptoms and be aimed at maintaining adequate nutritional balance.

Since there are no specific antiviral drugs against HAV and HEV, prevention remains the best form of control.

Prevention

Almost all hepatitis infections are spread by the faecal–oral route; therefore, good personal hygiene, high-quality standards for public water supplies and proper disposal of sanitary waste lower the prevalence of infection. Prevention in infants and young children is closely correlated with early and exclusive breastfeeding for the first 6 months of life, followed by continued breastfeeding with adequate and safe complementary foods for 2 years and beyond. Infants who are artificially fed need close monitoring and support.

The public should be educated on good hygiene and proper sanitation, as outlined below, with an emphasis on the importance of hand-washing and safe food handling practices, especially of shellfish.

For travellers to highly endemic areas, the usual elementary food hygiene precautions are recommended. These include avoiding drinking-water or ice of unknown purity, and avoiding eating uncooked shellfish or uncooked fruits or vegetables that are not peeled or prepared by the traveller.

Prevention of hepatitis in any community relies mainly on adequate water, sanitation and hygiene; this involves:

- *Provision of safe drinking-water*. The World Health Organization (WHO) minimum emergency, standard provision is 20 l per person per day.
- *Provision of adequate facilities for disposal of human waste*. The minimum emergency standard is one latrine for every 20 people.
- *Provision of adequate supplies for hand-washing, bathing and laundry needs*. The minimum emergency standard is 250–500 g of soap per person per month.
- *Promotion of hygiene* – including use of latrines and prevention of human defecation on the ground, or in or near water; disposal of children's excreta in latrines; thorough hand-washing before eating, after defecation, before food preparation, and after cleaning children or changing their diapers.
- *Promotion of food safety* – through:
    - provision of clean, appropriately cooled and spatially adequate food storage facilities (for both uncooked and cooked food, and clean cooking utensils), and adequate quantities of water and fuel for cooking and re-heating

- promotion of "five keys to safer food" – keep clean, separate raw and cooked food, cook food thoroughly, keep food at safe temperatures, use clean water and cooking tools.

- *Promotion of breastfeeding* – by providing information on the importance and protective qualities of breastfeeding for all infants and young children, and especially for those who are ill.

Immunization

Hepatitis A

*A*t least four inactivated vaccines are presently available commercially and their use is in line with WHO recommendations. Safety of the vaccines has not been determined during pregnancy. Passive immunization (by IgG) is also available. Neither active nor passive immunization is recommended for controlling community outbreaks.

Hepatitis E

Vaccines and specific immunoglobulin preparations are currently under development.

Epidemic control

Outbreaks of both HAV and HEV have been successfully controlled by chlorination of water supplies. To control an epidemic it is necessary to:

- determine the mode of transmission, whether by person to person or by common vector (vehicle)
- identify the population exposed to increased infection risk
- eliminate common infection sources
- improve sanitary and hygienic practices to eliminate faecal contamination of food and water.

Hepatitis A

Because most infections occur in young people in Sri Lanka, and illness is usually mild or asymptomatic, clinical hepatitis A is generally a minor public health problem and large-scale hepatitis A vaccination programmes are not recommended (*2*).

Hepatitis E

Immune globulin prepared from plasma collected in HEV-endemic areas has not been effective in preventing clinical disease during HEV outbreaks.

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Annexes:

- Annex 4: Safe water, sanitation and hygiene.
- Annex 6: WHO fact sheets and information sources (cholera; child health in emergencies; diarrhoeal diseases; food safety; water, sanitation and health).

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

HIV/AIDS

Description

Clinical description

Acquired immunodeficiency syndrome (AIDS) is the late clinical stage of human immunodeficiency virus (HIV) infection, and is defined as an illness characterized by one or more indicator diseases. Presentation may include, but is not limited to, persistent generalized lymphadenopathy, unexplained weight loss, persistent diarrhoea, recurrent oral ulceration, recurrent respiratory tract infections or herpes zoster.

Infectious agent

The infections agents are HIV-1 and HIV-2, which have similar epidemiological characteristics.

Case classification

HIV infection

In adults and in children 18 months or older, HIV infection is indicated by at least one of the following:

- a positive HIV antibody test (rapid or laboratory-based enzyme immunoassay [EIA]), confirmed by a second HIV antibody test relying on different antigens or of different operating characteristics
- a positive virological test for HIV or its components, confirmed by a second virological test obtained from a separate determination.

In children younger than 18 months, HIV infection is indicated by a positive virological test for HIV or its components, confirmed by a second virological test obtained from a separate determination taken more than four weeks after birth. Positive HIV antibody testing is not recommended for definitive or confirmatory diagnosis of HIV infection in children under 18 months of age.

Advanced HIV (including AIDS)

Advanced HIV, including AIDs, is indicated by at least one of the following:

- confirmed HIV infection and presumptive or definitive diagnosis of any stage 3 or stage 4 condition (Table 1)

Table 1. **Revised WHO clinical staging of HIV/AIDS for adults/adolescents and children, 2006 (*1*)**

Adults and adolescents	Children
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), neutropaenia (< 0.5×10^9/L) or chronic thrombocytopaenia (< 50×10^9/l)	
■ Unexplained severe weight loss (> 10% of presumed or measured body weight) ■ Severe bacterial infections (e.g. pneumonia, empyema, pyomyositis, bone or joint infection, meningitis or bacteraemia) ■ Acute necrotizing ulcerative stomatitis, gingivitis or periodontitis	■ Unexplained moderate malnutrition or wasting not adequately responding to standard therapy ■ Acute necrotizing ulcerative 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's 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's sarcoma ■ Cerebral or B-cell non-Hodgkin lymphoma	
■ HIV wasting syndrome ■ Recurrent severe bacterial pneumonia ■ Recurrent non-typhoidal *Salmonella* bacteraemia ■ Other solid HIV-associated tumours ■ Invasive cervical carcinoma ■ Atypical disseminated leishmaniasis	■ Unexplained severe wasting, stunting or severe malnutrition not responding to standard therapy ■ Recurrent severe bacterial infections (such as empyema, pyomyositis, bone or joint infection or meningitis but excluding pneumonia)

HIV/AIDS, human immunodeficiency virus/acquired immunodeficiency syndrome; WHO, World Health Organization.

- a CD4 count of less than 350 per 3 mm^3 of blood in an HIV-infected adult or child of 5 years or older
- a %CD4+ of less than 30 among those younger than 12 months
- a %CD4+ of less than 25 among those aged 12–35 months
- a %CD4+ of less than 20 among those aged 36–59 months.

Mode of transmission

HIV is transmitted:

- by sexual intercourse (vaginal or anal) with an infected partner, especially in the presence of a concurrent ulcerative or non-ulcerative sexually transmitted infection (STI)
- from an infected mother to her child during pregnancy, labour and delivery; or through breastfeeding (mother-to-child transmission)
- by transfusion of infected blood or blood products
- by contaminated needles or syringes through accidental injury of patients or service providers in occupational health, or those who are injecting drug users (IDUs).

Incubation period

The incubation period is variable. On average, the time from HIV infection to clinical AIDS is 8–10 years, although the disease may manifest in less than 2 years, or be delayed in onset beyond 10 years. Incubation times are shorter in resource-poor settings, infected infants and 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. The risk of infectiousness is increased by a high HIV viral load; however, the infection can be transmitted even in the presence of a low or undetectable viral load. The presence of concurrent STIs (particularly ulcerative) in either partner increases transmission risk.

Reservoirs

Humans are HIV reservoirs.

Epidemiology

Disease burden

The prevalence of HIV in Sri Lanka remains low, at less than 0.1%, with an estimated 3800 people living with HIV (*2*). The male:female ratio of HIV infection is 1.4:1. Populations at greatest risk include female sex workers. Lack of knowledge about HIV, and barriers to seeking services due to stigma and discrimination contribute to underdiagnosis. An estimated 85% of transmission in Sri Lanka is heterosexual, 11% in men who have sex with men and 3% from mother to child. Transmission by blood transfusion is negligible (0.003%). Cultural and religious beliefs are an obstacle to condom use (*3*).

Geographical distribution

No information on the geographical distribution of HIV is available for Sri Lanka.

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. Shortages of basic commodities for preventing HIV, such as condoms, can also increase the risk of transmission.

Poor access to health services

Lack of education on prevention and treatment of STIs and HIV, combined with poor access to condoms, increases the risk of transmission. Lack of testing and counselling services delays diagnosis. Failure to treat concomitant opportunistic infections or illnesses, and interruption or delayed commencement of antiretroviral therapy (ART) can increase the risk of illness and death among those already infected. Health-service quality may be compromised, with increased chances of transmission in the health-care setting, due to failure to observe universal precautions and to unsafe blood transfusion.

Food shortages

The need for food is paramount in emergency situations; this leads people to exchange sex for money and food, which can increase HIV transmission. Also, malnutrition adversely affects the health of people living with HIV/AIDS.

Lack of safe water, poor hygiene practices and poor sanitation

People living with HIV and AIDS may suffer from disease and may even die as a consequence of limited access to food, clean water and good hygiene, and a weakened immune system.

Prevention and control measures

Case management

Voluntary counselling and testing (provider-initiated and client-initiated) should be provided. People must be fully informed and must freely consent to testing. They should have both pre-test and post-test counselling. Confidentiality of the test result must be assured.

Increasing the capacity of voluntary counselling and testing through STI clinics in Sri Lanka is planned.

Essential care includes:

- psychosocial counselling and support
- disclosure, partner notification, and testing and counselling
- investigation for and treatment of (or prophylaxis against) opportunistic infections
- prevention and treatment of STIs
- vaccination against selected vaccine-preventable diseases (e.g. hepatitis B, pneumococcal, influenza)
- nutritional care (see above under "Food shortages")
- family planning
- needle–syringe programs and opioid substitution therapy
- adequate water, sanitation and hygiene.

Antiretroviral therapy

Sri Lanka has been offering free ART since late 2004 through a World Bank project. However, coverage was only about 14% in 2007 (*3*). Currently ART is only offered at the National STD/AIDS Control Programme Central Clinic in Colombo. Many seropositive individuals in rural areas (particularly in the conflict-affected north-east) have difficulty in accessing care.

Prevention

Universal precautions

Universal precautions against transmission of HIV include:

- washing hands thoroughly with soap and water, and using protective gloves and clothing when there is risk of contact with blood or body fluids
- using single-use needles and syringes
- ensuring safe handling and disposal of waste material, needles and other sharp instruments;
- using correct procedures for cleaning and disinfecting medical instruments between patients.

Blood transfusion safety

In an acute emergency, available resources for HIV testing should be devoted to ensuring a safe blood supply for transfusions.

Reduce sexual transmission

Sexual transmission can be reduced by providing high-quality condoms, accompanied by culturally sensitive promotion. The recent Behaviour Surveillance Survey in Sri Lanka found that condom use and knowledge about HIV risks among the groups most at risk is generally low (except among some subgroups of female sex workers).

Sexually transmitted infection management

STI management must include sex workers. The syndromic STI management approach requires partner notification and promotion of safer sex. Annual estimates of detected STI cases in Sri Lanka vary from about 60 000 to 200 000; however, only 10–15% of these are reported to government clinics (*4*).

Clinical management of rape

Clinical management in cases of rape requires a combination of:

- emergency contraception (if the person who has been raped presents within 5 days)
- presumptive treatment of STIs
- post-exposure prophylaxis (PEP) for HIV (for those presenting within 3 days)
- appropriate counselling and follow-up care.

Awareness and life skills education

Awareness and life skills education, especially for young people, helps to promote understanding of what does and does not constitute a mode of transmission. It also provides information on how and where to acquire condoms, how to acquire medical attention if necessary, and what is involved in basic hygiene.

Reduction of mother-to-child transmission of HIV

Mother-to-child (i.e. vertical) transmission of HIV can be reduced through primary prevention of HIV infection and prevention of unintended pregnancies, and where indicated, ART and lower risk infant feeding practices. A cumulative total of 33 infants have been infected through vertical transmission in Sri Lanka (*3*). The estimated number of HIV-infected women of child-bearing age in the country is less than 1000 (*2*).

Post-exposure prophylaxis in the health-care setting

PEP in the health-care setting includes administration of ART within 72 hours of exposure.

Prevention among injecting drug users

To prevent HIV transmission among IDUs requires ready access to, and safe disposal of, sterile needles, syringes and other injection equipment; education and counselling on risk-reduction techniques; provision of drug-dependence services; and easy access to STI and HIV/AIDS treatment. Current estimates of opiate users in Sri Lanka range from 30 000–240 000, of whom 2% are IDUs (*3*).

Physical protection of affected populations

Affected populations, especially women and children, should be protected from violence and abuse. This is an important principle of human rights, and is 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 the Expanded Programme on Immunization vaccines. Symptomatic HIV-infected children should NOT receive either bacille Calmette-Guérin (BCG) or yellow fever vaccine.

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Annexes:

- Annex 5: Injection safety.

CD-WGE technical focal point: Department of HIV/AIDS

INFLUENZA

This section covers both seasonal influenza (Section I) and avian influenza (Section II).

I. Seasonal influenza

Description

Clinical description

Influenza is a viral infection that affects mainly the nose, throat, bronchi and, occasionally, the lungs. Infection usually lasts for about a week, and is characterized by a sudden onset of high fever, aching muscles, headache and severe malaise, a nonproductive cough, sore throat and rhinitis.

Infectious agent

The infectious agents are the influenza viruses A, B and C. *Influenza virus A* has multiple subtypes, of which two (H1N1 and H3N2) are currently circulating widely among humans.

Case classification

Suspected case

A case is suspected when an individual presents with rapid onset of fever greater than 38 °C and a cough or sore throat, in the absence of other diagnoses. Diagnosis can be made when epidemiological characteristics or cases with similar clinical symptoms cluster or form an epidemic with typically short, intervening intervals (1–4 days), or when symptoms appear during a recognised seasonal epidemic period when reliable surveillance data are available.

Confirmed case

A confirmed case is one in which there is laboratory-confirmed antigen detection from respiratory specimens in a person with the clinical features of influenza. The laboratory confirmation can be made using one or more of the following:

- rapid diagnostic tests (for influenza viruses A and B), such as commercially available enzyme immunoassay (EIA) or neuraminidase assay; these have high specificity (90–95%) but low sensitivity (70–75%)

- polymerase chain reaction (PCR) – this detects both viable and nonviable influenza virus ribonucleic acid (RNA), and is generally more sensitive than culture; results can be available within a few hours
- virus culture is still regarded as the gold standard, but takes 2–10 days.

Antibody detection in serum specimens is rarely useful in immediate clinical management, but is significant for epidemiological purposes.

Mode of transmission

Influenza is transmitted as respiratory droplets at distances closer than 1 m and through direct, and perhaps indirect, contact (e.g. through fomites – inanimate objects such as dishes or metal tools that may carry infection – with hand contamination and self-inoculation into the nose or eye).

Incubation period

The incubation period is 1–7 days, but is usually 2 days.

Period of communicability

Influenza is communicable from 1–2 days before onset of symptoms, for up to 7 days after onset of illness in adults, and for up to 21 days after onset in children under 12 years.

Epidemiology

Disease burden

Influenza affects all countries and rapidly spreads around the world in seasonal epidemics. This imposes a considerable economic burden in the form of health-care costs and lost productivity. The impact of influenza in the developing world is less clear. Influenza outbreaks in the tropics, where viral transmission normally continues year round, tend to have high attack and case-fatality ratios (CFRs). Data collected during outbreaks should not be extrapolated to provide an annual burden of disease.

Geographical distribution

Influenza is found everywhere.

Seasonality

Sporadic influenza occurs throughout the year. In Sri Lanka in 2007, influenza A and B occurred in September and October; sporadic cases of influenza A occurred in November and sporadic cases of influenza B in December.

Alert threshold

An increase in the number of cases of influenza above that which is expected for a certain period of the year, or any increase in cases with a fever of unknown origin, should be investigated, after eliminating other causes. Accumulated surveillance data are required to determine the threshold for an influenza alert.

Outbreaks and epidemics

No recent outbreaks or epidemics of influenza have been reported in Sri Lanka.

Risk factors for increased burden

Population movement

Risk is increased by an influx of nonimmune 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 of influenza.

Poor access to health services

The CFR increases when access to health services is limited, particularly in the very young or very old, and in immunocompromised individuals.

Food shortages

Low birth weight, malnutrition, vitamin A deficiency and poor breastfeeding practices are important risk factors for the acquisition of influenza and development of complications.

Poor hygiene practices

Poor hand-washing may increase the spread of influenza.

Others

Low temperatures and dry conditions contribute to longer survival of the virus in the environment. Smoking is a risk factor for complications and prolonged illness.

Prevention and control measures

Case management

Early recognition of infection, and isolation of symptomatic patients using standard and droplet precautions (e.g. use of a surgical mask when in close contact with a patient) is recommended. For most of those infected, 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. Paracetamol may be used for management of fever, as clinically indicated. Antiviral drugs such as M2 inhibitors (amantadine or rimantadine for susceptible *Influenzavirus A*) and neuraminidase inhibitors (oseltamivir or zanamivir), given within the first 48 hours, can reduce symptoms and virus shredding.

Prevention

Public health measures, including respiratory etiquette (covering coughs and sneezes) and hand hygiene, help to prevent the spread of influenza seasonal infection during epidemics.

Immunization

The recommended composition of seasonal influenza vaccines is reviewed twice a year by the World Health Organization (WHO). These vaccines are the primary measure used to control seasonal influenza epidemics. The aim is to reduce disease morbidity and mortality in groups most at risk of severe illness and death; mainly the elderly, infants and young children, and those with chronic underlying conditions.

Surveillance

Influenza is under international surveillance. Countries are encouraged to report the disease activity and virus isolation to the WHO's FluNet. The official FluNet laboratory point of contact for Sri Lanka is based at the Medical Research Institute, Colombo.

II. Avian influenza

Description

Avian influenza, also known as "bird flu", is caused by influenza viruses that typically infect birds. Several of these have been able to cross the species barrier to infect humans. Only A(H5N1) and A(H7N7) have been reported to cause human death. However, these and other avian influenza viruses are a cause of concern to human health 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 Hong Kong 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 China, Thailand and Viet Nam. Since then, this virus has spread to wild birds and to poultry in Africa, Central Asia and Europe.

As of 11 March 2009, a total of 411 laboratory-confirmed cases, including 256 deaths, from human A (H5N1) avian influenza had been reported from 15 countries since November 2003. The countries involved were Azerbaijan, Bangladesh, Cambodia, China, Djibouti, Egypt, Indonesia, Iraq, Lao People's Democratic Republic, Myanmar, Nigeria, Pakistan, Thailand, Turkey and Viet Nam. No cases have been reported in Sri Lanka.

Clinical description

Common initial symptoms of H5N1 influenza include fever and cough. Lower respiratory tract symptoms are more common than upper respiratory tract symptoms. Symptoms progress rapidly to an acute respiratory distress syndrome. The overall CFR is high (63% as of March 2009).

Case definitions

The standardised case definitions given here were set in August 2006. They are applicable to the current phase 3 of pandemic alert, but may be altered as new information about the disease or its epidemiology becomes available. National authorities should formally notify only probable and confirmed H5N1 cases to the WHO.

Person under investigation

A person under investigation for avian influenza is someone being investigated by public health authorities for possible H5N1 infection.

Suspected H5N1 case

A suspected H5N1 case is presenting with unexplained acute lower respiratory illness with fever, cough, shortness of breath or difficulty breathing and at least one of the following exposures in the 7 days before onset of symptoms:

- close contact (within 1 m) 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 or 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
- handling of samples (animal or human) suspected of containing H5N1 virus in a laboratory or other setting.

Probable H5N1 case definition 1

The first definition of a probable H5N1 case is a case that meets the criteria for a suspected case plus at least one of the following criteria:

- infiltrates or evidence of an acute pneumonia on chest radiograph, plus evidence of respiratory failure (hypoxemia, severe tachypnoea)
- positive laboratory confirmation of an influenza A infection, but insufficient laboratory evidence for H5N1 infection.

Probable H5N1 case definition 2

The second definition of a probable H5N1 case is 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)

The WHO should be notified if a case of H5N1 is confirmed. A confirmed case is one in which a person meeting the criteria of a suspected or probable case has at

least one of the following laboratory results (from tests conducted in a national, regional or international influenza laboratory):

- 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 more than fourfold rise in neutralization antibody titre for H5N1, based on testing of an acute serum specimen (collected within 7 days of symptom onset) and a convalescent serum specimen; the convalescent neutralizing antibody titre must also be at least 1:80
- a microneutralization antibody titre for H5N1 of at least 1:80 in a single serum specimen collected at day 14 or later after symptom onset, and a positive result using a different serological assay (e.g. a horse red blood cell haemagglutination inhibition titre of ≥ 1:160 or an H5-specific positive result in an immunoblot).

Mode of transmission

Most human infection is reported to follow exposure to infected birds. Infected birds shed the virus in large quantities in their respiratory secretions and 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 through contact with contaminated environments.

Human-to-human transmission was suspected in several clusters and documented as probable in Thailand in 2004, Indonesia in 2006, and Pakistan and China in 2007. The transmission usually occurred in the context of intimate, unprotected prolonged contact between a severely ill patient and the contacts.

Incubation period

The incubation period is up to 7 days (often 2–5 days), and approximately 3–5 days for human-to-human transmission.

Period of communicability

The disease is communicable for as long as 3 weeks, and possibly longer in immunosuppressed patients.

Prevention and control measures

Case management

The patient should be isolated and treated under strict infection control measures (standard and droplet precautions). If aerosol-generating procedures are performed,

personal protection equipment should include a particulate respirator rather than a medical mask. Currently, the Infectious Disease Hospital in Angoda is the national referral and isolation facility in Sri Lanka, and the Lady Ridgeway Hospital for children for paediatric referrals. Other major hospitals have been identified as sentinel surveillance and isolation sites.

Treatment with antiviral drugs (oseltamivir, ± amantadine or rimantadine) should be administrated in cases of suspected infection, in the absence of an alternative diagnosis. Corticosteroids should not be used routinely, but may be considered for septic shock. Antibiotic chemoprophylaxis is not recommended.

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. In such cases, oseltamivir should be used for 7–10 days after the last exposure, or zanamivir as an alternative. Health monitoring (for temperature and cough) is recommended for close contacts (including health professionals) of cases for up to 7 days after the last exposure. 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

For individuals, prevention involves hand hygiene, hygiene precautions when handling birds (especially those that are sick or dead) or their products for consumption, or when in environments that may be contaminated with faeces of sick birds. Early detection and appropriate isolation are required.

In communities, prevention involves controlling the spread of the infection in the animal population, and reducing human contact with infected birds.

Surveillance

The early-warning system should be strengthened to support early detection, notification and quick response to initial suspected cases and clusters of H5N1 or a novel pandemic influenza virus in humans. Relevant authorities should be notified immediately if there is suspicion of human avian influenza, and in cases of any suspicious die-off or severe, unexplained illnesses in animals, especially in birds.

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

JAPANESE ENCEPHALITIS

Description

Clinical description

Japanese encephalitis (JE) is a mosquito-borne, viral infection that can result in a febrile illness of variable severity, associated with neurological symptoms ranging from headache to encephalitis or meningitis. Only about one in 250–500 infected individuals manifest clinical disease. JE is mostly characterized by the sudden onset of fever, chills, aches and headaches. Some individuals progress rapidly to severe encephalitis with mental disturbances, motor abnormalities and progressive coma. The high case-fatality ratio (CFR) (20–30%) and frequent residual neuropsychiatric damage in survivors (50–70%) make JE a major public health concern.

Most fatalities and residual sequelae occur in children under 10 years of age.

Infectious agent

The infectious agent is *Japanese encephalitis virus* (JE virus), which is a group B arbovirus (*Flaviviridae*).

Case classification

Suspected JE case: acute encephalitis syndrome

Clinically, a case of acute encephalitis syndrome (AES) is defined as a person of any age with the acute onset of fever at any time of year and with at least one of these symptoms:

- a change in mental status (including symptoms such as confusion, disorientation, coma or inability to talk)
- new onset of seizures, excluding simple febrile seizures (a febrile seizure is one that occurs in a child aged 6 months or younger, whose only finding is fever and a single generalized convulsion lasting less than 15 minutes, and who recovers consciousness within 60 minutes of the seizure).

Other early clinical findings can include an increase in irritability, somnolence or abnormal behaviour greater than that seen with usual febrile illness.

Laboratory-confirmed JE

Laboratory-confirmed JE depends on at least one of the following:

- presence of JE virus-specific IgM antibody in a single sample of cerebrospinal fluid (CSF) or serum, as detected by an IgM-capture enzyme-linked immunosorbent assay (ELISA) specifically for JE virus
- detection of JE virus antigens in brain tissue by immunohistochemistry or immunofluorescence assay
- detection of JE virus genome in CSF, serum, plasma, blood or brain tissue by reverse transcriptase polymerase chain reaction (RT-PCR), or by an equally sensitive and specific nucleic acid amplification test
- isolation of JE virus in CSF, serum, plasma, blood or brain tissue
- detection of a fourfold or greater rise in JE virus-specific antibody as measured by haemagglutination inhibition (HI) or plaque reduction neutralization assay in serum collected during the acute and convalescent-phase of illness.

The two specimens for IgG should be collected at least 14 days apart. Also, tests for JE should be performed in parallel with those for dengue and other flaviviruses.

Notes on laboratory-confirmed JE

- CSF is the preferred sample for diagnosis of JE.
- Most JE infections are asymptomatic; therefore, in areas that are highly endemic for JE, it is possible to have AES due to a cause other than JE virus, and yet have JE virus-specific IgM antibody present in serum. To avoid implicating asymptomatic JE as the cause of other AES illnesses, sterile collection and testing of a CSF sample from all individuals with AES is recommended when feasible.
- A serum sample should be obtained on admission. Because it may not yet be positive in a person infected with JE, a second serum sample should be collected at discharge or on the 10th day of illness onset (usually around 7 days after admission), or at the time of death. The second sample should be tested for the presence of JE virus-specific IgM.
- It is not necessary to test all specimens in a normal seasonal outbreak of JE after the outbreak has been confirmed by laboratory testing. If the outbreak is not an expected seasonal outbreak, or if there are unusual epidemiological features (e.g. age distribution of cases not consistent with pattern of JE infection), testing of CSF is especially important, because an encephalitis outbreak could be due to other etiologies.

Probable JE

A probable case of JE is an AES case that occurs in close geographical and temporal relationship to a laboratory-confirmed case of JE, in the context of an outbreak.

AES – other agent

An AES case due to an agent other than JE is one in which diagnostic testing is performed and an etiologic agent other than JE virus is identified.

AES – unknown

An unknown AES case is one in which either no diagnostic testing was performed, or testing was performed but no etiologic agent was identified, or the test results were indeterminate.

Mode of transmission

JE is transmitted by the bite of an infected mosquito. The vector is the *Culex* species of mosquito, belonging to the *C. tritaeniorhynchus* and the *C. vishnui* groups, which breed particularly in flooded rice fields. Larvae are found in many temporary, semipermanent and permanent groundwater habitats that are sunlit and contain vegetation. Habitats include ground pools, streams, swamps and low-salinity tidal marshes. The female mosquitoes bite mainly cattle and pigs. Humans are incidental hosts; they become infected with JE virus coincidentally when living with or travelling close to animals and birds infected with JE.

Incubation

The incubation period for JE is usually 4–14 days.

Period of communicability

Human-to-human transmission of JE is not thought to occur. Mosquitoes are infective for life. Pigs have a high level of viraemia of prolonged duration; in birds, viraemia usually lasts 2–5 days; in horses, viraemia rarely presents in high titre for long periods.

Reservoirs

Pigs, birds, horses, bats, cattle and reptiles are reservoirs for JE virus. Although many animals can be infected with the virus, only those that develop high viraemias are important in the natural cycle. Pigs are the most important natural host for transmission to humans, as they are often kept close to humans, have prolonged and high viraemias, and produce many offspring, which provide a continuous supply of uninfected new hosts. Birds maintain and amplify JE virus in the environment; they may also be responsible for the spread to new geographical areas.

Epidemiology

Disease burden

JE is the most important form of viral encephalitis in Asia. An estimated 3 billion people are at risk. The virus is estimated to cause at least 50 000 cases of clinical disease each year, resulting in about 10 000 deaths and 15 000 cases of long-term, neuropsychiatric sequelae. The most affected group is children under 10 years of age, but the disease has been reported in all age groups. Reported cases from Sri Lanka are given in Table 1.

Table 1. **Reported cases of Japanese encephalitis by year, Sri Lanka (*1*)**

Year	Reported cases	Reported deaths
2008	31	6
2007	39	0
2006	26	1
2005	65	6

Geographical distribution

JE is endemic in Sri Lanka, south India and other parts of south and South-East Asia and the Pacific where rice production and pig rearing are combined. In recent decades, outbreaks of JE have occurred in several previously nonendemic areas. The reasons for JE spread are not completely understood, but probably include changing agricultural practices, such as increased irrigation (which allows mosquito breeding over longer periods) and animal husbandry (which provides a steady supply of host animals). JE is reported annually in four countries of the World Health Organization (WHO) South-East Asia Region: India, Nepal, Sri Lanka and Thailand. Sporadic cases have also been reported in Indonesia and Myanmar. India and Nepal continue to report a high number of cases and deaths, with 9000 cases of AES and 1700 deaths reported in 2005. Although most cases occur in rural areas, JE virus is also found on the outskirts of cities.

Seasonality

In Sri Lanka and other southern areas, JE is endemic, and cases occur sporadically throughout the year with a peak after the start of the rainy season.

Alert threshold

No alert threshold has been defined for JE.

Outbreaks

Sri Lanka experienced a major outbreak of JE in 1985–86, with 410 cases and 75 deaths in the North Central province. Outbreaks occurred in 1986–87 and 1987–88, the latter being the largest; it caused 812 cases and 192 deaths, and spread to three districts adjoining North Central province. The affected areas were rice-cultivating fields with a network of irrigation canals supported by seasonal, moderate-to-heavy rainfall. Groups most affected were children aged 5–9 years and young adults aged 20–24 years.

Risk factors for increased burden

Population movement

Movement of immunologically naive populations into endemic areas where the vector is present will increase risk of disease. Transmission increases through the presence of mosquito breeding sites, water birds and pig farming.

Overcrowding

Proximity of human habitation to animal husbandry may increase transmission.

Poor access to health services

Lack of access to health services increases the risk of transmission.

Food shortages

Food shortages are not relevant to transmission of JE.

Lack of safe water, poor hygiene practices and poor sanitation

C. gelidus larvae are found in temporary and semipermanent groundwater habitats such as pools, puddles and small streams. They are also occasionally found in artificial containers, such as barrels and water tanks.

Prevention and control measures

Case management

Case management involves managing symptoms and monitoring their progression. No specific antiviral is available.

Prevention

Health centres comprise an alert network for early diagnosis and symptomatic treatment of suspected cases. Laboratory diagnostic capacity is essential for case confirmation.

Health education should involve:

- simple information on JE, including its cause and transmission, and the prevention of mosquito bites
- community action in reducing mosquito breeding places; for example, by filling pools, weekly drainage of accumulated water and lowering of water levels in rice fields.

Vector control

Insecticide spraying is not considered a major control strategy. However, for the immediate suppression of infective vectors, ULV (ultra-low volume) or thermal fogging with malathion (an insecticide) may be employed, even though it is not very cost effective. Environmental measures are recommended.

Short-term measures

Larvicide is impractical for widespread breeding habitats.

Long-term measures

Long-term measures include:

- water management (particularly in irrigated rice fields), for periodic drying of fields
- selection of rice varieties with minimum water requirements
- use of larvivorous fish
- manipulations of the environment (e.g. reduction of drainage, filling of pools and weeding).

Pig vaccination is costly, difficult and time consuming. Also, pig control by segregation or slaughtering is difficult, and the economic losses are high.

Immunization

Vaccination of humans is the only realistic tool for control of JE. Vaccination against the disease began in 1988 in the most affected areas of Sri Lanka, and 14

districts have now incorporated JE immunization into the routine Expanded Programme on Immunization. The live JE vaccine (SA 14–14–2) has been recently licensed in Sri Lanka, and will be introduced in place of the inactivated vaccine, because it is cheaper.

Epidemic control

Epidemic control involves early notification and investigation, including collection of appropriate specimens.

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CD-WGE technical focal points: Departments of Immunization, Vaccines and Biologicals (IVB) and Global Alert and Response (GAR)

LEISHMANIASIS – CUTANEOUS

Description

Clinical description

Several species of protozoan *Leishmania* cause the leishmaniasis disease that is characterized by a spectrum of clinical manifestations:

- ulcerative skin lesions developing at the site of the sandfly bite (localized cutaneous leishmaniasis [LCL])
- multiple non-ulcerative nodules (diffuse cutaneous leishmaniasis [DCL])
- destructive mucosal inflammation (mucosal leishmaniasis)
- disseminated visceral infection (visceral leishmaniasis).

The clinical manifestation depends on the host, the vector and the *Leishmania* species.

In foci where humans are the sole reservoir (anthroponotic foci), epidemics are linked to human migration to endemic areas. Camps for refugees or internally displaced persons can act as amplification sites – first in the camp and then, once the people are repatriated, in their place of origin. In 1992, an outbreak of anthroponotic cutaneous leishmaniasis occurred in refugee camps in the North West Frontier province of Pakistan; on repatriation, that outbreak was imported into Afghanistan, and resulted in 1.5 million cases (70 000 cases per year). Leishmaniasis is associated with refugees or internally displaced persons, malnutrition, human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS), poor sanitation and poverty. The disease affects poor and deprived populations, and exacerbates poverty, because the lesions are disfiguring and disempowering, and the treatment is expensive.

Cutaneous leishmaniasis has emerged in Sri Lanka in the last decade. It is usually associated with the appearance of one or more skin lesions, typically on uncovered parts of the body. The face, neck, arms and legs are the most common sites. Usually, a nodule appears at the site of inoculation; this may enlarge to become a chronic ulcer, which heals over a variable period, leaving a chronic scar. In Sri Lanka, anthroponotic *L. donovani* is the predominant causative organism of cutaneous leishmaniasis; it is usually associated with visceral leishmaniasis in other countries. There are only three recent reported cases of visceral leishmaniasis (one fatal) in Sri Lanka; however, there is potential for its emergence.

Case classification

WHO operational definition

The World Health Organization (WHO) operational definition of a case of cutaneous leishmaniasis is one that is confirmed by positive parasitological smear or culture in a person showing clinical skin signs.

Laboratory criteria for diagnosis

The laboratory criterion for diagnosis is identification of the parasite on stained smear or lesion culture.

Mode of transmission

In anthroponotic disease, human-to-human transmission occurs via sandfly bites and (rarely) through blood transfusion.

Incubation period

The incubation period is at least one week but may be up to many months.

Period of communicability

The disease is not directly transmitted from person to person; however, the disease can infect sandflies as long as parasites remain in the lesions of untreated cases, which is usually from a few months to 2 years.

Reservoirs

Humans act as reservoirs of anthroponotic *L. donovani* leishmaniasis.

Epidemiology

Disease burden

Cutaneous leishmaniasis is endemic in more than 80 countries. Its prevalence tends to be grossly underestimated, because most data surveillance is by passive case detection. The disease was initially exotic in Sri Lanka, but has become a public health problem in the last decade, particularly in the districts of Anuradhapura, Hambantota, Matara and Polonnaruwa. More than 1000 cases of cutaneous leishmaniasis have been reported during the last 5 years, plus a few cases of visceral leishmaniasis. The infections are found in all age groups,

although the most frequently affected group in Sri Lanka appears to be those aged 10–40 years.

More information is available from the relevant section of the WHO web site.[3]

Geographical distribution

The disease has been reported mostly in low-altitude areas of Sri Lanka.

Seasonality

No data on seasonality are available in Sri Lanka.

Alert threshold

No alert threshold has been defined for cutaneous leishmaniasis.

Outbreaks

There are no formal data on leishmaniasis outbreaks in Sri Lanka.

Risk factors for increased burden

Population movement

Population movement increases the risk of exposure to the sandfly vector, and can introduce the disease into a previously unaffected area where sandflies are present.

Overcrowding

Overcrowding promotes transmission, since the vector has a short flight range.

Poor access to health services

Poor access to health care increases the likelihood of transmission and the destructive consequences of the disease.

Food shortages

Malnutrition may increase susceptibility to cutaneous leishmaniasis through immunosupression.

3 http://www.who.int/leishmaniasis/en.

Lack of safe water, poor hygiene practices and poor sanitation

Overcrowded families, poor housing and sanitation contribute to the presence of sandflies.

Prevention and control measures

Case management

Case management involves investigating contacts and possible visceral leishmaniasis cases. Early detection and appropriate, rapid therapy prevents new case development. No drugs are registered for treatment of confirmed leishmaniasis in Sri Lanka. Cryotherapy is the only option, other than pentavalent antimonials (emetic), given either intra-lesionally or parenterally.

Prevention

Early detection and containment of cases are paramount to reduce transmission, and health education is necessary to reduce stigma associated with this potentially disfiguring disease. Prevention has focused mainly on controlling the vector and on environmental management.

Prevention involves:

- systematic detection of cases and rapid treatment
- vector control with insecticides, fine mesh screens and pyrethroid-treated bednets
- control of vector breeding habitats through management of the environment.

Immunization

No vaccine is available currently for cutaneous leishmaniasis, although work is in progress.

Epidemic control

Control of epidemics is mainly through control of the vector, plus diagnosis and treatment of cases.

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

LEPROSY

Description

Clinical description

Leprosy is a slowly progressive, bacterial infection that mainly affects the skin and peripheral nerves. It affects all ages and both sexes. It is easily treated with multidrug therapy (MDT). If untreated, progressive and permanent infection can damage the skin, nerves, limbs and eyes. Leprosy is an important cause of permanent disability in the world. Underserved and marginalized communities are most at risk. Historically, affected people have been subject to discrimination, stigmatisation and exclusion.

Infectious agent

The infectious agent is the bacterium *Mycobacterium leprae*.

Case classification

Operational case definition

The operational case definition of leprosy is a person who has not completed a full course of treatment and has at least one of the following features:

- definite loss of sensation in a pale (hypopigmented) or reddish skin patch
- thickened or enlarged peripheral nerve, with loss of sensation or weakness of muscles supplied by that nerve
- presence of acid-fast bacilli in a slit skin smear.

The operational case definition also includes:

- retrieved defaulters (i.e. those who have defaulted on treatment) with signs of active disease
- relapsed individuals who have previously completed a full course of treatment (not including cured individuals with late reactions or residual disabilities).

Microbiological classification

The microbiological classification of leprosy is as one of the following:

- paucibacillary – all smear-negative cases
- multibacillary – all smear-positive cases.

Clinical classification

The clinical classification of leprosy is as one of the following:

- paucibacillary leprosy: 1–5 patches on the skin
- multibacillary leprosy: more than 5 patches on the skin.

Laboratory criteria for confirmation

The laboratory test for confirmation of leprosy is detection of acid-fast bacilli in skin smears made by the scrape-incision method. In the paucibacillary form of leprosy, bacilli may be too few to be detected. In practice, clinical diagnosis is sufficient.

Mode of transmission

Leprosy is not highly infectious. The exact mode of transmission is unknown. The disease is thought to be transmitted by air through droplets from the nose and mouth, during close and frequent contacts with untreated cases.

Incubation

M. leprae has a long incubation time, from 9 months to 40 years, with an average of 3–4 years.

Period of communicability

Untreated leprosy carries a risk of transmission; this risk is higher for contacts of multibacillary cases. Infectivity is usually lost within a day of treatment with MDT.

Reservoirs

Humans are thought to be the only significant reservoir. Naturally occurring leprosy has been observed in armadillos, a mangabey monkey and a chimpanzee.

Epidemiology

Disease burden

Leprosy is the subject of a global elimination programme, which is aiming for a prevalence of fewer than one case per 10 000 people. This aim was achieved in 2000. At the beginning of 2008, the registered prevalence of leprosy globally was 21 280. The global annual detection of new cases has declined from a peak of more than 763 000 in 2001 to 254 525 in 2007.

Sri Lanka achieved a leprosy elimination goal nationally in 1996. In July 2006, the prevalence of leprosy in Sri Lanka was 0.65 per 10 000 population, although new cases continue to be detected (Table 1).

Table 1. **Annual reported new cases of leprosy, Sri Lanka, 2002–07 (*1*)**

Year	Number of reported new cases
2007	2 024
2006	1 993
2005	1 924
2004	1 995
2003	1 925
2002	2 214

Geographical distribution

New leprosy cases continue to be reported in all districts of Sri Lanka, albeit at low prevalence. Subnationally, elimination has been achieved in 20 of 25 districts. Approximately 40% of new cases occur in the Western district (Colombo, Gampaha and Kalutara) (Figure 1) (*2*).

Seasonality

Leprosy does not follow a seasonal pattern.

Risk factors for increased burden

Population movement

Transmission of leprosy may be increased by untreated multibacillary patients moving into areas with previously unexposed susceptible individuals.

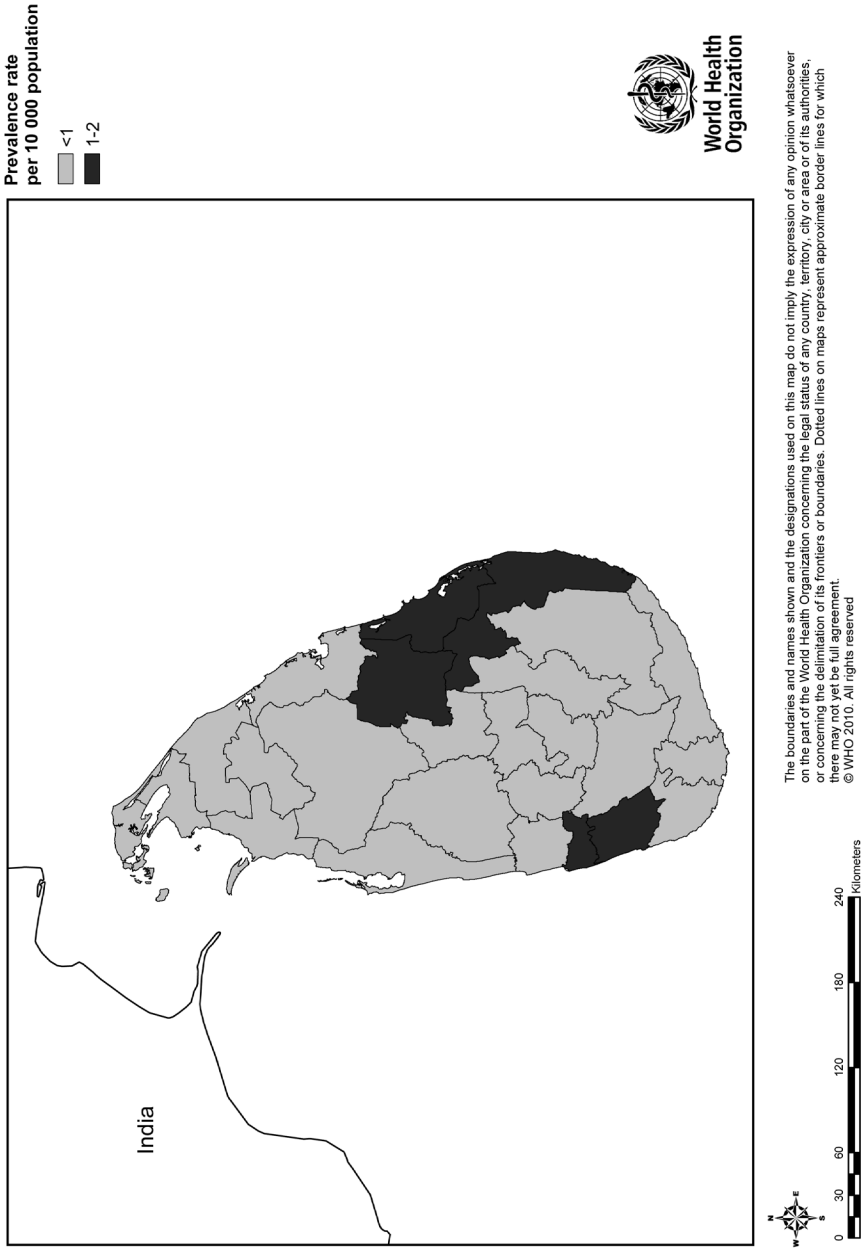
Overcrowding

Untreated individuals may transmit leprosy to susceptible individuals in overcrowded situations where there is frequent close physical contact, but the risk is low.

Poor access to health services

Poor access to services impedes timely diagnosis and treatment, and interruptions to treatment decrease recovery and increase transmission.

Figure 1. **Prevalence of leprosy in Sri Lanka**



Food shortages

Food shortages are not relevant to risk of infection with leprosy.

Lack of safe water, poor hygiene practices and poor sanitation

Lack of safe water, and poor hygiene practice and sanitation are not relevant to risk of infection with leprosy.

Prevention and control measures

Case management

Treatment by MDT is based on case classification (Table 2). MDT is provided free by the WHO. Relapses (recurrence of the disease after treatment is completed) are virtually unknown. No drug resistance to MDT has been reported. Patients should be educated on the possibility of skin and nerve reactions from MDT, and advised to return to the clinic for further management if this occurs.

Table 2. **Recommended drug treatment regimes for leprosy**

	Multibacillary	Paucibacillary
Duration	12 months	6 months
Adult combination doses (adult blister packs)	**Rifampicin:** 600 mg once a month **Dapsone:** 100 mg once a day **Clofazimine:** 50 mg once a day, and 300 mg once a month	**Rifampicin:** 600 mg once a month **Dapsone:** 100 mg once a day
Children (child blister packs)	Scaled-down combination doses	Scaled-down combination doses

In Sri Lanka, leprosy diagnosis and treatment is available at all dispensaries and health facilities, and patients are free to complete treatment at a health centre close to them. Leprosy drugs are part of the national treatment registry, and supplies are managed by the Medical Supplies Division. Leprosy activities are included in district health plans, and data are managed by the district epidemiologist.

Prevention

Prompt diagnosis and treatment prevents disability and impedes transmission. Thus, early detection and prompt treatment of cases with MDT is the cornerstone of global leprosy elimination. Health education should promote the availability of effective MDT, and emphasize that that leprosy is easily cured. Early

treatment will lead to minimal disabilities and will prevent transmission. Reducing contact with known leprosy patients is of dubious value and can lead to stigmatization.

Immunization

The bacille Calmette–Guérin (BCG) vaccination can induce protection against some forms of leprosy. However, this is part of tuberculosis control methods and must not be undertaken specifically against leprosy.

Immunization of contacts is not recommended.

Epidemic control

Epidemic control is not applicable for leprosy.

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

LEPTOSPIROSIS

Description

Clinical description

Leptospirosis is an under-reported zoonosis; most human infections are probably asymptomatic. The spectrum of illness is wide, ranging from undifferentiated febrile illness to severe multisystem disease with a high mortality rate. Generally, there are two phases in the illness: the leptospiraemic (febrile) stage, followed by a convalescent or immune phase. Clinical diagnosis is often difficult. Recovery of untreated cases can take several months. Overall, the case-fatality ratio (CFR) is low, but it increases with advancing age, and may reach 20% in patients with jaundice or kidney failure.

Infectious agent

The infectious agents are pathogenic leptospires bacteria of the order Spirochaetales, belonging to the species *Leptospira interrogans*. More than 200 serovars have been identified.

Case classification

Suspected case

A suspected case involves a person presenting with acute febrile illness; headache, myalgia and prostration; associated with any of the following symptoms:

- conjunctival suffusion; meningeal irritation; anuria, oliguria, or proteinuria; jaundice; haemorrhage (from intestines; lung bleeding often notorious); cardiac arrhythmia or failure; or skin rash
- a history of exposure to infected animals or an environment contaminated with infected animal urine.

Other common symptoms include nausea, vomiting, abdominal pain, diarrhoea and arthralgia.

Laboratory-confirmed case

Laboratory confirmation of leptospirosis involves isolation, positive serology or positive polymerase chain reaction (PCR) interpreted within the context of geography, patient history and laboratory results.

Isolation (and typing)

Isolation and typing is through culture of pathogenic leptospires from clinical material, including at least one of the following:

- isolation of leptospires from blood within the first 7 days of disease (due to cost considerations, the Sri Lankan Ministry of Health advises that samples be sent for culture only when the patient presents in the early stage of the disease and clinical suspicion is high)
- isolation of leptospires from cerebrospinal fluid (CSF) between days 4 and 10
- isolation of leptospires from urine after day 10.

Positive serology

Positive serology involves seroconversion or at least a fourfold increase in titre, preferably by the microscopic agglutination test (MAT) using a range of *Leptospira* strains for antigen that represent local strains.

In Sri Lanka, confirmatory tests with MAT can be performed at the Medical Research Institute, Colombo. The Sri Lankan Ministry of Health (MOH) recommends that a first blood sample should be taken and sent 5 days after onset of illness, and a second sample 4–5 days later if clinical suspicion is high but the MAT result for the first sample was equivocal or negative (i.e. to demonstrate a rising titre).

A PCR test for leptospirosis that has higher specificity and sensitivity than MAT is currently available at the Molecular Biology Unit, University of Kelaniya. This test may be subsidised if an appropriate request is made with the endorsement of the university's Epidemiology Unit.

Mode of transmission

Carrier animals excrete the organism in their urine, thereby contaminating the environment. Human infection occurs either by direct contact with infected urine or tissues, or by indirect exposure to the organisms in damp soil or water.

Incubation

The incubation period for leptospirosis is usually 10 days, with a range of 2–30 days.

Period of communicability

Direct transmission of leptospirosis from person to person is rare. Leptospires may be excreted in human and animal urine for a month after the acute illness (and in rare cases, for months or even years).

Reservoirs

Reservoirs include both wild and domestic animals; for example, rats, pigs, cattle and dogs.

Epidemiology

Disease burden

Leptospirosis is endemic to Sri Lanka (Table 1). National incidence estimates are 5.4/100 000 population, the highest reported in the region.

Table 1. **Number of reported clinical and confirmed leptospirosis cases by year, 2001–07, Sri Lanka (*1*)**

Year	Total number of clinical cases reported in Sri Lanka	Number of laboratory-confirmed cases
2007	1 197	700
2006	1 568	439
2005	3 900	450
2004	3 291	458
2003	4 713	685
2002	994	390
2001	1 402	599

Geographical distribution

Leptospirosis occurs worldwide, but is most common in tropical and subtropical areas, with the highest incidence in island or low-lying countries subject to frequent flooding. In the 2008 outbreak in Sri Lanka, the worst-affected districts were:

- Central province – Kandy and Matale
- North-western province – Kurunegala
- Sabaragamuwa province – Kegalle
- Southern province – Galle and Matara
- Western province – Colombo, Gampaha and Kalutara.

The highest incidence was in the Matale district, with 150.5 per 100 000 population (*2*).

Seasonality

Leptospirosis is reported throughout the year, but there is a peak at the time of the monsoons, a smaller one in March to May, and a larger one in October to December. High humidity and heavy rainfall intensify outbreaks, because of widespread contamination of floodwater by rodent urine when rodents are displaced from their burrows and drains.

Alert threshold

All suspected cases of leptospirosis must be investigated. Case-based reporting of suspected or confirmed cases from peripheral levels should be instituted immediately. In Sri Lanka, sentinel surveillance is undertaken in only 16 selected hospitals in the high-risk areas. The International Leptospirosis Society collects worldwide data.

Outbreaks

Leprospirosis outbreaks are reported about every 4–5 years. The largest recorded outbreak in Sri Lanka started in late 2008, with incidence of suspected leptospirosis at 35.7 per 100 000 population in 2008. Notification peaked in late September 2008, but the rates are currently declining. The total number notified up to 17 December 2008 was 7099 patients and 204 deaths with a CFR of 2.9% (higher than the 2007 CFR of 1.5%) (*2*).

Risk factors for increased burden

Population movement

Movements of animals, whether or not they are combined with population movement, increase the risk of transmission.

Overcrowding

Overcrowding may increase the risk by increasing the proximity of humans to animals and their excrement.

Poor access to health services

Poor access to health services causes delays in diagnosis, leading to higher CFR.

Food shortages

Food shortages are relevant to the burden of disease, because poor physical condition of patients is associated with increased severity of disease and death.

Lack of safe water, poor hygiene practices and poor sanitation

Conditions leading to an increase of contaminated water or soil (e.g. rain, floods and disasters) increase the risk of leptospirosis. Poor hygiene practices may lead to wounds being contaminated by water and soil that is stained with animal urine. During periods of drought, both humans and animal reservoirs may access a common water source.

Others

Other risk factors are:

- floods and disasters – these increase the risk of leptospirosis and may lead to epidemics
- exposure to mud and water (e.g. from rivers, streams, canals and lakes) contaminated by urine of domestic and wild animals – this can lead to outbreaks of disease
- occupations such as working in rice and sugarcane fields, farms, sanitation, abattoirs, fisheries and in the military.

In Sri Lanka, most patients with leptospirosis have been exposed to paddy fields (60.9% in 2007) or marshlands (23.7% in 2007).

Prevention and control measures

Case management

In Sri Lanka, febrile patients presenting with the following signs or symptoms and history should be admitted for inpatient management:

- a history of exposure to *Leptospira* in contaminated environments (e.g. work in local agricultural and gem-mining areas, cleaning of canals and drains, and swimming or playing in contaminated floodwater), and symptoms such as conjunctival suffusion and muscular pain or tenderness
- without a history of exposure, but strongly suspected of having leptospirosis, presenting with conjunctival suffusion and muscular pain or tenderness.

Management includes treatment with intravenous (IV) penicillin (six times hourly), initiated without delay, and maintenance of adequate hydration with IV fluids, if indicated. If the fever continues for more than 3–4 days, it is important to watch for signs and symptoms suggestive of possible complications, such as renal failure, heart failure and widespread haemorrhage.

Patients should be transferred to a higher level health-care institution if:

- despite adequate hydration, there is concern about urine output
- symptoms suggestive of cardiac involvement (e.g. hypotension and tachycardia) are present.

Prevention

Prevention involves:

- educating public health and veterinary workers about disaster implications following flooding
- educating the public on modes of transmission
- cautioning the public to avoid swimming or wading in potentially contaminated waters, or to use protection when work requires such exposure
- protecting workers in hazardous occupations by providing boots, gloves and aprons
- recognizing potentially contaminated waters and soil, and draining such waters where possible or placing warning signs
- controlling rodents in human habitations
- cleaning premises where humans live or work that attract rodents
- segregating infected domestic animals
- where possible, immunizing farm and domestic animals to prevent illness, even though this does not necessarily prevent infection and renal shedding of leptospires.

Chemoprophylaxis is not advocated in Sri Lanka as a routine and major preventive strategy. It is recommended only for well-recognized, high-risk groups. The problem should be discussed with farmers' organizations and agrarian services, to identify high-risk localities at the divisional level.

Immunization

Selective vaccination should be provided for those exposed through occupational risk. Vaccines must contain the dominant, local strains, since immunity to a specific serovar may not protect against infection with a different serovar.

Epidemic control

When an outbreak is suspected or identified, control measures include:

- identifying the source of infection
- eliminating the source of infection and prohibiting the use of contaminated water sources
- investigating industrial and occupational sources, including those with direct animal contact
- ensuring that veterinary experts and departments are included in the control management team.

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CD-WGE technical focal point: Department of Global Alert and Response (GAR)

LYMPHATIC FILARIASIS

Description

Clinical description

Lymphatic filariasis (LF), more commonly known as elephantiasis, is a painful and profoundly disfiguring disease. Although the disease is usually acquired in childhood, its visible manifestations occur in adults, leading to temporary and permanent disability. It has a major social and economic impact on endemic countries.

The disease is caused by thread-like worms, known as filariae, which lodge in the lymphatic system; that is, the network of nodes and vessels that maintain the delicate fluid balance between the tissues and blood, and are an essential component of the body's immune system. In its most obvious manifestations, LF causes enlargement of the entire leg or arm, the genitals, vulva and breasts. In endemic communities, 10–50% of men and up to 10% of women can be affected. In addition, filiarial fevers can occur several times a year, usually due to secondary bacterial infections and acute adenolymphangitis (ADL), with painful swollen lymph nodes in the groin or axilla. Most infected people are asymptomatic, but most also have clinical lymphatic damage, and as many as 40% have renal involvement, with proteinuria and haematuria.

Infectious agent

LF is caused by three types of parasitic worms: *Wuchereria bancrofti*, *Brugia malayi* and *B. timori*. In Sri Lanka, *W. bancrofti* is the only current cause of LF. *B. malayi* infection may have been eliminated.

Case classification

Probable

A probable case of LF is one in which there is hydrocoele or lymphoedema, for which other causes have been excluded, in a resident of an LF-endemic area.

Confirmed

A confirmed case of LF is one in which the positive laboratory criteria are met, even if the person does not meet the clinical case definition.

Laboratory criteria

The laboratory criterion for LF is positive parasite identification by either direct blood examination, ultrasound or positive antigen-detection test.

Identification by microscopic examination of microfilariae in a blood smear (preferably collected at night, when microfilariae circulate most in blood) confirms the diagnosis of active infection. However, because lymphoedema often develops many years after infection, laboratory tests are typically negative with these patients. A rapid immunology-based card has now been developed.

Mode of transmission

Repeated bites of infected, blood-feeding, female mosquitoes (mainly *Culex* species) 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 (this is known as the pre-patent period).

Period of communicability

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

Reservoirs

Humans are the only reservoirs for *W. bancrofti*.

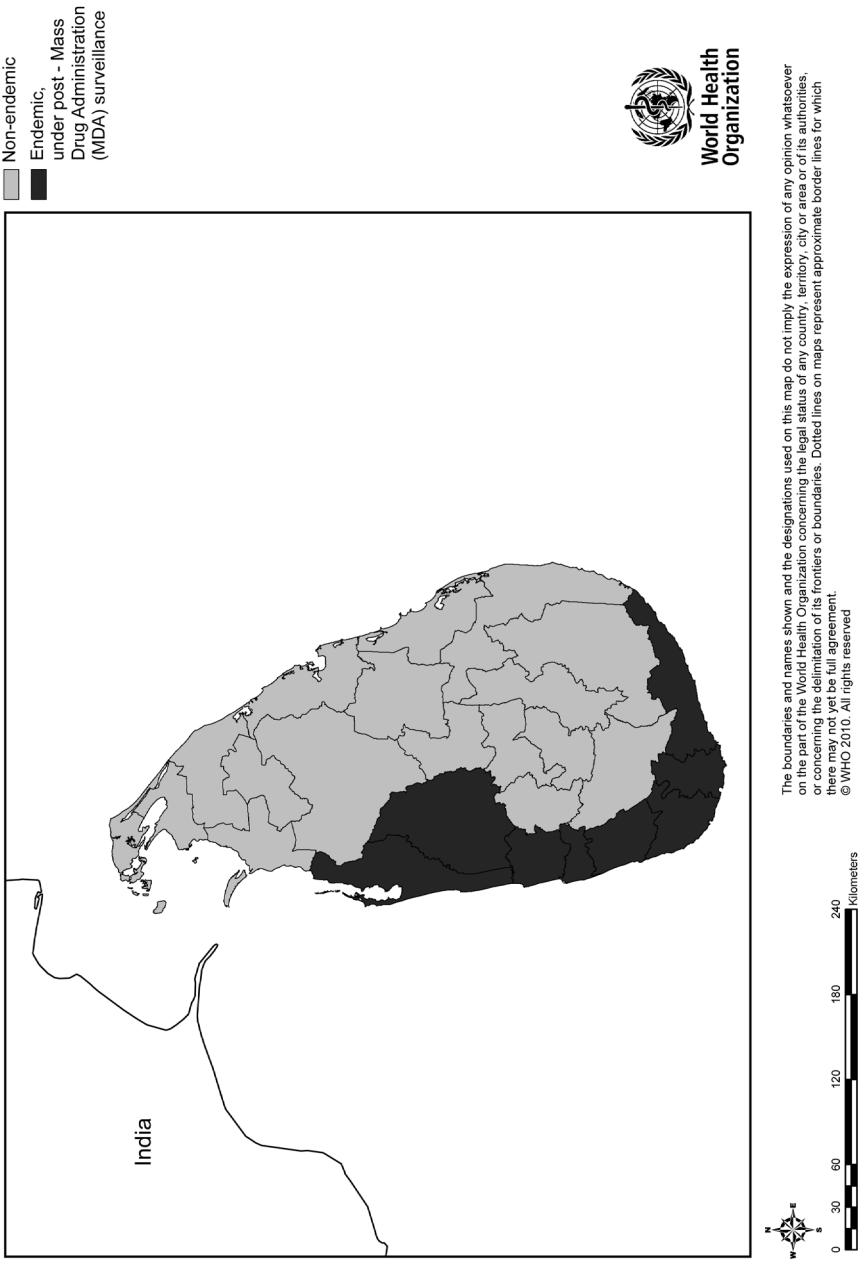
Epidemiology

Disease burden

The burden of LF, as measured in disability-adjusted life years (DALYs), is the highest of all tropical diseases apart from malaria. Countries are categorized as endemic when the population prevalence of microfilaraemia or antigenaemia is greater than 1%.

Sri Lanka is one of the nine known endemic countries in the World Health Organization (WHO) South-East Asia Region. The anti-filariasis campaign in Sri Lanka started in 1947 with selective chemotherapy, health education and recurrent vector control measures. An increased effort is being made to eliminate LF from Sri Lanka by 2010. LF prevalence (as assessed by the "microfilaria rate") decreased in Sri Lanka from 0.24% in 1981 to 0.05% in 2007.

Figure 1. **Lymphatic filariasis (LF) endemicity by district, Sri Lanka 2005 (*1*)**



Geographical distribution

LF is endemic in 8 of Sri Lanka's 25 districts (Figure 1). About 10 million people were considered at risk of acquiring infection before the start of the mass drug administration (MDA) campaign.

Seasonality

Transmission risk is likely to increase during the rainy season, when mosquitoes are abundant. The frequency of ADL also increases during the rainy season because of increased bacterial or fungal infections in humans; for example, between the toes.

Outbreaks

LF is not outbreak prone.

Risk factors for increased burden

Population movement

Movement of chronically infected individuals and migration of infected mosquitoes into disease-free areas both increase the geographical spread of LF.

Overcrowding

Living in crowded conditions increases the risk of transmission of LF.

Poor access to health services

Lack of early diagnosis and treatment increases the risk of transmission and morbidity. The shame and stigma associated with the disease often lead to delay in seeking health-care services.

Food shortages

Food shortages have no direct effect on burden, but malnutrition leading to hypoalbuminaemia may compound limb oedema and delay wound healing.

Lack of safe water, poor hygiene practices and poor sanitation

Providing safe water can help to prevent secondary complications such as adenolymphangitis attacks and bacterial infections. Good hygiene is essential for affected body parts. Poor sanitation may create breeding sites for mosquito vectors (especially *Culex* spp.).

Others

LF prevalence is higher when there is low productivity and poverty.

Prevention and control measures

Case management

Case management involves treatment of the patient to kill the adult-stage filarial parasites (e.g. with diethylcarbamazine citrate [DEC], at, for example, 6 mg/kg single dose) or albendazole. This can improve elephantiasis and hydrocoele, particularly in the early stages of the disease.

Rigorous hygiene and other measures to minimise infection and promote lymph flow can reduce the frequency of ADL and improve elephantiasis; such measures include:

- washing the affected parts twice daily with soap and clean water, and keeping the affected parts 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).

Treatment for large hydrocoele is mostly surgical.

Prevention

The WHO Global Program to Eliminate Lymphatic Filariasis, launched in 1997, has two main goals – to interrupt transmission of LF infection and to prevent disability caused by the disease.

Transmission of infection can be interrupted by:

- minimizing contact between humans and vectors (through control of mosquitoes and larvae, and avoiding mosquito bites)
- reducing the amount of infection the vector can acquire (by treating the human host).

The vector control programme of Sri Lanka's anti-filariasis campaign consists of treating permanent breeding sites of the vector mosquito with a larvicide (fenthion) at a concentration just sufficient to kill the larvae. Larviciding is confined to selected urban areas.

In communities where LF is endemic, treating the affected community for long enough can eliminate microfilariae from the blood of infected individuals, and thus interrupt transmission. The use of single doses of two drugs (e.g. albendazole with DEC or ivermectin) is 99% effective in removing microfilariae from the blood for a full year after treatment. Sri Lanka began annual, single-dose MDA (using albendazole with DEC) in one district in 2001, expanding nationally in 2002. Of the 10 million people exposed in 2004, a total of 9.85 million received MDA. Global efforts are aimed at eliminating LF as a public health problem by 2020.

In 2003, Sri Lanka also started a district-wide, community, home-based prevention of disability. A total of 1848 patients with lymphoedema and hydrocoele have been identified. Training in skin care has been initiated, and hydrocoelectomies are planned.

Immunization

Immunization is not available for LF.

Epidemic control

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

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

MALARIA

Description

Clinical description

The first symptoms of malaria are nonspecific, and are similar to the symptoms of a minor, systemic viral illness. The symptoms comprise headache, lassitude, fatigue, abdominal discomfort, and muscle and joint aches, followed by fever, chills, perspiration, anorexia, vomiting and worsening malaise. At this stage, with no evidence of vital organ dysfunction, the case-fatality ratio (CFR) is low (about 0.1% for *Plasmodium falciparum* infections, and other human malarias are rarely fatal), provided that prompt and effective treatment is given. However, if ineffective drugs are administered, or treatment is delayed in *P. falciparum* malaria, the parasite burden continues to increase and severe malaria may ensue. A patient may progress from minor symptoms to severe disease within a few hours. Severe disease usually manifests with one or more of the following: coma (cerebral malaria), metabolic acidosis, severe anaemia, hypoglycaemia and, in adults, acute renal failure or acute pulmonary oedema. By this stage, mortality in people receiving treatment rises to 15–20%. If untreated, severe malaria is almost always fatal.

Infectious agent

The main malaria parasite species in Sri Lanka is *Plasmodium vivax*. Only 6.5% of cases in 2008 were transmitted by *P. falciparum*. In other countries, *P. malariae* and *P. ovale* also cause malaria.

Case classification

Suspected but uncomplicated malaria

Malaria is classified as suspected but uncomplicated if the patient has fever or a 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).

Suspected severe malaria

Malaria is classified as suspected severe malaria if a patient with symptoms of uncomplicated malaria also has 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 cases (both uncomplicated and severe)

Malaria is classified as confirmed in a patient with uncomplicated or severe malaria plus symptoms, when there is laboratory confirmation by blood film examination for malaria parasites or other diagnostic tests for *Plasmodium* antigen (e.g. rapid diagnostic testing [RDT], enzyme-linked immunosorbent assay [ELISA] or polymerase chain reaction [PCR]).

Mode of transmission

Malaria is transmitted by mosquitoes. The vector is the female *Anopheles* mosquito, which bites mainly between dusk and dawn. *A. culicifacies* is the principal vector in Sri Lanka.

Incubation period

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

Period of communicability

Transmission is related to the presence of infective female *Anopheles* mosquitoes and of infective gametocytes in the blood of patients. (A gametocyte is 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.) Patients who are untreated or insufficiently treated may be a source of mosquito infection 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 for malaria.

Epidemiology

Disease burden

Malaria remains a major public health problem globally. In 2006, there were an estimated 247 million cases and 881 000 deaths (*1*). Of the deaths, 91% occurred in Africa, and 85% were of children under 5 years of age.

In Sri Lanka, the burden of malaria is decreasing (Table 1); in 2008, 670 cases of laboratory-confirmed malaria were reported. None of the cases were severe and no deaths were reported. A total of 46 (6.8%) cases were due to *P. falciparum*. Sri Lanka is reorienting its programme towards elimination of malaria. Although malaria control in conflict-affected areas remains a challenge, no outbreaks were reported from these areas in 2008.

Table 1. **Number of reported malaria cases and deaths by year, 2001–08, Sri Lanka (*1*)**

Year	Annual reported cases	*P. falciparum* cases	Annual deaths
2008	670	46	0
2007	198	7	1
2006	591	18	0
2005	1 640	94	0
2004	3 720	500	1
2003	10 510	1 273	4
2002	41 411	4 661	30
2001	66 522	10 241	53

Geographical distribution

Malaria transmission occurs throughout most of Sri Lanka, with the exception of the districts of Colombo, Galle, Gampaha, Kalutara, Matara and Nuwara Eliya. The districts that are most affected are located in the northern and eastern parts of the country. Local outbreaks are occasionally reported in connection with population movements. The latest was reported in 2007 in the district of Trincomalee, when 82 laboratory-confirmed cases were recorded for the year.

Seasonality

Factors that influence malaria transmission include: altitude (in Sri Lanka malaria can occur up to 1200 m), rainfall, humidity, temperature and vegetation. Transmission occurs year round throughout the country, with seasonal peaks during and just after the monsoon rains.

Outbreaks

Outbreaks in Sri Lanka since 1967 are shown in Table 2.

Table 2. **Outbreaks of malaria in Sri Lanka since 1967–97**

Year	Outbreak details
1967–68	> 500 000 cases
1975	400 777 cases; SPR 26.7%[a]
1986	412 521 cases; SPR 28.1%
1987	687 599 cases; SPR 34.6%
199	400 263 cases; SPR 28.6%
1997	218 550 cases; SPR 16.4%

SPR, slide positivity rate.

[a]The proportion of *P. falciparum* infections also increased from < 0.5% in 1968 to 8.3% in 1974, and 15.9% in 1975.

More recently, local outbreaks have occasionally been reported in connection with population movements (see Geographical distribution section above for recent local outbreaks).

Alert threshold

Any confirmed case of malaria should prompt an epidemiological investigation and response that is consistent with the elimination strategy. In the Northern province, where the elimination strategy has yet to commence, more than three confirmed cases in a week should trigger an outbreak investigation.

Risk factors for increased burden

Population movement

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

Overcrowding

Increased population density may lead to increased exposure to mosquitoes in poorly protected 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 malaria gametocyte carriers, and therefore increases transmission.

Food shortages

Malnutrition increases vulnerability to severe malaria once a human is infected. This malnutrition can mask obvious malaria symptoms, and delay clinical diagnosis and treatment, thereby increasing mortality.

Lack of safe water, poor hygiene practices and poor sanitation

Temporary standing water may increase malaria vector breeding, especially in arid environments. Different vectors have different preferences for breeding sites.

Others

Human immunodeficiency virus (HIV) co-infection worsens both the manifestations and the severity of malaria.

Prevention and control measures

Case management

Prompt access to effective treatment is the cornerstone of malaria control. Globally, resistance has eventually arisen to every class of antimalarial drug. Parasite tolerance to artesunate has already been reported in south-western provinces of Cambodia. Sri Lanka bans oral artemisinin monotherapies.

Malaria diagnosis should be confirmed by microscopy or RDT before initiation of treatment. Where diagnosis is delayed, treatment for *P. vivax* should be started on the basis of clinical diagnosis until confirmation. Treatment guidelines for malaria in Sri Lanka are given in Table 3.

Table 3. **Recommended treatment guidelines for malaria in Sri Lanka (*2*)**

Condition	Antimalarial therapy recommended in Sri Lanka
P. vivax (confirmed) and first-line treatment of suspected malaria	Chloroquine (25 mg base/kg divided over 3 days) and primaquine (0. 25 mg/kg daily for 14 days)
P. falciparum (confirmed)	Artemether–lumefantrine (20 mg of artemether + 120 mg of lumefantrine coformulated tabs): 5–14 kg, 1 tab; 15–24 kg, 2 tabs; 25–34 kg, 3 tabs; > 34 kg, 4 tabs per dose (3-day course of 6 doses total, taken at 0, 8, 24, 36, 48 and 60 hours) plus 0.75 mg/kg of primaquine as a single dose
Treatment failure of *P. falciparum* and malaria in first trimester of pregnancy	Oral quinine (10 mg base/kg) 3 times daily for 7 days
Treatment of severe malaria due to *P. falciparum*	Quinine 7 days (parenteral, loading dose 20 mg salt/kg; maintenance at 30 mg salt/kg/day divided into 3 doses, given 8-hourly); change to artemether–lumefantrine tablets as soon as patient is well enough to take medication orally (doses are as given for confirmed *P. falciparum*)

Laboratory capacity

Laboratory tests (microscopy) for malaria diagnosis are widely available throughout Sri Lanka. RDT is available in areas without microscopy, particularly in the population living in areas of conflict and the bordering villages. There are several types of RDT; those commonly used in Sri Lanka detect plasmodium lactate dehydrogenase (pLDH) to detect falciparum and nonfalciparum species. In other countries, where *P. falciparum* infection predominates, tests that detect histidine-rich protein-II (HRP-II) are often used to identify *P. falciparum* infection. RDTs may lose their sensitivity when stored in hot and humid conditions, and should usually be stored below 30 °C. Heat-stability data should be requested from the manufacturer before purchase of RDTs.

Prevention

In Sri Lanka, malaria control activities are integrated with the general health services. In view of the elimination context, all malaria cases have to be laboratory confirmed and epidemiologically investigated, and control interventions must be implemented in close collaboration with and under supervision of the Sri Lankan Ministry of Health (MOH) Anti-Malaria Campaign.

Key preventive strategies are listed below.

Health education

Active health education at community level is important for improving the likelihood that fever cases will rapidly seek treatment, for effective use of insecticide-treated nets, and for improving acceptability of indoor residual spraying with insecticide.

Vector control

Long-lasting insecticidal net

Long-lasting insecticidal nets (LLINs) provide increased personal protection for those who sleep under such nets. When coverage is above 80%, LLINs can significantly reduce the adult mosquito population and community impact, thereby reducing transmission and subsequent morbidity and mortality. Insecticide-treated nets and LLINs can be distributed as part of integrated mass-vaccination campaigns or as stand-alone distributions. Health education on proper use and care is vital for the success of LLIN programs. Unlike insecticide-treated nets, LLINs do not need to be retreated with insecticides every 6 months; therefore, they are the intervention of choice, especially for displaced and conflict-affected populations. Nationwide,

100 000 households were estimated to be protected by insecticide-treated nets or LLINs in 2006, thereby providing protection for more than 10% of the at-risk population.

Indoor residual spraying

Indoor residual spraying has been the main vector control measure used in Sri Lanka. In view of the resistance to the insecticides DDT and malathion, Sri Lanka is now using a rotation of fenitrothion and pyrethroids to reduce the load of malaria transmission. In 2007, 92 609 households were estimated to have been treated with indoor residual spraying (*2*).

Periodic indoor spraying of shelters with residual insecticide can reduce transmission, provided the spray is applied according to World Health Organization (WHO) recommendations and the following conditions are met:

- a high percentage of structures in an operational area have adequate sprayable surfaces, and can be expected to be well covered by spray
- most of the vector population is endophilic (i.e. rests indoors)
- the vector is susceptible to the insecticide in use.

The main purpose of indoor residual spraying is to reduce transmission by reducing the survival of malaria vectors entering houses or sleeping units. Such spraying is not applicable during acute phases of emergencies, but may be useful in well-organised temporary settlements and camps.

Environmental control

Environmental control may be difficult during the acute phase of an emergency, except on a local scale, and its impact is often limited. The number of vector breeding sites can be reduced by:

- draining water around water tap stands and rainwater drains
- using larvicides in vector breeding sites if these sites are limited in number (after seeking expert advice)
- draining ponds (although this may not be acceptable if ponds are used for washing or for animals).

In view of the elimination approach in Sri Lanka, malaria control interventions, including programmes to distribute LLIN and carry out indoor residual spraying, should be guided by geographical identification of transmission foci. Also, such

programmes should be implemented in close collaboration with the MOH Anti-Malaria Campaign.

Health advice for international travellers

There is a relatively low risk of becoming infected with malaria in Sri Lanka (*3*).

The malaria parasites are mainly *P. vivax* (93.1%) and *P. falciparum* (6.9%), which are found throughout the year, except in the districts of Colombo, Galle, Gampaha, Kalutara, Matara and Nuwara Eliya. Resistance to chloroquine, pyrimethamine and sulfadoxine has been reported for *P. falciparum*.

Recommended preventive measures for travellers to areas of risk are to avoid mosquito bites and to take chloroquine plus proguanil chemoprophylaxis.

Immunization

No immunization for malaria is currently available. Several vaccine candidates are under investigation.

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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 (i.e. red eyes), coryza (i.e. runny nose), cough, small whitish spots on the buccal mucosa (inside the mouth) called Koplik spots, and a characteristic, red blotchy rash appearing between days 3 and 7 of the illness. The rash usually begins on the face, and then spreads to rest of the body; it lasts for 4–7 days. Most children have uncomplicated measles. Infants and adults tend to suffer more complications including acute otitis media, diarrhoea, bronchopneumonia, laryngotracheobronchitis and encephalitis. In developing countries, deaths generally occur in 1–5% of cases, but may be as high as 25% in populations with elevated levels of malnutrition and poor access to health care.

Infectious agent

The infectious agent is the measles virus (genus *Morbillivirus*, family *Paramyxoviridae*).

Case classification

Clinically confirmed case

A clinically confirmed case of measles is one in which a clinical health worker suspects measles infection, or in which all of the following are seen:

- fever
- maculopapular (i.e. non-vesicular) rash
- cough, coryza or conjunctivitis.

Laboratory confirmed

A laboratory-confirmed case of measles is one that meets the clinical case definition and a positive result for measles-specific IgM antibodies.

Confirmed through epidemiologic linkage

A case of measles confirmed through epidemiologic linkage is one that meets the clinical case definition and is from a district or zone where an outbreak has been laboratory confirmed during the previous 30 days.

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

Mode of transmission

Measles is spread by airborne droplets (through coughing and sneezing), direct contact with nasal and throat secretions of infected individuals, or through objects, such as toys, that have been in close contact with an infected person. The virus remains alive in the air or on infected surfaces for about 2 hours.

Incubation period

The incubation period is generally 10–12 days (range 7–18 days).

Period of communicability

Measles is most infectious from 4 days before until 4 days after the onset of the rash. The disease symptoms are minimal after the second day of rash.

Reservoir

Humans are the reservoir for measles.

Epidemiology

Disease burden

Measles is one of the most contagious diseases in humans, and it remains a leading cause of death among children worldwide. In 2006, an estimated 242 000 deaths were attributed to measles globally. The current burden of measles in Sri Lanka is relatively low, although there has been a concerning rise in recent years, as shown in Table 1.

Table 1. **Reported measles cases and vaccination coverage, Sri Lanka, 2003–08 (*1*)**

Year	Reported cases	Vaccination coverage (%)
2008	33	98
2007	44	98
2006	0	99
2005	3	99
2004	35	96
2003	65	99

Geographical distribution

There is no specific pattern of geographical distribution of measles in Sri Lanka.

Seasonality

No specific seasonality pattern has been observed for measles in Sri Lanka.

Alert threshold

One case of measles 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.

Outbreaks

A large outbreak of measles occurred in Sri Lanka between September 1999 and June 2000, during which more than 15 000 suspected cases of measles were reported.

Risk factors for increased burden

Population movement

Transmission of measles cases may increase if nonimmune populations move into areas where the pathogen is circulating, or infected individuals move into areas where the population is not immunized. Large population migration, particularly with war and conflict, may compromise vaccination programmes.

Overcrowding

Crowded conditions and poor indoor ventilation facilitate rapid transmission of measles.

Poor access to health services

Poor immunization programmes may increase the numbers of people susceptible to measles infection. Delayed case recognition delays both patient isolation and immunization of contacts. The case-fatality ratio (CFR) increases when effective management of severe cases is absent.

Food shortages

Disease is more severe among children with malnutrition and vitamin A deficiency. Because measles can actually trigger acute protein-energy malnutrition and worsen vitamin A deficiency, malnourished children are at an even higher risk of complications and death following measles.

Lack of safe water, poor hygiene practices and poor sanitation

Children living with lack of safe water, poor hygiene practices and poor sanitation usually have poor nutrition and repeated attacks of communicable diseases, making them further malnourished and thus at greater risk of contracting measles.

Prevention and control measures

Case management

Case management involves reporting cases to the local health authority and isolating the case where possible (e.g. by keeping measles-infected children away from school). Because there is no specific antiviral treatment, supplemental vitamin A is essential in resource-poor settings, because it minimises the complications of the disease.

Uncomplicated cases

Management of uncomplicated cases involves:

- giving vitamin A immediately upon diagnosis and ensuring that the child receives a second dose the next day (this can be given to the parent to administer at home; doses are given in Table 2, below)
- advising the parent to treat the child at home (by controlling the fever and providing nutritious food).

Cases with non-severe eye, mouth or ear complications

Children with non-severe eye, mouth or ear complications can be treated at home. Management involves:

- giving vitamin A immediately upon diagnosis and ensuring that the child receives a second dose the next day (this can be given to the parent to administer at home; doses are given in Table 2, below)
- cleaning the eyes if pus drains from the eyes, and treating with 1% tetracycline eye ointment
- treating the mouth with gentian violet if there are mouth ulcers
- cleaning ear discharge if pus drains from either ear, and treating with antibiotics for 5 days (amoxicillin as first-line and cotrimoxazole as second-line antibiotics), as per national acute respiratory infection policy and Integrated Management of Childhood Illness (IMCI) guidelines

- treating malnutrition and diarrhoea, if present, with sufficient fluids and a high-quality diet.

Cases of severe, complicated measles

Severe, complicated cases of measles are those with any general danger signs (e.g. inability to drink or breastfeed, repeated vomiting, convulsions, lethargy or unconsciousness) clouding of cornea, deep or extensive mouth ulcers, or pneumonia. Management of such cases involves:

- referring the patient immediately to hospital
- treating the pneumonia with an appropriate antibiotic
- cleaning the eyes and applying 1% tetracycline eye ointment if there is clouding of the cornea or pus draining from the eye
- giving vitamin A at days 1, 2, and 15, using the dosages given in Table 2.

Table 2. **Dosages of vitamin A by age (*2*)**

Age	Dosage (international units)
Infants < 6 months	50 000
Infants 6–11 months	100 000
Children > 11 months	200 000

Vitamin A should not be given in the doses shown in Table 2 to females who may be pregnant, because of the presumed risk of birth defects associated with high doses (> 25 000 international units in a single dose) of vitamin A (*3*).

Prevention

Immunization is the key to prevention of measles. It is usually implemented through mass campaigns or routine immunization programmes. Improving and maintaining high levels of routine immunization coverage is important to prevent outbreaks.

Immunization

The measles vaccine was included in the Expanded Programme on Immunization in 1984. It is currently offered to children at 9 months of age as the live attenuated measles vaccine, and at the age of 3 years as the live attenuated measles–rubella vaccine. A single dose of this vaccine should induce active immunity in 94–96%

of susceptible individuals, probably for life; a second dose may increase immunity levels to 99%. About 5–15% of nonimmune individuals may develop fevers and malaise within 5–12 days of vaccination; this lasts for 1–2 days. The live measles vaccine should also be administered to contacts within 72 hours of exposure. Immunoglobulin has been used post-exposure, in immunocompromised patients, where live vaccines are relatively contraindicated; however, it is not readily available in Sri Lanka.

Epidemic control

In an acute emergency, the population at risk should be immunized as soon as possible if vaccination coverage is below 80–90% or is 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 – 12 years or 6 months – 5 years). Vitamin A supplementation should be administered in the doses shown in Table 2 to those under 5 years of age, and should be considered in those older if clinical vitamin A deficiency is suspected. Children who are vaccinated against measles and are below 9 months of age must receive a second measles vaccination, which should be given as soon as possible after reaching 9 months of age, with an interval of at least 1 month between doses.

Outbreak response should include:

- informing health authorities if one or more suspected cases are identified
- confirming the suspected outbreak following World Health Organization (WHO) guidelines (Annex 2)
- investigating any suspected case (i.e. checking whether a case fulfils the case definition, and recording the onset date, age and vaccination status)
- confirming the diagnosis (i.e. collecting blood specimens from 3–5 initial reported cases)
- assessing the outbreak extent and population at risk
- implementing outbreak response measures as follows
    - giving priority to proper case management and immunization of groups at highest risk (e.g. children aged 6 months – 15 years) as soon as possible, even in areas not yet affected but where the outbreak is likely to spread
    - reducing the age range if vaccines are limited
    - through social mobilization, ensuring that parents bring previously unvaccinated children for immunization.

The presence of several cases of measles in an emergency setting is an indication that a measles immunization campaign should be implemented. Even among individuals who have already been exposed and are incubating the natural virus, measles vaccine may provide protection or modify illness severity, if it is given within 3 days of exposure.

Isolation is not indicated, and children should not be withdrawn from feeding programs.

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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 presents in three main clinical forms: the meningeal syndrome, the septic form (meningococcemia or meningococcal sepsis) and pneumonia. Meningeal syndrome is the most common presentation, but meningococcal sepsis is the most severe form, leading to disseminated intravascular coagulation and multiorgan failure. The onset of symptoms is sudden, and death can follow within hours. As many as 10–15% of survivors have persistent neurological defects, including hearing loss, speech disorders, intellectual impairment and paralysis. Other forms of the disease (e.g. pneumonia) are less common.

Infectious agent

The bacterium *Neisseria meningitidis* causes meningococcal disease. The serogroups are A, B, C, W135, X and Y. Serogroups A, B and C account for 90% of cases.

Case classification

Suspected

A suspected case of meningococcal disease is one in which there is illness with sudden fever onset (greater than 38.5 °C rectal or greater than 38.0 °C axillary) 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

A probable case of meningococcal disease is a suspected case with one or more of the following:

- turbid cerebrospinal fluid (CSF) (with or without positive Gram stain)
- leukocytosis (> 100 cells/mm^3)

- leukocytosis (10–100 cells/mm^3) AND either an elevated protein (> 100 mg/dl) or decreased glucose (> 40 mg/dl)
- continuing epidemic.

Confirmed

A confirmed case of meningococcal disease is a suspected or probable case with laboratory confirmation through one or more of the following:

- positive CSF antigen detection
- positive bacterial culture of CSF or blood
- polymerase chain reaction (PCR) (if available) to detect meningococcal deoxyribonucleic acid (DNA)
- Gram staining of CSF to identify *N. meningitidis*.

Mode of transmission

Meningococcal disease is transmitted by direct contact with respiratory droplets (5–10% of the population may be asymptomatic nasopharyngeal carriers).

Incubation period

The incubation period for meningococcal disease is 2–10 days (most commonly 3–4 days).

Period of communicability

The disease is communicable while live meningococci are present in discharges from the nose and mouth. This is usually from the beginning of symptoms until 24–48 hours after the start of therapy. Asymptomatic carriers are the most important source of infection.

Reservoirs

Humans are the reservoir for meningococcal disease.

Epidemiology

Disease burden

Meningococcal meningitis occurs sporadically in small clusters throughout the world, with seasonal variations. It accounts for a variable proportion of endemic bacterial meningitis, with five serogroups (*N. meningitidis* A, B, C, X and W135)

recognized as producing epidemics. Serogroup A is usually the cause of meningococcal disease in Asia. There is evidence of serogroup W135 being associated with outbreaks of considerable size, mainly in Africa. In 2000 and 2001, several hundred pilgrims attending the Hajj in Saudi Arabia were infected with *N. meningitidis* W135. No data on meningococcal disease are available for Sri Lanka; however, 783 cases of meningitis (undifferentiated) were reported in 2007, and 1300 in 2008 (*1*).

Geographical distribution

No information is available on the geographical distribution of meningococcal disease in Sri Lanka.

Seasonality

No information is available on the seasonality of meningococcal disease in Sri Lanka.

Alert threshold

The alert threshold for meningococcal disease is an increase in the number of cases compared with previous years.

Outbreaks

No outbreaks of meningococcal disease have been reported in Sri Lanka.

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, in cattle or fishing-related activities, in military camps and in schools facilitates spread of the disease.

Poor access to health services

Poor access to health services prevents rapid diagnosis and treatment; the case-fatality ratio in the absence of treatment can be as high as 50%.

Food shortages

Food shortages are not relevant to the risk of meningococcal disease.

Lack of safe water, poor hygiene practices and poor sanitation

Poor respiratory etiquette facilitates transmission.

Others

Smoking, mucosal lesions and concomitant respiratory infections may contribute to meningococcal disease.

Prevention and control measures

Case management

Meningeal syndrome and meningococcal sepsis are potentially fatal and should always be considered a medical emergency.

Nonepidemic conditions

In the absence of an epidemic, case management should involve:

- admission to a hospital or health centre for diagnosis (lumbar puncture and CSF examination)
- commencement of antimicrobial therapy as soon as possible after lumbar puncture (without waiting for laboratory results), combined with supportive treatment.

Infectivity of patients is moderate and disappears quickly following antimicrobial treatment; therefore, isolation of the patient is not necessary.

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. The recommended treatment in this case is the administration of ceftriaxone once daily for 5–7 days. Alternatively, ampicillin or amoxicillin may be used.

See Table 1 below.

Presumptive treatment with a single dose of either ceftriaxone or oily chloramphenicol is appropriate once there is confirmation of a diagnosis of meningococcal disease or an outbreak of meningococcal origin.

Table 1. **Meningococcal epidemic situations: presumptive treatment of bacterial meningitis with oily chloramphenicol (OC) in children over 2 years of age and in adults, in the absence of laboratory confirmation (*2*)**

Age group	Principal causes	Treatment	Monitoring
2–5 years	*Streptococcus pneumoniae* *Haemophilus influenzae* *Neisseria meningitidis*	OC 100 mg/kg single dose IM	Clinical monitoring at 24 and 48 hours. If no improvement after 24 hours,[a] give second dose of OC 100 mg/kg, or refer.
> 5–14 years	*N. meningitidis* *(S. pneumoniae)*	OC 100 mg/kg single dose IM	Clinical monitoring at 24 and 48 hours. If no improvement after 24 hours,[a] give second dose of OC 100 mg/kg, or refer.
> 14 years	*N. meningitidis* *(S. pneumoniae)*	OC 100 mg/kg (max. 3 g) single dose IM	Clinical monitoring at 24 and 48 hours. If no improvement after 24 hours,[a] give second dose of OC 100 mg/kg (max. 3 g), or refer.

OC, oily chloramphenicol; IM, intramuscular.

[a] Repeated convulsions, fever > 38.5 °C after 24 hours, appearance or aggravation of a reduced level of consciousness since admission, appearance or aggravation of neurological signs since admission.

Prevention

Sri Lanka's national guidelines recommend chemoprophylaxis for close contacts of cases (recommended doses are given in Table 2). They also recommend that patients with meningococcal infection who are treated with antibiotics that do not eliminate the carrier state (penicillins or chloramphenicol) should receive chemoprophylaxis with an effective antibiotic (ciprofloxacin, rifampicin or ceftriaxone) upon hospital discharge.

Table 2. **Recommended chemoprophylaxis dosages for meningococcal infection (*3*)**

Drug	Age group	Dose
Ciprofloxacin	Adults	20 mg/kg, single dose 500 mg single dose
Rifampicin	< 1 month	5 mg/kg, twice a day for 2 days
	≥ 1 month	10 mg/kg, twice a day for 2 days
	Adults	600 mg single dose
Ceftriaxone	< 15 years	125 mg, single dose, intramuscularly
	≥ 15 years	250 mg, single dose, intramuscularly

Intervention

Inform authorities; investigate; confirm; treat cases; strengthen surveillance; prepare for epidemic.

Immunization

A number of polysaccharide vaccines are available: tetravalent (serogroups A, C, W135 and Y), trivalent (A, C and W135) and bivalent (A and C). Polysaccharide vaccines are effective for outbreak control (although ensuring that vaccination takes place early in the outbreak is difficult), and for prevention in high-risk groups. Saudi Arabia requires pilgrims to the Hajj and Ramadan Omra to obtain a tetravalent vaccine (against serogroups A, C, W135 and Y) at least 10 days before their arrival in the country (*4*).

Epidemic control

The activities required in control of an epidemic of meningococcal disease are listed below.

- Cases should be diagnosed and treated immediately.
- Overcrowding should be reduced.
- Mass chemoprophylaxis of contacts of meningitis patients is not warranted during an epidemic. However, in outbreaks involving small populations (e.g. in a school or prison) chemoprophylaxis to all members may be considered.
- An epidemic protocol should be disseminated. The protocol should be simple and clear, to permit the health system to respond rapidly to increasing numbers of cases and be able to treat every suspected case of meningitis. Adequate quantities of treatments should be available at health centres.
- A mass vaccination campaign should be instituted, concentrated in the area where the epidemic is maximal; this can halt an epidemic if carried out appropriately. Laboratory diagnosis and confirmation of epidemic serogroups will guide the type of vaccine needed. The vaccine should be meningococcal polysaccharide bivalent A/C (if serogroup A or C is confirmed as the epidemic serogroup), or meningococcal polysaccharide tetravalent A/C/Y/W135 (if serogroup W135 is confirmed).
    - In camp settings, once two cases have been confirmed (serogroup identified), mass vaccination is recommended with the appropriate vaccine for the responsible serogroup (as outlined in the previous point). At-risk populations should be given priority.

- In the 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 groups at highest risk.

- A public information campaign should be implemented.

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CD-WGE technical focal point: Department of Global Alert and Response (GAR)

PERTUSSIS (WHOOPING COUGH)

Description

Clinical description

The initial stage – the catarrhal stage – of pertussis (also known as "whooping cough") is similar to the common cold. It is characterized by a runny nose, sneezing, low-grade fever and a mild occasional cough. An irritating cough then develops within 1–2 weeks. Typically, the patient has a sudden burst of numerous rapid (near-violent) coughs followed by a long inspiratory effort, which is usually accompanied by a characteristic high-pitch whoop. This may last 4–8 weeks, with minimal associated fever. In younger infants, periods of apnoea (cessation in breathing) may follow the coughing spasms, and the patient may look blue. Complications such as pneumonia, encephalopathy and even death are more frequent and severe in younger infants. In developing countries, case-fatality ratios (CFRs) are estimated at 3.7% for those under 1 year of age, and 1% for children aged 1–4 years. In malnourished, unvaccinated populations with high prevalence of coinfections, CFR can reach 15%. Older individuals and those partially protected by the vaccine may become infected, but usually have milder disease.

Infectious agent

The infectious agent for pertussis is *Bordetella pertussis*.

Case classification

Clinically confirmed

A clinically confirmed case is one of the following:

- A case diagnosed as pertussis by a physician.
- A person with cough lasting at least 2 weeks and presenting 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.

Laboratory confirmed

A laboratory-confirmed case of pertussis is one that meets the clinical case definition and has been confirmed through one or more of the following:

- isolation of *B. pertussis*
- detection of genomic sequences by polymerase chain reaction (PCR)
- positive paired serology.

Mode of transmission

Pertussis is transmitted primarily by direct contact with discharges from the respiratory mucous membranes of infected people via the airborne route.

Incubation period

The incubation period is usually 7–10 days, and is rarely more than 14 days.

Period of communicability

Pertussis is highly communicable in the early (catarrhal) stage, and has a high secondary attack rate, approaching 90%, in household contacts. Communication gradually decreases after onset of the paroxysmal cough. Patients may be contagious for up to 3 weeks after paroxysmal cough onset when untreated, or up to 5 days after onset of treatment.

Reservoirs

Humans are the only hosts of pertussis.

Epidemiology

Disease burden

Worldwide, an estimated 152 535 cases of pertussis were reported in 2007. Sri Lanka has seen a progressive reduction in the disease, and has maintained high vaccination coverage of the third dose of diphtheria–tetanus–pertussis vaccine (DTP3) for more than a decade (Table 1).

Table 1. **Reported pertussis cases and immunization coverage, Sri Lanka, 2003–08 (*1*, *2*)**

Year	Reported cases of pertussis	DTP3 immunization coverage (%)
2008	9	98
2007	0	98
2006	0	99
2005	0	99
2004	45	97
2003	82	99

DTP3, third dose of diphtheria–tetanus–pertussis vaccine.

Geographical distribution

Pertussis is found worldwide; no specific information is available on its geographical distribution in Sri Lanka.

Seasonality

Pertussis generally has no distinct seasonal pattern.

Alert threshold

One case is sufficient for an alert, and it must be investigated, especially if the case occurs in a high-risk area (e.g. one with low vaccination coverage).

Outbreaks

The last significant outbreak of pertussis in Sri Lanka occurred in 1997, when 405 cases were reported, mainly from Colombo, Gampaha and Kurunegala districts. Most cases were in infants aged 3–12 months.

Risk factors for increased burden

Population movement

Mass population movement promotes the spread of *B. pertussis*.

Overcrowding

Crowded conditions facilitate transmission. The disease is usually introduced into a household by an older sibling or parent.

Poor access to health services

All nonimmunized individuals are susceptible; therefore, low vaccination coverage is a major risk factor for increased transmission and outbreaks.

Food shortages

Malnutrition increases the severity of pertussis.

Lack of safe water, poor hygiene practices and poor sanitation

Lack of safe water, poor hygiene practices and poor sanitation are not relevant to the risk of transmission of pertussis.

Prevention and control measures

Case management

Erythromycin should be administered for 7 days to all cases and close contacts of people with pertussis, regardless of age and vaccination status, and to all those living in households where there is an infant under 1 year of age. Clarithromycin and azithromycin are also effective. If initiated early, drug administration modifies the course of the illness and eradicates the organism from secretions, thereby reducing communicability. However, it does not reduce the 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. Antibiotics should be administered (as indicated above) during management of an outbreak.

Immunization

Whole-cell vaccine

Active primary immunization with the whole-cell pertussis vaccine (wP) is recommended, in association with the administration of vaccines containing diphtheria and tetanus toxoid at 6, 10 and 14 weeks of age, 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. 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.

Acellular vaccines

Although acellular pertussis vaccines (aP) are less commonly associated with adverse reactions, price considerations affect their use; therefore, wP vaccines are the vaccines of choice for some countries.

Except for cases where prior pertussis vaccination resulted in anaphylactic reaction, there are no strict contraindications to these vaccines. All infants, including those who are human immunodeficiency virus (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 vaccines have limited impact on the circulation of *B. pertussis*, even in countries with high vaccination coverage. Nonimmunized children and older individuals with waning immunity may serve as reservoirs for the infection, and may transmit *B. pertussis* to nonimmunized infants. Susceptible adolescents and adults contribute to pertussis outbreaks, although high vaccination coverage may prolong the time between epidemics.

Epidemic control

The highly contagious nature of pertussis leads to large numbers of secondary cases among nonimmune contacts. Prophylactic antibiotic treatment (erythromycin) in the early incubation period may prevent disease, but difficulties of early diagnosis, costs and concerns about drug resistance all limit prophylactic treatment to selected individual cases.

Priority must be given to:

- protecting children under 1 year of age and pregnant females in the final 3 weeks of pregnancy, because of the risk of transmission to the newborn
- stopping infection among household members, particularly if these include children under 1 year of age and pregnant women in the final 3 weeks of pregnancy.

The strategy relies on chemoprophylaxis of contacts within at least 14 days of the first contact with the index (initiating) 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 (WHO), 2010 (http://www.who.int/immunization_monitoring/en/globalsummary/timeseries/tsincidenceper.htm, accessed 9 August 2010).
2. *WHO vaccine-preventable diseases: monitoring system — country profiles*. Geneva, WHO, 2010 (http://www.who.int/vaccines/globalsummary/immunization/countryprofileselect.cfm, accessed 9 August 2010).

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

POLIOMYELITIS

Description

Clinical description

The most recognizable feature of poliomyelitis is onset of acute flaccid paralysis (AFP); however, this symptom occurs in less than 1% of *Poliovirus* infections. More than 90% of those infected have asymptomatic infections or a mild, non-specific febrile illness that lasts a few days. An abrupt onset of meningitic and neuromuscular symptoms, such as neck stiffness and pain in the limbs follow in a few cases, associated with fatigue, headache, vomiting and constipation (or less commonly, diarrhoea).

AFP, when it occurs, is of gradual onset (2–4 days); typically asymmetric and usually involves lower rather than upper limbs. Bulbar (brainstem) paralysis leading to respiratory depressions accounts for the 2–10% mortality rate associated with paralytic poliomyelitis.

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 (post-polio syndrome) may develop in 25–40% of patients.

Infectious agent

The infectious agent is *Poliovirus* (of the *Enterovirus* genus group). Types 1, 2 and 3 can all cause paralysis. Type 1 is the type most often isolated from paralytic cases and associated with epidemics. Most vaccine-associated cases are classified as type 2 or 3.

Case classification

Suspected

A suspected case of poliomyelitis is one in which there is one or both of the following:

- AFP in a child under 15 years of age, including Guillain–Barré syndrome (for practical reasons, this syndrome is considered as poliomyelitis until proven otherwise)
- any paralytic illness in a person of any age when polio is suspected.

Confirmed

A confirmed case of poliomyelitis is one in which there is AFP with laboratory-confirmed wild *Poliovirus* in a stool sample.

Polio compatible

A polio compatible case of poliomyelitis is one in which there is AFP that is clinically compatible with poliomyelitis, but insufficient virological investigation to confirm the diagnosis.

Mode of transmission

Poliovirus is transmitted mainly via the faecal–oral route.

Incubation period

The incubation period for poliomyelitis is commonly 7–14 days for paralytic cases; but the reported range is from 3 up to 35 days.

Period of communicability

Poliovirus is highly communicable. The virus is demonstrable in throat secretions as early as 36 hours after exposure to infection, and in faeces after 72 hours. The virus persists in throat secretions for 1 week and in faeces for 3–6 weeks. Cases are most infectious during the days before and after the onset of symptoms.

Reservoir

Humans are the reservoir for *Poliovirus*. Those with asymptomatic infections, especially children, are the main reservoir.

Epidemiology

Disease burden

As of 2009, poliomyelitis has been eliminated from most of the world – only four countries remain polio-endemic (Afghanistan, India, Nigeria and Pakistan). This represents the lowest number of countries ever with circulating wild *Poliovirus*. At the same time, the areas of transmission are more concentrated than ever – 98% of all global cases are found in India, Nigeria and Pakistan. However, all countries remain at risk of importations of the disease, regardless of their geographical proximity to polio-endemic countries. It is therefore important that certification-standard surveillance is widely maintained, to avoid late detection of any wild

Poliovirus importation. In 2007, 1385 cases of poliomyelitis globally were reported to the World Health Organization (WHO).

In Sri Lanka, the last case of poliomyelitis was reported in 1993, and high vaccination coverage has been maintained for over a decade (Table 1). However, its close proximity to polio-endemic India places Sri Lanka at constant risk of new cases.

Table 1. **Poliomyelitis cases and immunization coverage reported in Sri Lanka, 2003–07 (*1*, *2*)**

Year	Reported cases of poliomyelitis	Oral *Poliovirus* trivalent vaccine (OPV) third dose immunization coverage (%)
2007	0	98
2006	0	98
2005	0	99
2004	0	97
2003	0	98

Geographical distribution

Although substantial progress has been made towards eradicating poliomyelitis, the goal of eradication is at constant risk from outbreaks of the disease following importation of wild *Poliovirus* into polio-free areas and ongoing transmission of wild *Poliovirus* in endemic areas. The success of eradication depends largely on progress in Afghanistan, India, Nigeria and Pakistan. Within the South-East Asia region, India remains the only polio-endemic country, but Bangladesh, Indonesia and Nepal have reported recent importations of wild *Poliovirus*. The last cases of polio reported in Sri Lanka occurred in Kataragama in the Moneragala district in 1993.

Seasonality

Transmission of poliomyelitis is higher during the rainy season.

Alert threshold

Any case of AFP must be notified and investigated.

Outbreaks

There have been no recent outbreaks of poliomyelitis in Sri Lanka.

Risk factors for increased burden

Population movement

Population movement promotes transmission of poliomyelitis from infected to nonimmune populations.

Overcrowding

Overcrowding increases the transmission of poliomyelitis.

Poor access to health services

Limited access to routine immunization services will lead to low vaccination coverage (defined as < 80%) for the routine Expanded Programme on Immunization, thereby increasing the proportion of susceptible individuals and the risk of outbreaks. Undiagnosed poliomyelitis increases the pool of undetected circulating *Poliovirus*.

Food shortages

Food shortages are not relevant to the transmission of poliomyelitis.

Lack of safe water, poor hygiene practices and poor sanitation

Because the spread of poliomyelitis is by the faecal–oral route, lack of water, poor hygiene and poor sanitation increase the risk of its transmission.

Prevention and control measures

Case management

No specific antiviral treatment is available for poliomyelitis. Therefore, treatment involves support and management of symptoms, including:

- close monitoring of respiration
- respiratory support in case of respiratory failure or pooling of pharyngeal secretions
- moist hot-packs for muscle pain and spasms
- passive physical therapy to stimulate muscles and prevent contractures
- anti-spasmodic drugs
- frequent patient turning to prevent bedsores.

Patients should be isolated to avoid contact with children, in particular. Safe disposal of discharge and faeces, disinfection of soiled articles and immediate reporting of further cases are essential.

Prevention

Immunization is vital for the prevention of poliomyelitis.

Immunization

The routine immunization schedule in Sri Lanka includes oral *Poliovirus* vaccine (OPV) given at 2, 4, 6 and 18 months, and at 5 years. This oral vaccine is based on live, attenuated strains of all three virus types. It is easily administered, induces good immunogenicity and is considerably cheaper than the injectable inactivated *Poliovirus* vaccine. Vaccine-related paralytic poliomyelitis is rare (1 in every 2.5 million doses).

Sri Lanka has conducted national immunization days (NIDs) from 1995 to 2000 and sub-NIDs (SNIDs) from 2001 to 2003. To counteract the risk of importing disease from polio-endemic neighbouring countries, a supplementary dose of OPV is given to children under 15 years of age who return to Sri Lanka from India.

Epidemic control

Epidemic control involves investigation and intervention, as outlined below.

Investigation

Investigation should include:

- clinical and epidemiological investigation
- rapid virological investigation (two stool samples within 14 days of symptoms onset must be sent to a WHO-accredited laboratory)
- outbreak confirmation, based on isolation of wild *Poliovirus* from a stool sample obtained from an AFP case.

Intervention

Intervention should include the following:

- House-to-house "mop-up" campaigns with OPV covering a wide geographical area (at least in the province involved and relevant neighbours) conducted within 4 weeks of confirmation of the wild *Poliovirus* case. Such campaigns should target a minimum of 500 000 to 1 million children.

- If NIDs and SNIDs were already planned, intervention should focus on the quality of supplementary immunization activities in the area of outbreak and adjacent districts.
- Surveillance should be enhanced through intensive monitoring of all reporting units, to ensure active surveillance and reporting, extensive retrospective record reviews, and active case-finding in surrounding areas.

These measures have been incorporated into the Sri Lankan polio eradication programme.

References

1. *Polio reported cases*. Geneva, World Health Organization (WHO), 2010 (http://www.who.int/immunization_monitoring/en/globalsummary/timeseries/tsincidencepol.htm, accessed 9 August 2010).
2. *Reported third dose coverage estimates for trivalent oral polio vaccine (OPV),* Geneva, WHO, 2010 (http://apps.who.int/immunization_monitoring/en/globalsummary/timeseries/tscoveragepol3.htm, accessed 9 August 2010).

CD-WGE technical focal point: Polio Eradication Initiative (POL)

RABIES

Description

Clinical description

Rabies is an acute zoonotic viral disease, transmitted through contact (mainly bites and scratches) with infected animals, both domestic and wild. It causes paresis or paralysis, delirium and convulsions. Rabies is invariably fatal in the absence of adequate post-exposure prophylaxis.

Infectious agent

The infectious agent is *Rabies virus*, a rhabdovirus of the genus Lyssavirus.

Case classification

Human rabies

Suspected

A suspected rabies case is one in which there is an acute neurological syndrome (encephalitis) dominated by forms of hyperactivity (furious rabies) or paralytic syndrome (dumb rabies) that progresses to coma, respiratory failure and death within 1–2 weeks of the first symptoms if no intensive care is instituted. A history of bites or scratches from suspected animal is usually evident.

Probable

A probable case of rabies is a suspected case plus a history of contact with a suspected rabid animal.

Confirmed

A confirmed case of rabies is 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 postmortem)
- detection by FA on skin biopsy or corneal smear (collected antemortem)
- FA positive after inoculation of brain tissue, saliva or cerebrospinal fluid (CSF) in cell culture, or after intracerebral inoculation in mice or suckling mice
- detectable rabies-neutralizing antibody titre in the serum or CSF of an unvaccinated person

- detection of viral nucleic acids by polymerase chain reaction (PCR) on tissue collected postmortem or in a clinical specimen (brain tissue or skin, cornea, urine or saliva).

Human exposure to rabies

Human exposure to rabies is classified as one of the following:

- *possibly exposed* – the person has had close contact (usually a bite or a scratch) with a rabies-susceptible animal in (or originating from) a rabies-infected area
- *exposed* – the person has had close contact (usually a bite or a scratch) with a laboratory-confirmed, rabid animal.

Mode of transmission

Rabies is usually transmitted by the bite of an infected mammalian species (e.g. dog, cat, fox or bat). The bites or scratches introduce virus-laden saliva into the human body.

No human-to-human transmission of rabies has been documented.

Incubation period

The incubation period for rabies usually ranges from 2–10 days, but may be as long as 7 years.

Period of communicability

In dogs and cats, the disease is usually communicable 3 or 4 days before the onset of clinical signs (but occasionally up to 7 days before onset of symptoms), and throughout the course of the disease.

Reservoir

Dogs are the main reservoir and vector of rabies in Sri Lanka, accounting for 81% of all cases in 2007 (stray dogs 38% and pets 35%, Ministry of Healthcare and Nutrition, Sri Lanka).[4]

Epidemiology

Disease burden

Rabies is an important but declining public health issue in Sri Lanka, with reported rabies deaths declining from 377 in 1975 to 51 in 2008 (Table 1). Men are more

4 http://www.health.gov.lk

affected than women (male:female ratio 2:1 in 2007). The age group 20–59 years is most affected, with an increasing proportion of cases among those 60 years or older. An estimated 200 000 people per year receive post-exposure prophylaxis (PEP) after being exposed to suspected rabies-infected animals.

Table 1. **Number of reported rabies death for selected years, Sri Lanka, 1975–2007 (*1*)**

Year	Number of reported rabies deaths
2008 (as of 26 Dec 2008)	51
2007	55
2006	68
2005	55
2004	98
2003	76
2002	64
2001	83
2000	109
1995	124
1990	154
1985	113
1980	209
1975	377

Geographical distribution

Most rabies deaths in Africa and Asia occur in rural areas. In Sri Lanka, cases are reported from all districts (*1*).

Seasonality

No seasonality of rabies has been reported.

Alert threshold

The alert threshold for rabies is one case in a human or a susceptible animal species.

Outbreaks

Rabies is a sporadic illness in humans.

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

Overcrowding increases the risk of transmission because in overcrowded settings, an infected animal has the opportunity to bite more people. Dog population density parallels human population density.

Poor access to health services

Poor access to health services increases the risk of death from rabies, because prompt administration of postexposure vaccine (plus immunoglobulin if exposure is heavy) is the only way to prevent death of an infected person.

Food shortages

Food shortages are not relevant to rabies infection.

Lack of safe water, poor hygiene practices and poor sanitation

Lack of safe water and poor hygiene make it difficult to clean a wound from an infected animal, and thus increase the risk of rabies infection.

Prevention and control measures

Case management

There is no specific treatment for rabies once the symptoms have started. Prevention of rabies after animal bites (through post-exposure prophylaxis – PEP) includes:

- first aid – the wound or point of contact should be flushed with soap and water or detergent, and ethanol or iodine (tincture or aqueous solution)
- vaccination with antirabies vaccine.

Table 2, below, summarises the recommended care according to type of contact with a suspect animal. Vaccine should be given as soon as possible for Category II and III exposures, according to WHO-recognized regimens.

Table 2. **Recommended care for a suspected rabies case according to type of contact with a suspect animal (*2*)**

Category	Type of contact with a suspect animal (confirmed rabid domestic or wild animal, or animal unavailable for testing)	Type of exposure	Recommended care
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	■ Flush the wound or point of contact with soap and water or detergent; ethanol or iodine ■ Administer vaccine immediately ■ Stop treatment if the animal remains healthy throughout an observation period of 10 days, or if the animal is found 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	■ Flush the wound or point of contact with soap and water or detergent; ethanol or iodine ■ Administer rabies immunoglobulin and vaccine immediately ■ Stop treatment if the animal remains healthy throughout an observation period of 10 days or if the animal is found to be negative for rabies by a reliable laboratory using appropriate diagnostic techniques.

When caring for patients with rabies, universal barrier-nursing practices should be applied.

Prevention

The most cost-effective strategy for preventing rabies in people is by eliminating rabies in dogs through animal vaccinations. In Sri Lanka, the National Rabies Control Programme was started in 1975. The programme comprises:

- surveillance for rabies
- immunization of domestic, community and stray dogs against rabies
- promotion of responsible dog ownership and birth control for dogs

- destruction of stray dogs suspected of incubating the *Rabies virus* (ceased in 2007)
- PEP, training and health education
- enforcement of rabies control legislation.

The Public Health Veterinary Services (PHVS) controls rabies in Sri Lanka. Surveillance of both human and animal rabies is important in detecting high-risk areas and implementing control activities. Human rabies is a notifiable disease in Sri Lanka.

Immunization

Since their development more than 4 decades ago, concentrated and purified cell-culture and embryonated egg-based rabies vaccines (here jointly referred to as CCVs) have proven to be safe and effective in preventing rabies. These vaccines are intended for pre-exposure prophylaxis (PrEP) as well as PEP, and have been administered to millions of people worldwide.

Rabies differs from many other infections in that the development of clinical disease can be prevented through timely immunization, even after exposure to the infecting agent. In Asia and Africa, the present level of rabies PEP prevents about 272 000 deaths each year.

The recommendations given below are intended as a general guide, for more detailed information, the recently revised guidance documents should be consulted (*2, 3*).

Pre-exposure prophylaxis

PrEP may be performed with any of the modern cell-derived vaccine; it is recommended for anyone at increased risk of exposure to rabies virus. Traditionally, PrEP is recommended for anyone who is at continual, frequent or increased risk of exposure to the rabies virus either as a result of their residence or occupation (e.g. laboratory work with rabies virus and other lyssavirus, veterinarians and animal handlers). Travellers with extensive outdoor exposure and children living in rural areas are at particular risk.

The PrEP schedule requires intramuscular doses of 1 ml or 0.5 ml, depending on the vaccine type, or intradermal administration of 0.1 ml volume per site (one site each day) given on days 0, 7 and 28.

Post-exposure prophylaxis

PEP – which consists of local treatment of the wound, followed by vaccine therapy (with or without rabies immunoglobulin) – should be initiated immediately after

a transdermal bite or scratch by an animal suspected of being rabid, or when possibly infectious material (usually saliva) comes into direct contact with the victim's mucosa or fresh skin wounds (see Table 2). Prompt post-exposure use of CCVs, combined with proper wound management and simultaneous administration of rabies immunoglobulin, is almost invariably effective in preventing rabies, even following high-risk exposure. However, delays in starting PEP, or failure to complete correct prophylaxis, may result in death, particularly following bites in highly innervated regions, such as the head, neck or hands, or following multiple wounds. Treatment should be started as soon as possible after exposure, but in no case should it be denied to exposed persons, whatever time interval has elapsed.

Treatment may be discontinued if the animal involved (e.g. dog or cat) remains healthy throughout an observation period of 10 days; or if the animal is found to be negative for rabies by laboratory examination.

The indication for PEP with or without rabies immunoglobulin depends on the type of contact with the rabid animal.

Combined immunoglobulin and vaccine

Combined immunoglobulin–vaccine treatment is the best specific systemic treatment available for PEP of rabies in humans, although vaccine alone is sufficient for minor exposures (Category II, see Table 2). Rabies immunoglobulin for passive immunization is administered only once; preferably at, or as soon as possible after, the initiation of post-exposure vaccination. Beyond the 7th day after exposure, rabies immunoglobulin is not indicated because an active antibody response to the CCV is presumed to have occurred. The dose of human rabies immunoglobulin is 20 IU/kg body weight; for equine immunoglobulin and F(ab')2 products it is 40 IU/kg body weight. All of the rabies immunoglobulin, or as much as anatomically possible to avoid possible compartment syndrome, should be administered into or around the wound site or sites. The remaining immunoglobulin, if any, should be injected intramuscularly at a site distant from the site of vaccine administration. The treating physician should be prepared to manage anaphylaxis which, although rare, could occur during any stage of administration.

References

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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 and abdominal pain), general malaise and weakness. These symptoms may affect working and learning capacities and impair physical growth.

All soil-transmitted helminths compete with the host for nutrients, causing malabsorption of fats, proteins, carbohydrates and vitamins, and contribute directly to malnutrition. Among infants and children, they can cause growth retardation.

Ascaris infection exacerbates vitamin A deficiency (which leads to night blindness), and elimination of ascarids may result in rapid clinical improvement in night blindness and dryness around the eye.

Hookworm infections may cause an itchy maculopapular rash at the site of larval penetration. Transient pneumonitis, epigastric pain, anorexia, diarrhoea and eosinophilia may occur, complicated by iron-deficiency anaemia due to chronic intestinal blood loss.

Heavy *Trichuris* infection may cause diarrhoea and severe malabsorption of nutrients. In endemic regions, typically only 10% of those infected have heavy worm burdens and suffer most from the disease due to 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 soil-transmitted helminths are:

- *Ascaris lumbricoides* or roundworm (white or pinkish adult worm, 15–30 cm long)
- hookworms – *Ancyclostoma duodenale* and *Necator americanus* (small, cylindrical, greyish white nematodes, 7–13 mm long)
- *Trichuris trichiura* or whipworm (pinkish grey adult worm, 4 cm long).

Case classification

Ascariasis

Suspected

A suspected case of ascariasis is one in which there are abdominal symptoms (mild abdominal discomfort, dyspepsia, loss of appetite, nausea and malnutrition) or respiratory symptoms (nonproductive cough, chest discomfort and eosinophilic pneumonitis) and a history of worms in the stools.

Confirmed

A confirmed case of ascariasis is a suspected case in which there is passage of *A. lumbricoides* (anus, mouth and nose), or presence of *A. lumbricoides* eggs in stools (confirmed on microscopic examination).

Hookworm infection

Suspected

A suspected case of hookworm STH infection is one in which there is severe anaemia with no other obvious cause.

Confirmed

A confirmed case of hookworm STH infection is a suspected case in which there are hookworm eggs in the stools (shown by microscopic examination).

Trichuriasis

Suspected

A suspected case of trichuriasis is one in which there are bloody and mucoid stools.

Confirmed

A confirmed case of trichuriasis is a suspected case in which the presence of *T. trichiura* eggs in stools is confirmed.

Mode of transmission

Ascariasis and trichuriasis are transmitted by ingestion of their eggs, mainly as contaminants of food. Hookworm lavae in the soil are transmitted by active penetration of the skin.

Incubation period

The incubation period is:

- 4–8 weeks for *A. lumbricoides*
- a few weeks to many months for hookworm disease
- a few weeks to 3 months for *T. trichiura*.

Period of communicability

Ascariasis

A. lumbricoides eggs appear in the stools 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; they 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 infection

Hookworm eggs appear in the stools 6–7 weeks after infection. As larvae, they become infective in soil after 7–10 days, and can remain infective for several weeks. Infected individuals can contaminate soil for several years (3–5 years for *N. americanus*, and about 1 year for *A. duodenale*).

Tricuriasis

T. trichiura eggs appear in the stools 70–90 days after ingestion, and become infective in soil after 10–14 days. Infected individuals can contaminate soil for 1–3 years.

Reservoirs

Humans are the reservoir for STH infections.

Epidemiology

Disease burden

STH infections are among the most prevalent infections in the world; they are a leading cause of morbidity, particularly in the developing world. Sri Lanka is endemic for STH infections, with ideal climatic conditions for their transmission. Those most at risk include preschool and school-age children, women of childbearing age, women in the second or third trimester of pregnancy, lactating

mothers and adults in certain occupations (e.g. tea-pickers and miners). Due to the lack of epidemiological data in most countries and territories, and the close link between infection and poverty, the World Health Organization (WHO) has assumed that all preschool and school-age children are at risk in endemic countries and territories. Details for Sri Lanka are given in Table 1.

Table 1. **Population indicators for soil-transmitted helminths, Sri Lanka (*1*)**

	Preschool-age children			School-age children		
Year	Population at risk of STH infection	Reported number treated	Epidemiological coverage (%)	Population at risk of STH infection	Reported number treated	Epidemiological coverage (%)
2007	1 170 507	–	–	3 043 721	349 224	11.47
2005	1 197 342	387 631	32.37	3 118 104	2 317 296	74.32
2003	1 240 529	393 654	31.73	3 229 765	1 488 293	46.08

STH, soil-transmitted helminth.

Definitions

The following definitions are used in STH infection:

- *population at risk* – total population of preschool and school-age children living in all the endemic areas in a country who require preventive chemotherapy
- *geographical coverage* – proportion (%) of endemic administrative units in a country covered by preventive chemotherapy
- *programme coverage* – the proportion (%) of individuals treated as per the programme target
- *epidemiological coverage (national)* – the proportion (%) of the population at risk in the country treated with preventive chemotherapy.

Declining prevalence in previously high-transmission areas has been noted in the absence of a national STH control programme. This could be due to parents regularly treating their children with inexpensive, broad-spectrum anthelminthics, such as mebendazole, which is easily available. More recently, a national control programme in Sri Lanka has reportedly reduced prevalence to less than 5%.

Geographical distribution

Ascaris and *Trichuris* occur in rural areas and in urban environments, especially urban slums. The prevalence of *Ascaris* infection is actually greatest in some urban

environments. In contrast, high rates of hookworm infection are typically restricted to rural areas where poverty predominates.

Seasonality

Distribution is influenced by environmental parameters – especially temperature, humidity and soil dryness – that affect the survival of eggs and larvae in the environment. Transmission is most intense immediately after rainy seasons, and is lowest during prolonged dry seasons. Studies in Sri Lanka have correlated wet days with increased *Ascaris* infections.

Risk factors for increased burden

Population movement

If population displacement for extended periods leads to poor sanitary facilities, risk of infection is increased.

Overcrowding

Overcrowding may increase transmission through poor sanitation and poor hand-washing practices.

Poor access to health services

Poor access to health services is not a major risk factor, because treatment of entire endemic communities at regular intervals has only a limited effect on breaking the transmission cycle and reducing overall transmission rates. However, increased morbidity in individuals with STH infections is likely, as poor access to health services will limit access to preventive chemotherapy.

Food shortages

Malnutrition and STH infections are synergistic in causing iron-deficiency anaemia and vitamin A deficiency.

Lack of safe water, poor hygiene practices and poor sanitation

Poor sanitation and poor hygiene practices can increase the risk of STH infection.

Prevention and control measures

Case management

STH infections can be controlled with inexpensive interventions. The average treatment cost during a school-based treatment campaign (including drugs, distribution and monitoring activities) is approximately US$0.10–0.15 per child.

For treatment, the WHO recommends any of the following drugs, all of which can be safely administered to children more than one year of age:

- albendazole, 400 mg single dose (200 mg in children aged 1–2 years)
- mebendazole, 500 mg single dose
- levamisole, 2.5 mg/kg body weight single dose
- pyrantel, 10 mg/kg body weight single dose.

Notes on these drugs are as follows:

- Albendazole and mebendazole are used in preference to levamisole and pyrantel, which are more difficult to administer in large-scale drug distribution interventions.
- These drugs must not be given to women during their first trimester of pregnancy.
- Where mass treatment with albendazole for lymphatic filariasis is envisaged, the drug will also be effective against STH infections.
- Iron supplementation is also recommended in communities with high prevalence of iron-deficiency anaemia (e.g. 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. However, even if there is no improvement in sanitation, regular treatment of school-age children and other high-risk groups will help to avoid the worst effects of infection. Implementing and sustaining universal treatment twice a year is recommended for high-risk communities with a prevalence of STH above 50%. Once-a-year treatment is sufficient in low-risk communities with a prevalence of STH of 20–50%. High-risk groups are described above under 'Disease burden'.

In 2001, World Health Assembly Resolution 54.19 urged all Member States endemic for STH to attain "a minimum target of regular administration of chemotherapy

to at least 75% and up to 100% of all school-age children at risk of morbidity by 2010". Although no target was set for preschool-age children, they are also at high risk.

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 reduce 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.

In Sri Lanka, school children are currently dewormed as part of the school health inspection for students in years 1, 4 and 7. All students are dewormed in schools with less than 200 children in the 5 districts where school children have particularly high levels of anaemia (Ampara, Anuradhapura, Hambanthotam, Ratnapura and Vavuniya).

A recommended treatment strategy is given in Table 2. Health education regarding safety of food and water, and proper sanitation is also important.

Table 2. **Recommended treatment strategy for soil-transmitted helminth infections in preventive chemotherapy[a]**

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-age children ■ women of childbearing age, including pregnant women in the 2nd and 3rd trimesters, and lactating mothers ■ adults in high-risk occupations (e.g. tea-pickers and miners).
Low-risk community	20–50%	Treat all school-age children (enrolled and not enrolled) once each year	Also treat: ■ preschool-age children ■ women of childbearing age, including pregnant women in the 2nd and 3rd trimesters, and lactating mothers ■ adults in high-risk occupations (e.g. tea-pickers and miners)

STH, soil-transmitted helminth.

[a] When prevalence of any STH infection 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, appropriate treatment frequency would be every 4 months.

Immunization

No vaccine against STH infections is available.

Epidemic control

STH infection is usually endemic, with little likelihood of rapid changes in incidence.

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

TETANUS

Description

Clinical description

Tetanus results from contamination of wounds with the spore-forming bacterium *Clostridium tetani*. The bacterium produces tetanospasmin (tetanus toxoid, TT), a neurotoxin that causes muscle spasm and contraction, with the characteristic fixed smile, locked jaw and arching back, and sudden, generalized seizures.

Infectious agent

The infectious agent is the bacterium *C. tetani*.

Case classification for neonatal tetanus

Suspected

A suspected case of tetanus is a neonatal death between 3 and 28 days of age in which the cause of death is unknown, or any neonate between 3 and 28 days of age reported as having neonatal tetanus and not investigated.

Confirmed

A confirmed case of tetanus is a neonate with a normal ability to suck and cry during the first 2 days of life, who, between 3 and 28 days of age, cannot suck normally, and becomes stiff or has spasms (i.e. jerking of the muscles).

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.

Mode of transmission

Infection occurs when *C. tetani* spores – found worldwide in soil and in the gastrointestinal tracts of animals (including humans) – are introduced into the body through any type of wound (open wounds, puncture wounds or surgical sites) via soil or objects contaminated with animal or human faeces. Cases have followed wounds considered too trivial for medical attention.

Neonatal tetanus usually occurs through the introduction of spores via the umbilical cord (e.g. through use of a contaminated instrument to cut the umbilical cord during delivery, or through the application of contaminated materials to the umbilical stump after delivery).

Incubation period

The incubation period for tetanus is usually 3–21 days (average 10 days; range from 1 day to several months). Shorter incubation periods are associated with heavily contaminated wounds, more severe disease and poor prognosis.

Period of communicability

There is no direct person-to-person transmission of tetanus.

Reservoirs

C. tetani is a normal and harmless inhabitant of the intestines of horses, other animals and humans. Tetanus spores are ubiquitous in the environment.

Epidemiology

Disease burden

Despite being an easily preventable disease, tetanus – in particular maternal and neonatal tetanus – remains a major cause of mortality in the developing world. In 2007, 17 012 cases of tetanus and 6067 of neonatal tetanus globally were reported to the World Health Organization (WHO). By the end of 2008, 46 countries had yet to meet the target for elimination of neonatal tetanus. Sri Lanka has seen a steady decline in the incidence of adult tetanus and neonatal tetanus since the launch of its Expanded Program on Immunization in 1978, and has maintained relatively low numbers of tetanus cases compared to its neighbours (Table 1).

Table 1. **Reported cases of tetanus and vaccination coverage by year, Sri Lanka, 2003–07 (*1*)**

Year	Reported cases of neonatal tetanus	TT2+ vaccination coverage (%)	Reported cases of tetanus	DTP3 vaccination coverage (%)*
2007	0	96	44	98
2006	2	95	37	98
2005	0	90	20	99
2004	1	95	32	97
2003	1	96	30	99

TT2+, at least 2 doses of tetanus toxoid in pregnancy; DTP3: third dose of diphtheria–tetanus–pertussis vaccine.

Geographical distribution

Worldwide, tetanus is more common in agricultural areas, where contact with animal excreta is more frequent and immunization coverage is inadequate. It is also more common in rural, impoverished and tropical areas that bear the bulk of tetanus occurrence.

Seasonality

Tetanus is not seasonal.

Risk factors for increased burden

Population movement

Mass population displacement may increase the risk of tetanus by putting people at risk of accidental injuries and inadequate wound care.

Overcrowding

Overcrowding is not relevant to risk of tetanus.

Poor access to health services

In settings where access to health services is poor, proper wound management and adequate care during childbirth are less likely. Poor access to wound care and immunization programmes increases the risk of acquiring tetanus.

Food shortages

Food shortages are not relevant to tetanus.

Lack of safe water, poor hygiene practices and poor sanitation

Poor wound care allows tetanus spores to settle on open wounds. Poor sanitation practices allow for increased contamination of fomites with spores.

Prevention and control measures

Case management

Case management for tetanus involves:

- prompt treatment with tetanus immunoglobulins (if available), tetanus antitoxin (if immunoglobulins not available), or tetanus toxoid (TT), plus antibiotics

- appropriate wound treatment
- airway management
- intensive care (e.g. sedation and muscle relaxants).

Recovery from clinical tetanus does not provide protection against the disease in the future; hence, individuals who have recovered from infection require immunization.

Annex 6 has information on management of tetanus-prone wounds.

Prevention

Immunization is the best means of prevention; strategies include:

- education of public on the necessity of immunization
- universal active immunization with TT (or vaccines containing TT)
- a full primary course of TT plus tetanus immunoglobulin (if available) for tetanus-prone wounds such as puncture, missile, burn and sepsis wounds; those contaminated with soil or manure; and any wound more than 6 hours old.

Prevention of maternal and neonatal tetanus requires maternal immunization with TT and use of hygienic delivery practices (e.g. assistance by a trained attendant, delivery in a clean health facility and modification of harmful traditional practices).

Immunization

Sri Lanka currently provides 6 doses of TT in its national immunization schedule:

- 4 doses in combination with diphtheria and pertussis-containing vaccines at 2, 4, 6 and 18 months of age
- one dose of a diphtheria–tetanus (DT) vaccine at school entry
- one dose of a diphtheria–tetanus (Td) vaccine during adolescence.

Pregnant women receive 2 doses of TT during a first pregnancy, and a single dose each in 3 subsequent pregnancies. In addition, TT is widely used for post-exposure vaccination in all medical institutions (*2*).

Epidemic control

Outbreaks of tetanus are rare, but when they occur, it is important to carry out a thorough case investigation and a search for a common source (e.g. repeated use

of needles for injections, unhygienic medical or delivery procedures, low vaccination coverage or contaminated drugs).

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Further reading

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Annex:

- Annex 6. WHO fact sheets and information sources

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

TUBERCULOSIS

Description

Clinical description

The most important symptom of pulmonary tuberculosis (TB) is a productive cough of long duration (> 2weeks). Other symptoms include haemoptysis, significant weight loss, chest pain, breathlessness, fever, night sweats, tiredness and loss of appetite. Extrapulmonary TB may involve bone, the brain (meningitis), lymph nodes and any organ including the kidney, the liver and the spleen.

Infectious agent

The infectious agent in TB is the bacterial complex *Mycobacterium tuberculosis.* This complex 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 classification

Suspected tuberculosis

A suspected case of TB is one in which the person has symptoms or signs suggestive of TB; in particular, cough of long duration (> 2 weeks or in accordance with current recommendations of Sri Lanka's National Programme for Tuberculosis Control and Chest Diseases).

Pulmonary tuberculosis

Pulmonary TB refers to disease involving the lung parenchyma. Tuberculous intrathoracic lymphadenopathy (mediastinal and/or hilar) or tuberculous pleural effusion without lung involvement are extrapulmonary TB. A patient with both pulmonary and extrapulmonary TB should be classified as having pulmonary TB.

Smear-positive pulmonary tuberculosis

The current case definition of sputum smear-positive pulmonary TB is a patient with at least one sputum sample microscopically positive for acid-fast bacillus (AFB).

Smear-negative pulmonary tuberculosis

(a) The revised case definition of smear-negative pulmonary tuberculosis is all of the following:

- two or more sputum specimens negative for AFB
- no clinical response to a course of broad-spectrum antibiotics
- radiographic abnormalities consistent with active pulmonary TB
- a decision by a clinician to treat with a full course of anti-TB chemotherapy.

(b) In HIV-prevalent and resource-constrained settings, the case definition of smear-negative pulmonary tuberculosis is all of the following:

- two or more sputum specimens negative for AFB
- radiographic abnormalities consistent with active tuberculosis
- laboratory confirmation of human immunodeficiency virus (HIV) infection or strong clinical evidence of HIV infection
- a decision by a clinician to treat with a full course of anti-TB chemotherapy.

If there is no laboratory confirmation of HIV infection or the patient has no strong clinical evidence of HIV infection, the criteria listed in (a) should be used.

(c) This group also includes cases without a smear result, which is rare in adults, but more common in children. A patient whose initial sputum smears were negative, but whose subsequent sputum culture result is positive is termed a "smear-negative pulmonary TB case", regardless of HIV status.

Extrapulmonary tuberculosis

Extrapulmonary TB (EPTB) refers to TB of organs other than the lungs; for example, the 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 EPTB, 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; for example, peripheral lymphadenitis with swelling of cervical or axial lymph nodes, chronic evolution or production of caseous discharge. Other cases usually require a referral to a hospital for assessment; for example, TB of bone or joints, TB peritonitis, TB laryngitis and severe life-threatening forms (e.g. miliary TB and TB meningitis).

Mode of transmission

TB is transmitted by 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 unpasteurised milk or dairy products, but is rare.

Incubation period

The incubation period for TB is 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

TB is communicable 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 are the main reservoir for TB; in some areas, diseased cattle are reservoirs.

Epidemiology

Disease burden

The World Health Organization (WHO) South-East Asia Region bears 35% of the global TB burden; Sri Lanka is an intermediate TB burden country. The estimated number of TB cases has progressively increased, from 10 353 in 1990 to 11 676 in 2007, while the estimated incidence remained stable at 60 new cases per 100 000 population over the same period. The rate of TB case detection and treatment success was 87 in 2006 and 86 in 2007 (*1*). Currently, the main challenge is to provide TB services in conflict-affected areas.

HIV prevalence in Sri Lanka is low in the general population; it is less than 1% among those being treated for TB. As 80% of the HIV cases are in the age group 20–44 years, the TB–HIV association is of potential significance and increasing numbers of people with TB–HIV coinfection can be expected.

The emergence of multidrug-resistant tuberculosis (MDR-TB) – defined as resistance to at least isoniazid and rifampicin – threatens TB programmes worldwide. Among the 827 positive cultures on which drug sensitivity testing was performed in 2004, 12 were detected as MDR. At present, MDR-TB among newly diagnosed cases in Sri Lanka is 1.1%.

Geographical distribution

The highest rates of infection with TB in Sri Lanka are in the most densely populated areas, such as Colombo and other urban areas.

Seasonality

TB does not show any seasonality.

Alert threshold

An alert threshold is not applicable for TB.

Risk factors for increased burden

Population movement

Population displacement increases the risk of transmission by disrupting existing TB control activities. Also, movement of untreated TB patients into new areas spreads the disease, and movement of susceptible individuals (e.g. immunosuppressed, malnourished, or HIV-infected individuals) into TB-endemic areas or camps increases the risk of infection. Treatment interruptions, treatment failure, relapse and nonadherence to combination therapy lead to persistent reservoirs of TB and increase the risk of MDR-TB.

Overcrowding

Overcrowding and poor indoor ventilation contribute to increased risk of transmission of TB.

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 in the absence of effective treatment. Irregularity in treatment administration is one of the most important causes of development of MDR-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 hygiene practices and poor sanitation

Lack of safe water, poor hygiene practices and poor sanitation are not relevant to transmission of TB.

Other

HIV–TB coinfection is associated with higher mortality.

Prevention and control measures

Case management

Two sputum samples should be examined by light microscopy for AFB by Ziehl-Neelsen staining. Early morning sputa have better yield; therefore, at least one sample should be from an early morning collection. If more than 1 sputum smear is positive, the patient should be registered with the health authorities and started on anti-TB treatment. If both sputum smears are negative but suspicion of TB remains high, a short (≥ 1 week) trial of antibiotics should be given as treatment for acute respiratory infections (e.g. amoxicillin or cotrimoxazole; patients should not be given 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 pulmonary TB cases are expected to be confirmed by positive sputum smear examination. Chest X-ray (CXR) lesions compatible with active TB should encourage further sputum examination if the two sputum smear examinations were negative. CXR itself is not a diagnostic tool for pulmonary TB; however, in some circumstances, a compatible CXR, together with symptoms consistent with TB, will lead to the diagnosis of pulmonary TB in smear-negative cases. Thus, if the two samples are again negative after a trial of antibiotics, the decision as to whether the patient is categorized as having smear-negative pulmonary TB-negative should depend on either a compatible CXR interpreted by an experienced physician or, in the absence of X-ray facilities, the experienced physician's judgement alone.

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 in children should be considered in a child if there is one or more of the following:

- 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
- increasing irritability and drowsiness.

A hospital referral for X-ray and special examinations (e.g. lumbar puncture in a case of TB meningitis suspicion) is often required. Children with headaches, change

of temperament, recent squint or ocular muscle paralysis should be suspected of TB meningitis. Children with high fevers, dyspnoea, gastrointestinal symptoms or confusion should be suspected of acute miliary TB. Suspected TB of the bone, tuberculous arthritis or pleural effusions also require referral.

Close contacts (household or shelter members) should be screened for TB, giving priority to children and people with underlying conditions such as HIV infection.

Following the diagnosis of TB, and before the beginning of treatment, patients should be questioned carefully about whether they have ever taken anti-TB drugs before. Patients are classified according to:

- disease site
- disease severity
- bacteriological status (assessed by sputum microscopy)
- history of anti-TB treatment (new or previously treated).

For new cases that are sputum smear positive, good case management includes directly observed treatment during the intensive phase, continuation of rifampicin-containing regimens, and the entire re-treatment regimen. Sri Lanka has a well-established directly observed treatment, short-course (DOTS) programme that achieves 98% coverage, except in conflict areas. The DOTS success rate was 87% in a 2006 case cohort (*2*).

The management approach is summarized in Figure 1. There are three main types of treatment regimen:

- *Category I* for new smear-positive pulmonary cases and severely ill TB patients
- *Category II* for re-treatment cases
- *Category III* for smear-negative pulmonary or extrapulmonary cases (not severely ill patients).

TB drugs should be given to TB patients in fixed-dose combination forms. The chemotherapeutic regimens are based on standardized combinations of five essential anti-TB drugs:

- ethambutol (E)
- isoniazid (H)
- pyrazinamide (Z)
- rifampicin (R)
- streptomycin (S).

Each of the standardized chemotherapeutic regimens consists of an initial and a continuation phase, as outlined below.

Initial (intensive) phase

The initial phase lasts for 2–3 months, with 3–5 drugs given daily under direct observation, for maximum reduction in the number of TB organisms.

The number of drugs used relates to the risk of failure of treatment due to possible bacterial resistance.

Continuation phase

The continuation phase lasts for 4–6 months, with 2–3 drugs (including rifampicin) given 3 times a week under direct observation; alternatively, in some cases (e.g. during repatriation of refugees), 2 drugs (ethambutol and isoniazid) are given daily unsupervised for 6 months, but in a fixed-dose combination form.

- All doses of rifampicin-containing regimens should be directly supervised.
- Actual swallowing of medication must be observed by a health worker or treatment supporter.
- Hospitalized patients should be kept in a separate ward for the first 2 weeks of treatment.

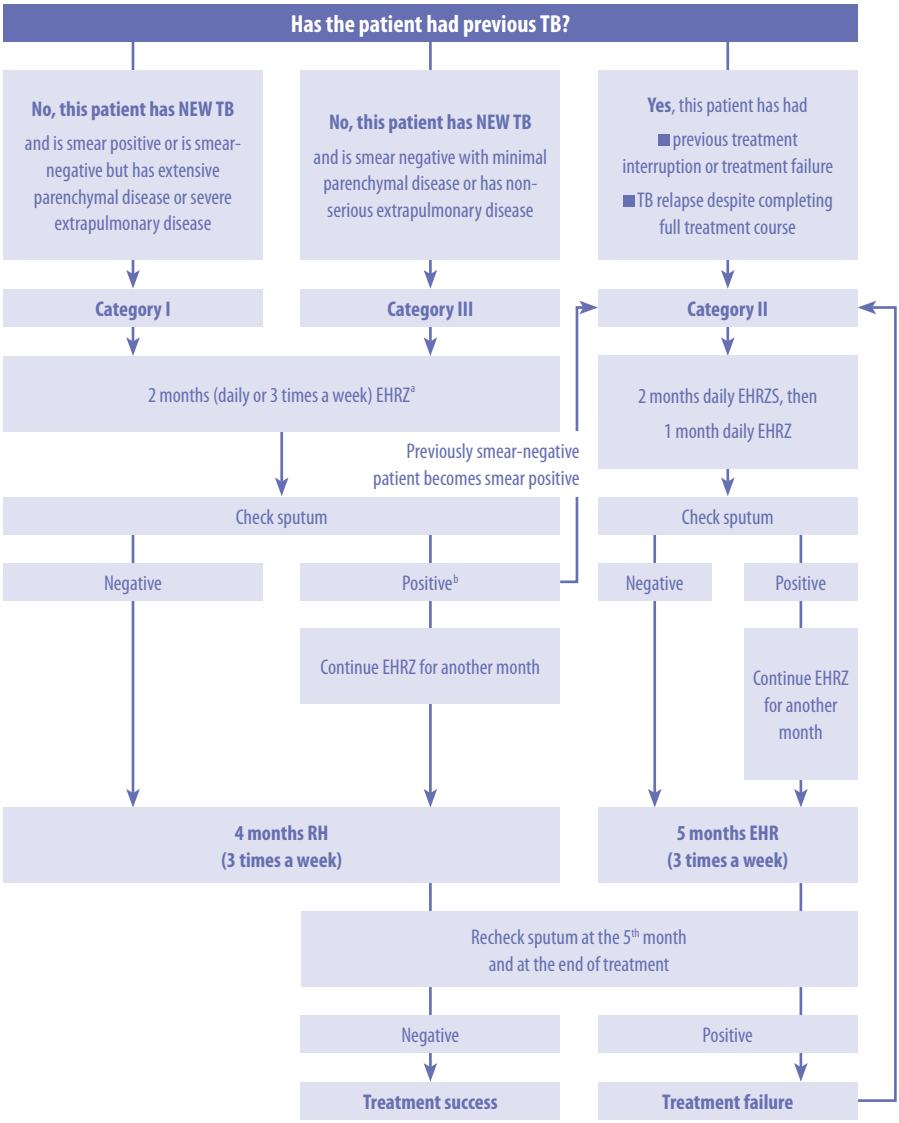
Notes on regimens

Regimens are written in short form, with the number of months the medication is to be given in front of the letter, and the doses per week written after the letter. If there is no number after the letter used to denote the anti-TB drug, a daily dosage is given. The symbol "/" separates the different phases of the therapy. Thus, 2RHZE/4H3R3 means that for the first 2 months of treatment, ethambutol, isoniazid, pyrazinamide and rifampicin should be given daily, followed by 4 months of isoniazid and rifampicin given regularly, but each given only three times per week.

Programmatic considerations

Implementation of a TB programme is an important component of health-care services for emergency-affected and displaced populations, once basic primary health-care services are in place. Despite the challenges of these settings, it is feasible to put in place an effective programme. For example, in 2005, the WHO global target to control TB (70% case detection and 85% treatment success) was reached in Somalia, where civil war has been ongoing for more than 15 years.

Figure 1. **Flowchart for the management of tuberculosis (TB)**



[a] 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.
[b] A previously smear-negative patient who becomes smear positive while on TB treatment should be re-registered as treatment failure and started on Category II treatment.

E, ethambutol; H, isoniazid; R, rifampicin; S, streptomycin; Z, pyrazinamide

The following criteria are essential before a decision is made to implement a TB programme for emergency-affected and displaced populations:

- data from the refugee or displaced population indicate that TB is an important health problem
- the emergency phase is over (death rates are < 1/10 000 population per day)
- basic needs of water, adequate food, shelter and sanitation are met
- essential clinical services and basic drugs for common illnesses are available
- basic health services are accessible to a large part of the population, so that suspected TB patients can be identified, and appropriate investigation or referral arranged.

The basis of an effective TB programme is in line with the Stop TB Strategy. The following elements must be considered a priority:

- political commitment to TB control with sustained funding
- case detection through quality-assured bacteriology
- standardized treatment with supervision and patient support
- an effective system for drug supply and management
- an effective monitoring and evaluation system, with impact measurement.

Prevention

Detection and treatment of smear-positive (infectious) TB cases are the most effective interventions to prevent the transmission of TB. Children under 5 years of age who are close contacts of smear-positive pulmonary TB patients and who, after investigation, have no active TB, should receive isoniazid prophylaxis as follows: 5 mg/kg per day for 6 months, with a steady follow-up (e.g. every 2 months). This will significantly reduce the likelihood of TB disease occurrence. Children over 5 years of age who are well do not require prophylaxis, but only clinical follow-up.

Infection control measures include good ventilation and reduction of overcrowding in health clinics, and placement of hospitalized patients in a dedicated ward (separate from HIV-positive individuals) for at least the first 2 weeks of treatment.

Key elements of community health education are de-stigmatization of TB patients, curability of TB disease, early (self) referral of TB suspects, importance of adherence to treatment and contact investigation.

Immunization

The bacille Calmette-Guerin (BCG) vaccine has been shown to be effective in preventing severe forms of the disease, such as TB meningitis and miliary TB, in young children. BCG vaccination of newborns is part of Sri Lanka's national immunization schedule. The coverage reached 99% in 2007, and has been consistently high since at least 2003 (*3*).

Epidemic control

Epidemic control for TB involves recognizing and treating 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, with symptoms including mental dullness and meningitis with intestinal haemorrhage and perforation in 1% of severe cases.

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 resistant to all three of the first-line antibiotics (chloramphenicol, ampicillin and cotrimoxazole) 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 under 2 years of age.

Paratyphoid fever is similar in its symptoms to typhoid fever, but tends to be milder, with a lower CFR.

Infectious agent

The infectious agent for typhoid fever is the bacterium *Salmonella enterica* subsp. *enterica* serovar Typhi (commonly known as *S.* Typhi, the latter not italicized in this new nomenclature).

The infectious agent for paratyphoid fever is *Salmonella enterica* subsp. *enterica* serovar Paratyphi var. A & B (commonly known as *S.* Paratyphi A & B; again, new nomenclature).

Case classification

Suspected

Clinical diagnosis of typhoid fever 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 supportive.

Confirmed

A confirmed case of typhoid fever is a suspected case with isolation of *S.* Typhi from blood or stool cultures.

Carrier

A carrier is an individual with *S.* Typhi organisms persisting in stools or urine for more than a year after disease onset.

Mode of transmission

Transmission of typhoid fever is by the faecal–oral route, particularly from ingestion of food and water contaminated by faeces and urine of patients and carriers. Other sources of infection include shellfish taken from sewage-contaminated beds, vegetables fertilized with human waste and eaten raw, contaminated milk and milk products. The highest incidence usually occurs where faecal matter contaminates water supplies serving large populations. About 2% of infected adults are faecal carriers. Patients with concurrent *Schistosoma haematobium* infection are at higher risk of becoming urinary carriers of *S.* Typhi.

Incubation period

The incubation period for typhoid fever depends on the inoculum size and host factors, but is usually 8–14 days (range 3 days to > 2 months). For paratyphoid, it is 1–10 days.

Period of communicability

Typhoid and paratyphoid are communicable from the symptomatic period for 2 weeks, but people can transmit the disease as long as the bacteria remains in their body, and 2–5% of infected cases remain carriers for several months or longer. Chronic asymptomatic carriers are strongly implicated in spread of the disease.

Reservoirs

Humans are the reservoir for both typhoid and paratyphoid. Domestic animals can be the reservoir for paratyphoid, but this is rare.

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 that of other febrile illnesses; also, the laboratory diagnosis in developing countries is not standardized.

The highest incidence of typhoid fever occurs in Asia, with 274 cases per 100 000 population, especially in South-East Asian countries and the Indian subcontinent.

Recent studies conducted in China, India, Indonesia, Pakistan and Viet Nam revealed high rates of typhoid fever among children (including those under 5 years of age) in urban slums. Other studies in South-East Asia suggest that the incidence is highest in children under 5 years of age, with higher rates of complications and hospitalization.

A total of 1933 cases of typhoid fever were notified in 2008.

Geographical distribution

In 2008, the highest number of notified cases in Sri Lanka were reported from the following districts – Nuwara Eliya (261), Jaffna (261), Colombo (205), Mannar (165) and Puttalam (165). The following districts recorded less than 10 cases – Ampara (9), Hambatota (8) and Kilinochchi (1) (*1*).

Seasonality

Most cases occur between January and March; the fewest occur between July and September.

Alert threshold

Two or more linked cases of typhoid fever must lead to an epidemiological investigation.

Outbreaks

No recent outbreaks of typhoid fever have been reported in Sri Lanka.

Risk factors for increased burden

Population movement

Complex emergency settings with population movement, particularly those in which there is a lack of safe food and water, and a lack of access to adequate sanitation facilities, can lead to outbreaks of typhoid fever. Dissemination of multidrug-resistant (MDR) strains of *S.* Typhi as a result of population movement is an emerging issue.

Overcrowding

Overcrowding increases contact with infected individuals and increases transmission of typhoid fever.

Poor access to health services

Early detection and containment of cases are vital for 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

Food shortages are not relevant to the risk of infection with typhoid fever.

Lack of safe water, poor hygiene practices and poor sanitation

In the general population, the risk of typhoid fever is related to the availability of safe food and water, and access to adequate sanitation facilities. Poor hygiene practices in food preparation and handling, washing, and after defecation are further contributors. Although largely considered an endemic disease, epidemics of typhoid fever do occur, frequently as a result of breakdowns in water supplies and sanitation systems.

Others

MDR strains of *S.* Typhi, including strains resistant to ciprofloxacin, are emerging. Milk and dairy products are an important source of infection.

Prevention and control measures

Case management

Case management involves early antimicrobial treatment, selected according to the antimicrobial resistance pattern of the strain. Quinolones (e.g. ciprofloxacin), cotrimoxazole, chloramphenicol and ampicillin are generally used to treat typhoid fever.

Multiresistant strains of *S.* Typhi are becoming increasingly common worldwide. Strains resistant to chloramphenicol and other recommended antibiotics (ampicillin, cotrimoxazole and even ciprofloxacin) have become prevalent in several areas of the world, particularly in typhoid endemic areas such as South-East Asia.

Dehydration prevention and case management using oral rehydration salts (ORS) therapy is important.

Prevention

Prevention of typhoid fever in any community relies mainly on adequate water, sanitation and hygiene; this involves:

- *Provision of safe drinking-water*. The World Health Organization (WHO) minimum emergency, standard provision is 20 l per person per day.
- *Provision of adequate facilities for disposal of human waste*. The minimum emergency standard is one latrine for every 20 people.
- *Provision of adequate supplies for hand-washing, bathing and laundry needs*. The minimum emergency standard is 250–500 g of soap per person per month.
- *Promotion of hygiene* – including use of latrines and prevention of human defecation on the ground, or in or near water; disposal of children's excreta in latrines; thorough hand-washing before eating, after defecation, before food preparation, and after cleaning children or changing their diapers.
- *Promotion of food safety* – through:
  - provision of clean, appropriately cooled and spatially adequate food storage facilities (for both uncooked and cooked food, and clean cooking utensils), and adequate quantities of water and fuel for cooking and re-heating
  - promotion of "five keys to safer food" – keep clean, separate raw and cooked food, cook food thoroughly, keep food at safe temperatures, use clean water and cooking tools.
- *Promotion of breastfeeding* – by providing information on the importance and protective qualities of breastfeeding for all infants and young children, and especially for those who are ill.

Immunization

Two new-generation typhoid vaccines have been shown to be safe and efficacious, and are internationally licensed for people over 2 years of age:

- Ty21a – this is a live, attenuated, oral vaccine available as
  - a liquid suspension – licensed for use in people over 2 years of age; the recommended schedule is 3–4 doses (1 dose every 2nd day), and this form provides 53–78% protection;
  - capsules – licensed for use in those aged 5 years or more; the recommended schedule is 4 doses (1 dose every 2nd day) and provides similar levels of protection to those seen with the liquid form.
- Vi polysaccharide – this is a single-dose injectable vaccine. It provides about 70% protection, and the protection lasts at least 3 years.

The need for revaccination is not well defined. However, in most endemic settings, a single booster dose of the particular vaccine 3–7 years after primary immunization seems appropriate.

In view of the continued high burden of typhoid fever and increasing antibiotic resistance, countries should consider the programmatic use of typhoid vaccines for controlling endemic disease. This is particularly appropriate given the safety, efficacy, feasibility and affordability of Vi and Ty21a.

In most countries, the control of typhoid fever will require vaccination only of those at high risk. Given the epidemic potential of typhoid fever, and observations on the effectiveness of vaccination in interrupting outbreaks, typhoid fever vaccination is also recommended for outbreak control.

Immunization of preschool and school-age children is recommended in areas where typhoid fever in these age groups is a significant public health problem, particularly where antibiotic-resistant *S.* Typhi is prevalent. Typhoid fever vaccination may be offered to those travelling to destinations where the risk of typhoid fever is high, especially to those staying in endemic areas for more than 1 month or in locations where antibiotic-resistant strains of *S.* Typhi are prevalent.

All typhoid fever vaccination programs should be implemented in the context of other efforts to control the disease, including health education, improvements in water quality and sanitation, and training of health professionals in diagnosis and treatment.

Epidemic control

Epidemics often occur as point-source epidemics, transmitted from healthy carriers to food (e.g. through 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 such as chlorination or boiling of water, and selective elimination of suspect food.

The health authorities should be informed if one or more suspected cases are identified. Typhoid is a notifiable disease in Sri Lanka. Controlling an epidemic involves:

- confirming the outbreak, following WHO guidelines (Annex 2)
- confirming the diagnosis and ensuring prompt treatment
- monitoring antibiotic sensitivity, where possible.

References

1. *Weekly Epidemiological Report*, Vol 36, No 1. Sri Lanka, Ministry of Healthcare and Nutrition, 2009 (http://www.epid.gov.lk/wer.htm, accessed 9 August 2010).

Further reading

- *Interagency diarrhoeal disease kits — information note*. Geneva, World Health Organization (WHO), February 2009 (http://www.who.int/topics/cholera/materials/en/index.html, accessed 11 August 2010).
- Prevention of foodborne disease: five keys to safer food [various resources]. Geneva, WHO, 2009 (http://www.who.int/foodsafety/consumer/5keys/en/index.html, accessed 10 August 2010).
- WHO position paper on typhoid vaccines. *Weekly Epidemiological Record*, 8 February 2008, Vol.83, 6:49–60. WHO, 2008 (http://www.who.int/wer/2008, accessed 11 August 2010).
- *Operational guidance for emergency relief staff and programme managers*. Infant Feeding in Emergencies Core Group, UNICEF et al., 2007 (http://www.who.int/child_adolescent_health/news/2008/13_05/en/index.html)
- Technical notes for emergencies [various topics]. Geneva, WHO, 2005 (http://www.who.int/water_sanitation_health/hygiene/envsan/technotes/en/index.html, accessed 10 August 2010).
- *The treatment of diarrhoea: a manual for physicians and other senior health workers*. Geneva, WHO, 2005 (WHO/FCH/CAH/05.1), (http://www.who.int/entity/child_adolescent_health/documents/ 9241593180/en, accessed 10 August 2010).
- *Acute diarrhoeal diseases in complex emergencies: critical steps*. Geneva, WHO, 2004 (http://www.who.int/topics/cholera/publications/critical_steps/en/index.html, accessed 11 August 2010).
- *Background document: the diagnosis, treatment and prevention of typhoid fever*. Geneva, WHO, 2003 (WHO/V&B/03.07) (http://www.who.int/topics/cholera/publications/en, accessed 11 August 2010).
- *First steps for managing an outbreak of acute diarrhoea*. Geneva, WHO, 2003 (WHO_CDS_CSR_NCS_2003.7) (http://www.who.int/cholera/publications/first_steps/en/index.html, accessed 11 August 2010).

Annexes:

- Annex 3: Flowcharts for the diagnosis of communicable disease outbreaks.
- Annex 4: Safe water, sanitation and hygiene.
- Annex 6: WHO fact sheets and information sources (cholera; child health in emergencies; diarrhoeal diseases; food safety; water, sanitation and health).

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

PART III

Annexes

ANNEX 1. KEY NATIONAL INDICATORS AND BACKGROUND INFORMATION

Key national indicators, Sri Lanka

Indicator		Year
Population (in thousands) total	19 207	2006
Population in urban areas (%)	15	2006
Life expectancy at birth (years) female; male	76; 69	2006
Maternal mortality ratio (per 100 000 live births)	58	2005
Neonatal mortality rate (per 1000 live births)	8	2004
Infant mortality rate (per 1000 live births) both sexes	11	2006
Under-5 mortality rate (probability of dying by age 5 per 1000 live births) both sexes	13	2006
Children under 5 years of age stunted for age (%)	18.4	2000
Children under 5 years of age underweight for age (%)	22.8	2000
Newborns with low birth weight (%)	22	2000
Births attended by skilled health personnel (%)	97	2000
Contraceptive prevalence (%)	70	2000
Nursing and midwifery personnel density (per 10 000 population)	17	2004
Physicians density (per 10 000 population)	6	2004
Population with sustainable access to improved drinking water sources (%)	82	2006
Population with sustainable access to improved sanitation (%)	86	2006
Gross national income (US$ per capita)	1 540	2006
Population living below the poverty line (% living on < US$ 1 per day)	5.6	2002
Adult literacy rate (%)	90.7	2001
Net primary school enrolment ratio female (%)	100	2003
Net primary school enrolment ratio male (%)	99	2003

Source: Global Health Observatory (http://apps.who.int/ghodata/)

National immunization schedule and coverage

National immunization schedule

Vaccine	Age
BCG	Birth
DT	5 years
DT, wP	2, 4, 6 and 18 months
DT, wP, Hib, Hep	2, 4 and 6 months (temporarily suspended in 2009)
Measles	9 months
OPV	2, 4, 6 and 18 months, 5 years
Rubella	8 years, 15–44 years women
TD	12 years
TT	First pregnancy: TT1 after 12 weeks , TT2 6–8 weeks after TT1 Subsequent pregnancies, one dose with each pregnancy up to TT5
Vitamin A	9 and 18 months, 3 years, postpartum

BCG, bacille Calmette-Guérin; DT, diphtheria–tetanus; Hep, hepatitis B; Hib, *Haemophilus influenza* B; OPV, oral polio myelitis vaccine; TD, tetanus–diphtheria; TT, tetanus toxoid; wP, pertussis, whole cell

Immunization coverage

Immunization	2007 (WHO/UNICEF estimates)
BCG	99
DTP 1st dose	99
DTP 3rd dose	98
Hep 3rd dose	98
Hib 3rd dose	–
MCV	98
TT2+	91
Polio 3rd dose	98

BCG, bacille Calmette-Guérin; DTP, diphtheria–tetanus–pertussis; Hep, hepatitis B; Hib, *Haemophilus influenza* B; MCV, measles–containting vaccine; TT2+, at least two doses of tetanus toxoid; WHO, World Health Organization; UNICEF, United Nations Childrens Fund

Reported cases of vaccine preventable diseases, 2007

Disease	Number of reported cases, 2007
Diphtheria	0
Japanese encephalitis	45
Measles	44
Mumps	1 153
Pertussis	0
Poliomyelitis	0
Tetanus neonatal	0
Tetanus total	44
Yellow fever	0

Source: WHO vaccine-preventable diseases: Monitoring system 2008 global summary http://whqlibdoc.who.int/hq/2008/WHO_IVB_2008_eng.pdf; http://www.who.int/vaccines/globalsummary/immunization/countryprofileresult.cfm?C=%27lka%27

Timeline for the humanitarian crisis

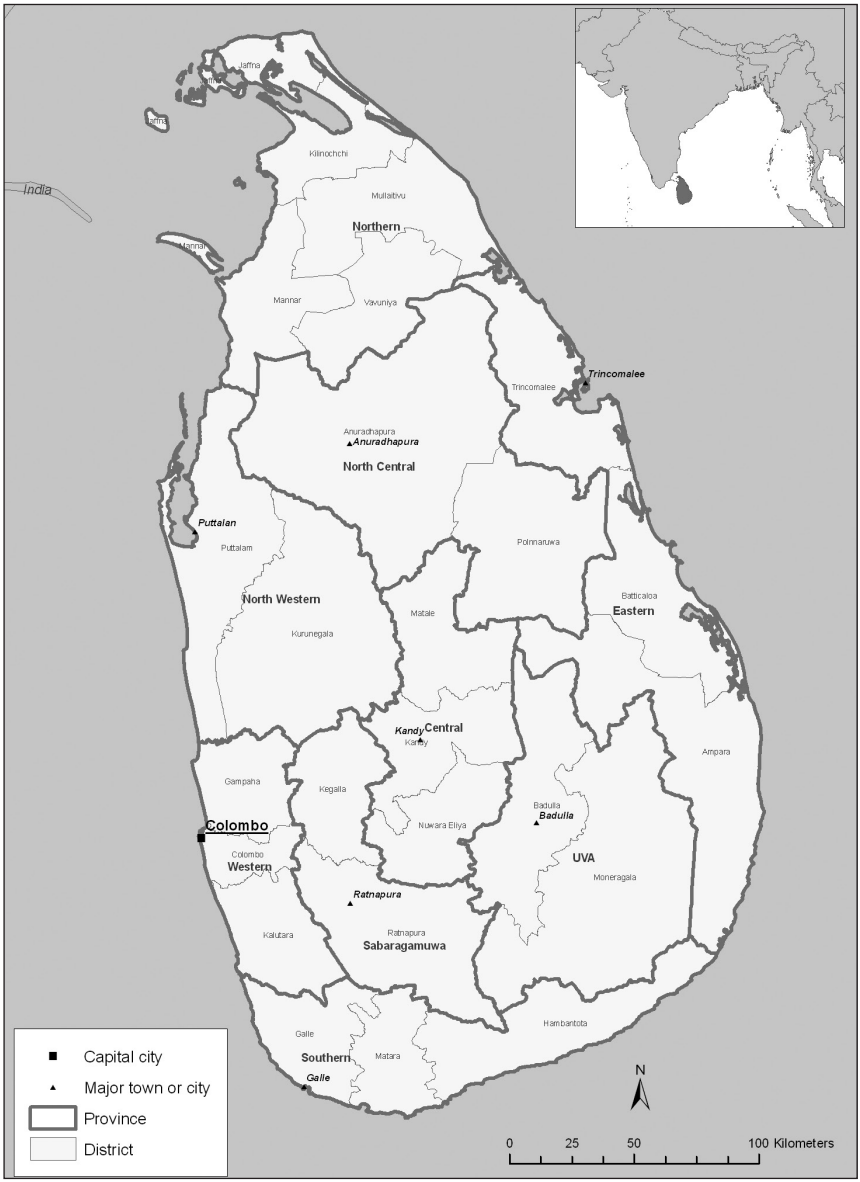
Year	Event
1948	Ceylon gains independence from Britain.
1956	Sinhala Language Act makes Sinhala the sole official language, restricting employment opportunities for Tamils, More than 10 Tamils killed in widespread violence after Tamil parliamentarians protect against new laws.
1958	Tamil riots leave more than 200 people dead. Thousands of Tamils displaced.
1972	Ceylon changes its name to Sri Lanka 1976 – LTTE formed as tensions increase in Tamil-dominated areas of north and east.
1983	Start of LTTEs "First Eelam War". Government forces deployed in the north and east.
1990	Indian peacekeeping troops leave. Start of LTTE's "Second Eelam War".
1991	Suspected LTTE suicide bomber kills Indian Prime minister Rajiv Ghandhi.
1993	Sri Lankan President Premadasa killed in LTTE bomb attack.
1994	President Kumaratunga comes to power pledging to end war. Peace talks opened with LTTE.
1995	"Third Eelam war begins".
1996	LTTE suicide bombers destroy central bank building.
1997	LTTE bombs World Trade Centre in Colombo.
1998	LTTE suicide attack on the holiest Buddhist shrine.
1999	President Kumaratunga loses an eye in LTTE suicide bomb attack.
2002	Government and LTTE sign a Norwegian-brokered ceasefire.
2004 (March)	Renegade LTTE commander, known as Karuna, leads split in rebel movement. LTTE offensive regains control of the east.
2004 (December)	More than 35 000 people are killed and one million displaced by a tsunami, devastating coastal communities.
2006/7	Multiple attacks, including the murder of 17 aid workers of Action Contre la Faim in the eastern town of Muttur (Aug 2006).
2008	Government pulls out of 2002 ceasefire agreement (Jan). Government asks UN and NGOs to be relocated from conflict-affected Vanni areas to Vavuniya.
2009	Government declares Tamil Tigers defeated after army forces overrun last patch of rebel-held territory in the northeast.

LTTE, Liberation Tigers of Tamil Eelam; NGO, nongovernmental organization; UN, United Nations

Source: Relief web Sri Lankan humanitarian profile (http://reliefweb.int/rw/fullMaps_Sa.nsf/luFullMap/A6EB798BF0DC01A08525754000599662/$File/090115_lka_hpm.pdf?OpenElement)
BBC. Timeline Sri Lanka (http://news.bbc.co.uk/1/hi/world/south_asia/country_profiles/1166237.stm)

Map resources

Map of Sri Lanka showing administrative boundaries



Additional map resources

Internal Displacement Monitoring Centre web site, IDMC, 2010 (http://www.internal-displacement.org/8025708F004CE90B/httpCountry_Maps?ReadForm&country=Sri%20Lanka&count=10000)

Relief Web, United Nations World Food Programme, 2009 (http://www.reliefweb.int/rw/fullMaps_Sa.nsf/luFullMap/F4CF81A20A51F47A8525760000708AA0/$File/map.pdf?OpenElement, accessed 10 October 2010)

ANNEX 2. STEPS IN MANAGING AN OUTBREAK

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

Establish list of diseases for surveillance and their alert thresholds – examples are given below in Table 1, and should be modified according to the context.

Table 1. Examples of list of diseases for surveillance and early warning and their alert thresholds

Health event	Alert threshold
Acute diarrhoea *Acute, bloody diarrhoea* *Acute, watery diarrhoea (suspect cholera)*	1.5 times the mean of cases calculated over the previous 3 weeks One death for acute, watery diarrhoea in patients 5 years of age or older A cluster of 5 cases in 1 week of watery diarrhoea in patients 5 years of age or older A cluster of 3–5 cases of acute, bloody diarrhoea in the same settlement in one week, or the doubling of cases in two consecutive weeks the last three weeks
Acute hemorrhagic fever syndrome	One case of acute hemorrhagic fever
Acute jaundice syndrome	A cluster of 3–5 cases of acute jaundice syndrome in the same settlement
Acute neurological syndrome *Suspected meningitis (including suspected encephalitis)* *Acute flaccid paralysis (suspected poliomyelitis)*	Two suspected cases of meningitis in the same week in a settlement One case of acute flaccid paralysis
Acute respiratory syndrome	1.5 times the mean of cases of acute lower respiratory infection reported calculated over the last three weeks
Suspected malaria	1.5 times the mean of cases calculated over the previous 3 weeks
Fever of unknown origin	Abnormal increase of fever of unknown origin associated with an unusual increase of specific mortality 1.5 times the mean of cases calculated over the previous 3 weeks
Cluster of unknown diseases	An aggregation of cases with related symptoms and signs of unknown cause that are closely grouped in time and/or place

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

- Active case-finding and confirm diagnosis (laboratory testing of samples – see Annex 3).
- Define outbreak case definition.
- Count number of cases and determine size of population (to calculate attack rate).
- Collect and analyse descriptive data to date (e.g. time and date of onset, place and location of cases, and individual characteristics such as age and sex).
- Follow up cases and contacts.
- Determine the at-risk population.
- Formulate hypothesis for pathogen/source/transmission.
- Conduct further investigation or 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).

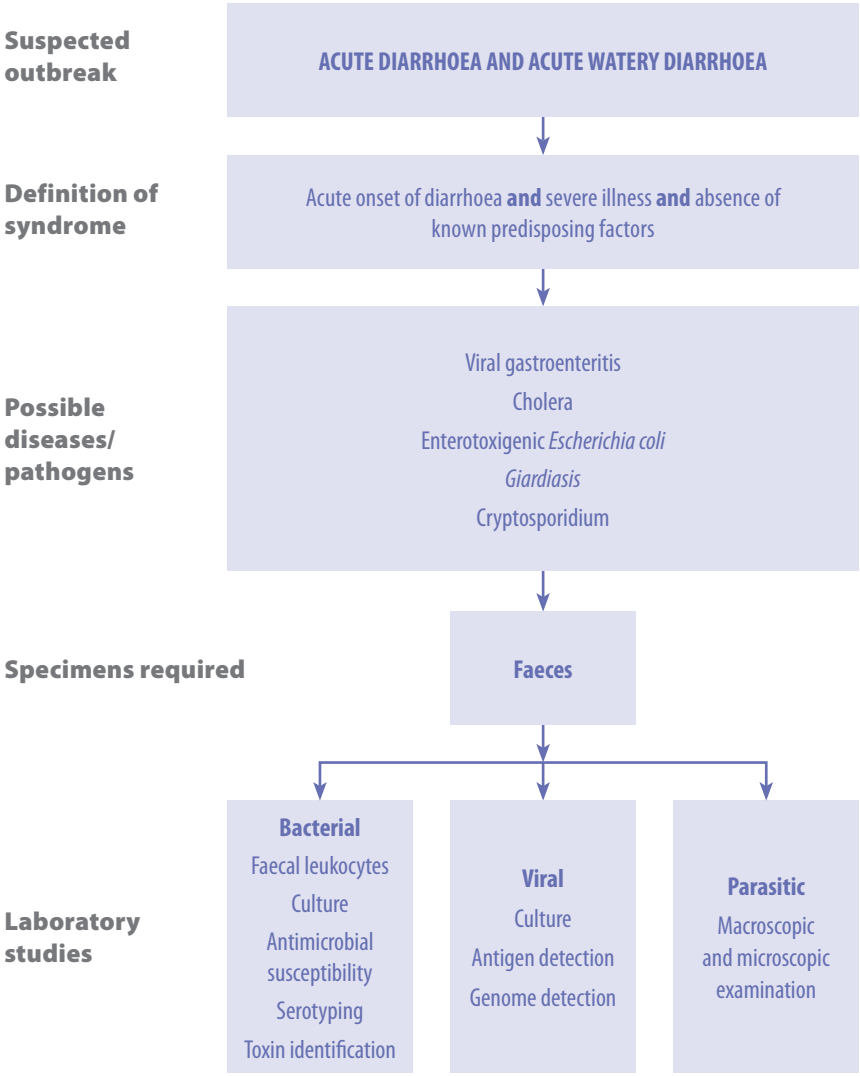
Control

- 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.
- Inform the population, follow risk communication guides (Annex 6).

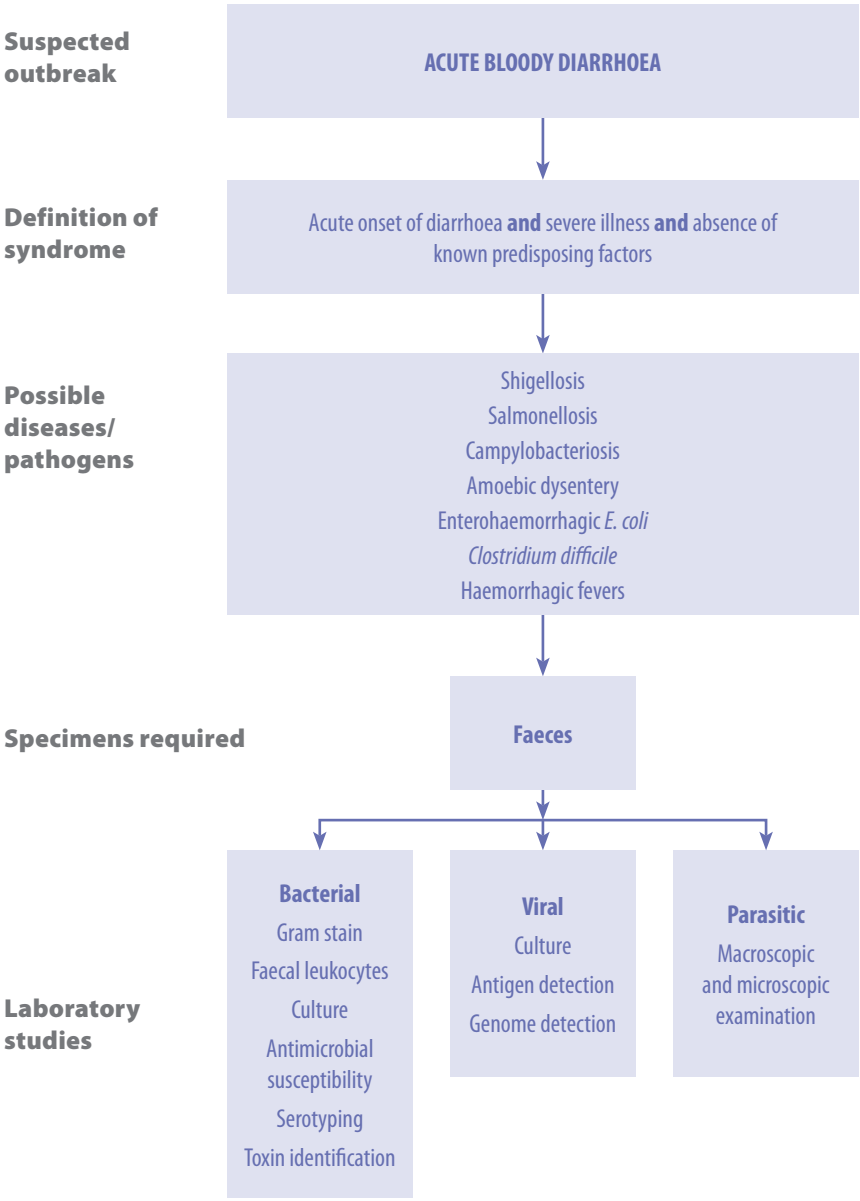
Evaluation

- 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.



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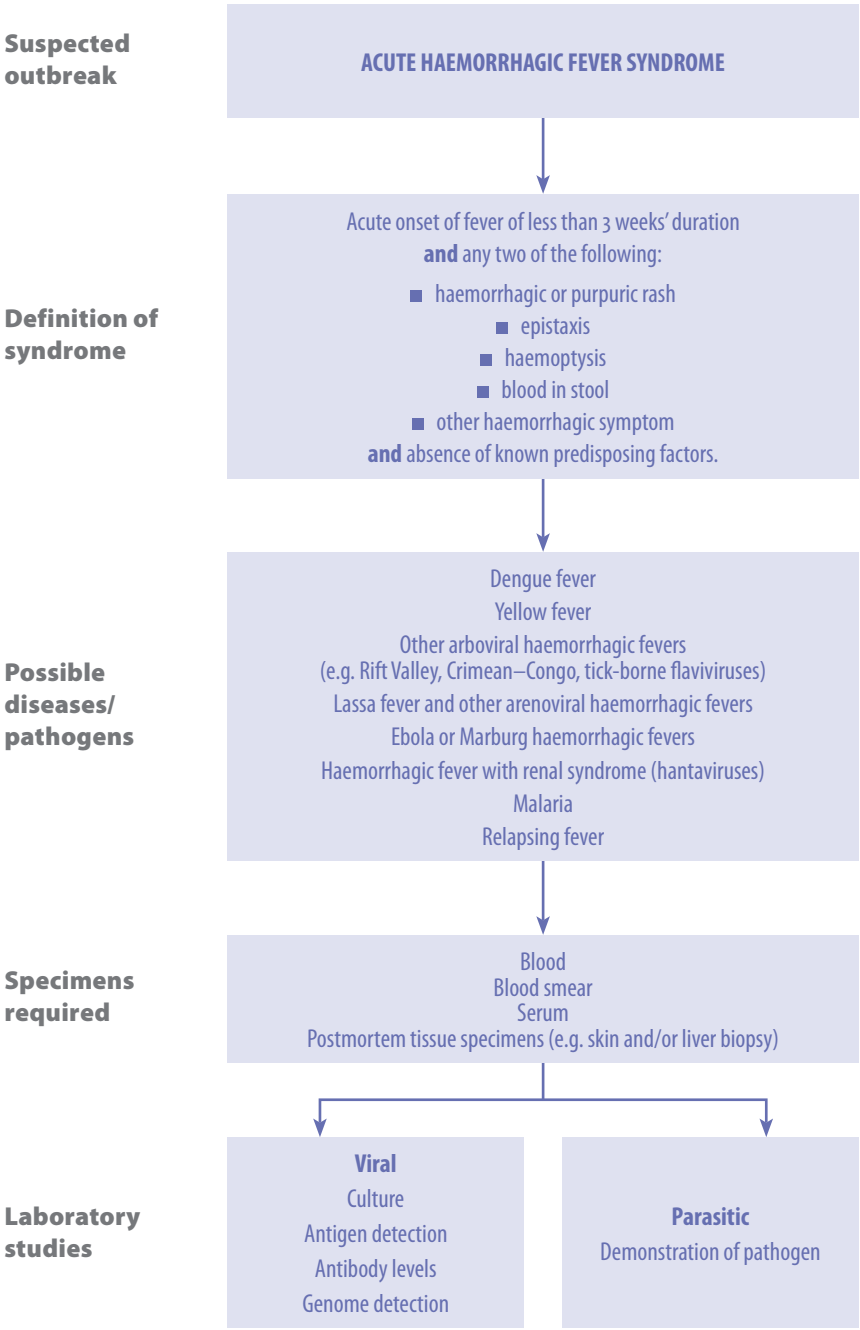
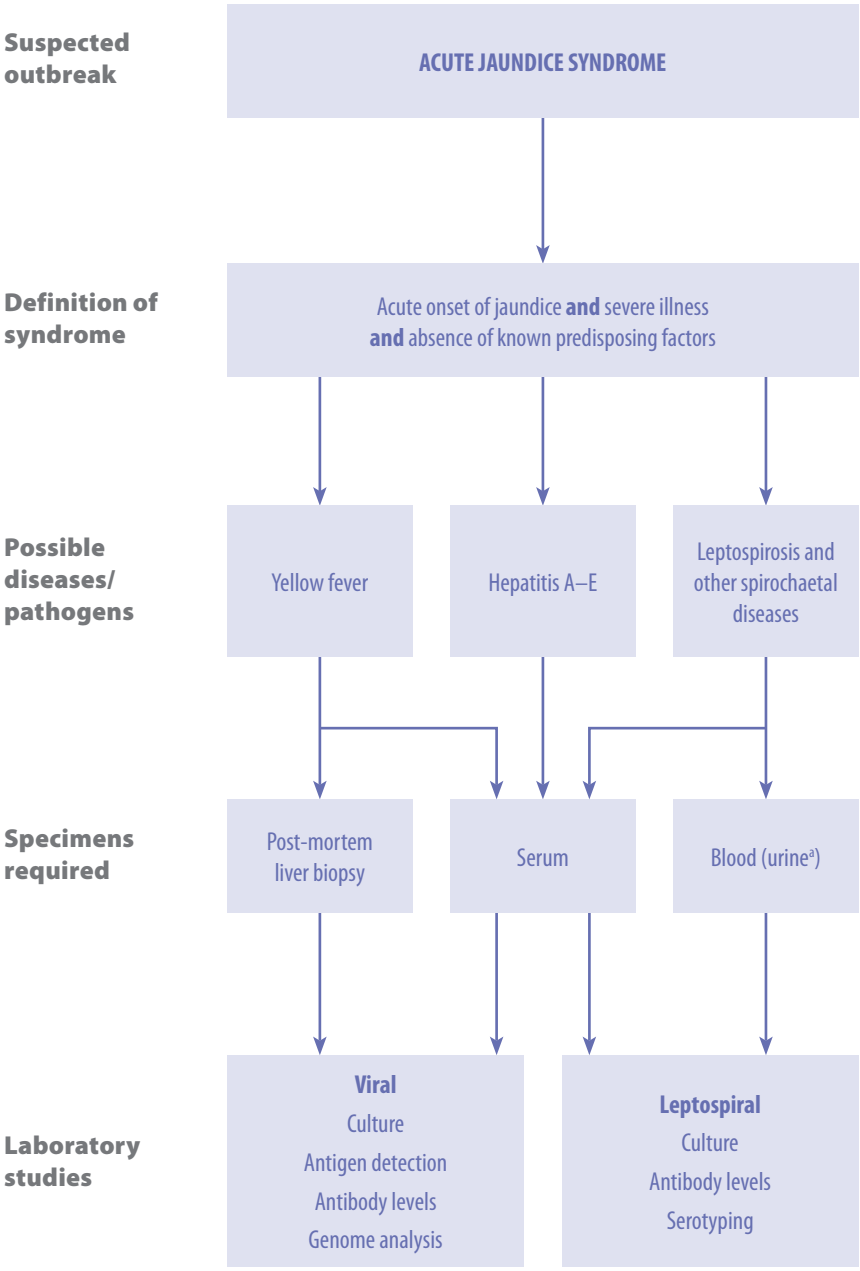
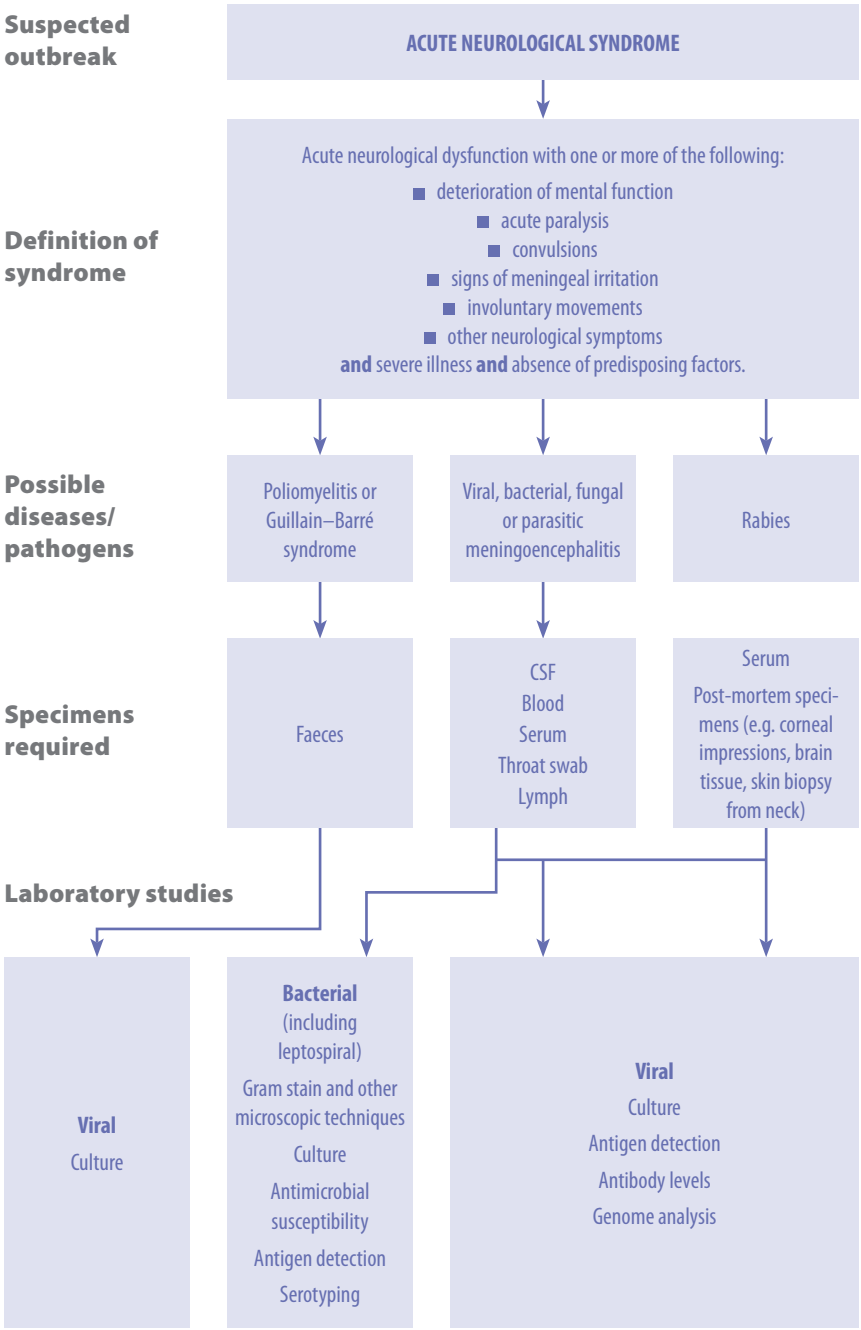
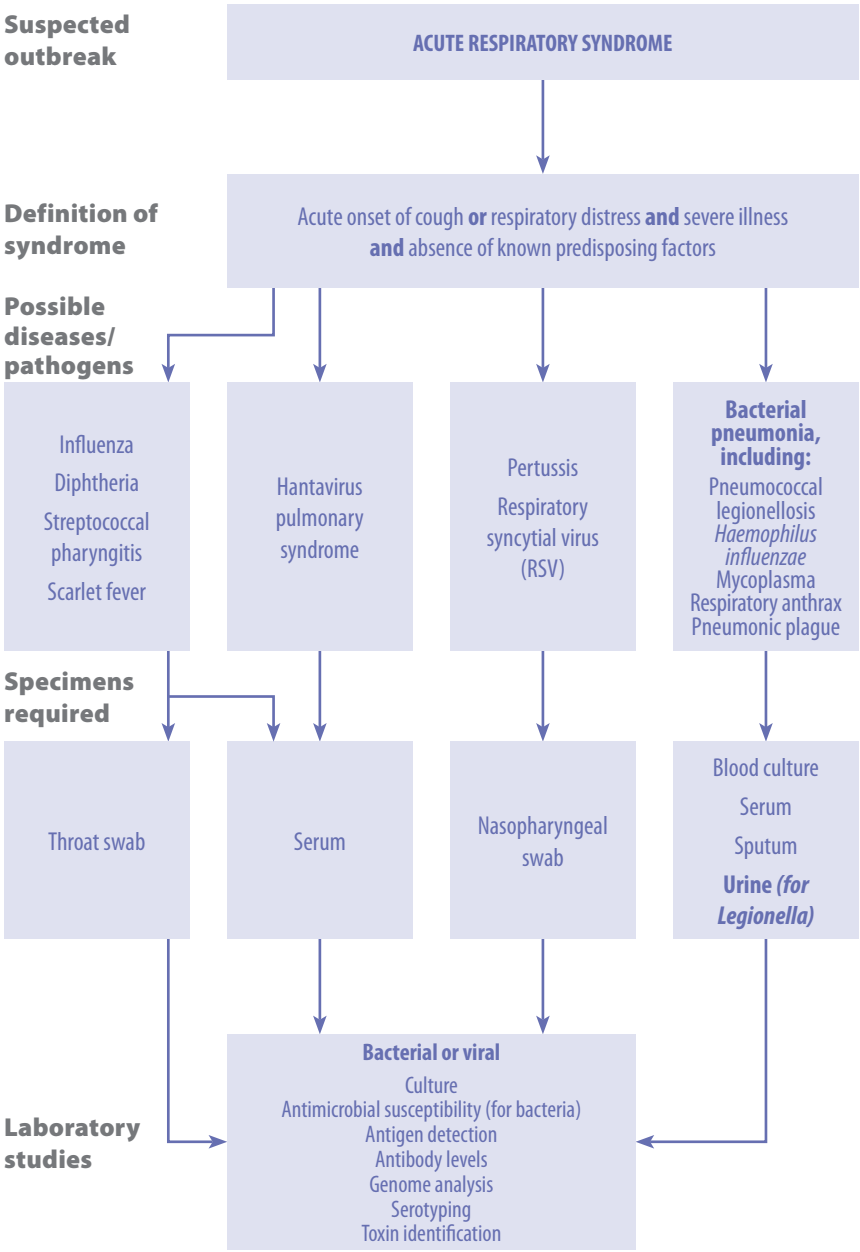

Suspected outbreak
ACUTE HAEMORRHAGIC FEVER SYNDROME
Definition of syndrome
Acute onset of fever of less than 3 weeks' duration
and any two of the following:
haemorrhagic or purpuric rash
epistaxis
haemoptysis
blood in stool
other haemorrhagic symptom
and absence of known predisposing factors.
Possible diseases/ pathogens
Dengue fever
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
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
Viral
Culture
Antigen detection
Antibody levels
Genome analysis



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

ANNEX 4. SAFE WATER, SANITATION AND HYGIENE

Safe water

The minimum emergency requirement is 20 l/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 involves adding one of the following products to one litre of water:

Product (concentration by weight of available chlorine)	Amount for 1 litre
Calcium hypochlorite (70%); or	15 g
Bleaching powder or chlorinated lime (30%); or	33 g
Sodium hypochlorite (5%); or	250 ml
Sodium hypochlorite (10%)	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 (litres)
0.6 ml or 3 drops	1
6 ml	10
60 ml	100

The chlorinated water should be mixed by stirring and allowed to stand for at least 30 minutes before use. 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, the water should be brought to a vigorous, rolling boil. This will kill, or inactivate, most of the organisms that cause diarrhoea.

Sanitation and hygiene

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. 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.

Good personal hygiene – including hand-washing after defecation and before eating or preparing food – can prevent the faecal–oral spread of communicable diseases. In emergencies, in addition to adequate quantities of water, a large-scale distribution of soap should be planned early on, with a minimum target of 250–500 g of soap per person per month, accompanied by appropriate hygiene promotion. Hygiene promotion should include latrine use; dangers of defecating on the ground, or in or near water; disposal of children's excreta in latrines; and thorough hand-washing with soap or ash after any contact with excreta.

Further reading

- Wisner B, Adams J, eds. *Environmental health in emergencies and disasters: a practical guide*. Geneva, World Health Organization (WHO), 2002 (http://www.who.int/water_sanitation_health/hygiene/emergencies/emergencies2002/en, accessed 7 October 2010).
- *Fact sheets on environmental sanitation*. Geneva, WHO, 1996 (http://www.who.int/water_sanitation_health/hygiene/emergencies/envsanfactsheets/en, accessed 7 October 2010).
- Franceys R, Pickford J, Reed R. *A guide to the development of on-site sanitation*. Geneva, WHO, 1992 (http://www.who.int/water_sanitation_health/hygiene/envsan/onsitesan/en, accessed 7 October 2010).

ANNEX 5. INJECTION SAFETY

To prevent bloodborne virus transmission through unsafe medical injection practice, all relief efforts should ensure safe and appropriate use of injections, by implementing the actions given below.

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 needlestick 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 funding.

Remember

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

ANNEX 6. WHO FACT SHEETS AND INFORMATION SOURCES

WHO Fact Sheets

Title	Publication no./date
Anthrax	Fact sheet no. 264 http://www.who.int/mediacentre/factsheets/fs264/en/
Cholera	Fact sheet no. 107 http://www.who.int/mediacentre/factsheets/fs107/en
Chikungunya	Fact sheet no. 327 http://www.who.int/mediacentre/factsheets/fs327/en
Dengue	Fact sheet no. 117 http://www.who.int/mediacentre/factsheets/fs117/en/
Diphtheria	Fact sheet no. 89 http://www.who.int/mediacentre/factsheets/fs089/en/
Ebola	Fact sheet no. 103 http://www.who.int/mediacentre/factsheets/fs103/en/
Food safety and food-borne illness	Fact sheet no. 237 http://www.who.int/mediacentre/factsheets/fs237/en/
Hepatitis B	Fact sheet no. 204 http://www.who.int/mediacentre/factsheets/fs204/en/
Hepatitis C	Fact sheet no. 164 http://www.who.int/mediacentre/factsheets/fs164/en/
Hepatitis E	Fact sheet no. 280 http://www.who.int/mediacentre/factsheets/fs280/en/index.html
Influenza	Fact sheet no. 211 http://www.who.int/mediacentre/factsheets/fs211/en/
Injection safety	Fact sheet no. 231 http://www.who.int/mediacentre/factsheets/fs231/en/
Leprosy	Fact sheet no. 101 http://www.who.int/mediacentre/factsheets/fs101/en/index.html
Leptospirosis	Fact sheet (WHO SEARO) http://www.searo.who.int/LinkFiles/CDS_leptospirosis-Fact_Sheet.pdf
Lymphatic filiariasis	Fact sheet no. 102 http://www.who.int/mediacentre/factsheets/fs102/en/index.html

Title	Publication no./date
Malaria	Fact sheet no. 94 http://www.who.int/mediacentre/factsheets/fs094/en/
Marburg haemorrhagic fever	Fact sheet http://www.who.int/mediacentre/factsheets/fs_marburg/en/index.html
Measles	Fact sheet no. 286 http://www.who.int/mediacentre/factsheets/fs286/en/
Meningitis	Fact sheet no. 141 http://www.who.int/mediacentre/factsheets/fs141/en/
Plague	Fact sheet no. 267 http://www.who.int/mediacentre/factsheets/fs267/en/
Poliomyelitis	Fact sheet no. 114 http://www.who.int/mediacentre/factsheets/fs114/en/
Rabies	Fact sheet no. 99 http://www.who.int/mediacentre/factsheets/fs099/en/
Salmonella – drug resistant	Fact sheet no. 139 http://www.who.int/mediacentre/factsheets/fs139/en/index.html
Schistosomiasis	Fact sheet no. 115 http://www.who.int/mediacentre/factsheets/fs115/en/
Smallpox	http://www.who.int/mediacentre/factsheets/smallpox/en/
Tuberculosis	Fact sheet no. 104 http://www.who.int/mediacentre/factsheets/fs104/en/ Stop TB Fact sheet http://www.who.int/tb/publications/2008/factsheet_april08.pdf
Yellow fever	Fact sheet no. 100 http://www.who.int/mediacentre/factsheets/fs100/en/

WHO information sources

Subject	Information source
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

Subject	Information source
	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
Communications during public health emergencies	Effective media communication during public health emergencies (WHO 2005) Field guide, hand book and wall chart http://www.who.int/csr/resources/publications/WHO_CDS_2005_31/en/index.html Outbreak communications – WHO Outbreak communication guidelines (WHO, 2005) http://www.who.int/infectious-disease-news/IDdocs/whocds200528/whocds200528en.pdf Outbreak communication – Best practices for communicating with the public during an outbreak. Report of the WHO Expert Consultation on Outbreak Communications held in Singapore, 21–23 September 2004 http://www.who.int/csr/resources/publications/WHO_CDS_2005_32web.pdf WHO Outbreak communication planning guide, 2008 http://www.who.int/ihr/elibrary/WHOOutbreakCommsPlanngGuide.pdf Resource centre for avian and pandemic influenza communication http://www.unicef.org/influenzaresources/ CREATE! For children – Communication resources, essentials and tools for emergencies http://keenhosting.net/unicef/
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

Subject	Information source
	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. 3rd ed. (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 Other WHO Diphtheria related links http://www.who.int/immunization/documents/immunological_basis_series/en/index.html http://www.who.int/immunization/topics/diphtheria/en/index.html http://www.who.int/topics/diphtheria/en/
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 Food safety in emergencies http://www.who.int/foodsafety/foodborne_disease/emergency/en/

Subject	Information source
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_GBVGuidelines2005.pdf 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 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

Subject	Information source
	Global influenza preparedness plan (WHO, 2005) http://www.who.int/csr/resources/publications/influenza/WHO_CDS_CSR_GIP_2005_5.pdf
Japanese encephalitis	http://www.who.int/water_sanitation_health/diseases/encephalitis/en/
Laboratory specimen collection	Guidelines for the collection of clinical specimens during field investigation of outbreaks (WHO, 2000) 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/
Leprosy	WHO resource link http://www.who.int/lep/resources/pubs/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

Subject	Information source
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/immunization_delivery/adc/measles/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 heath in emergencies http://www.who.int/mental_health/resources/emergencies/en/index.html 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
Poliomyelitis	WHO-recommended surveillance standard of poliomyelitis http://www.who.int/immunisation_monitoring/diseases/poliomyelitis_surveillance/en/index.html WHO polio vaccine position paper http://www.who.int/immunisation/wer7828polio_Jul03_position_paper.pdf
Pertussis	WHO pertussis vaccine position paper http://www.who.int/immunisation/topics/wer8004pertussis_Jan_2005.pdf
Rabies	Current strategies for human rabies pre and post-exposure prophylaxis http://www.who.int/rabies/human/WHO_strategy_prepost_exposure/en/print.html Rabies vaccines: WHO position paper http://www.who.int/wer/2010/wer8532.pdf

Subject	Information source
Social mobilization / COMBI	Mobilizing for action, WHO Mediterranean Centre for Vulnerability Reduction, Tunis 2004, http://wmc.who.int/images/stories/pdf/Mobilizing_for_Action.pdf Planning COMBI programmes for health: COMBI Manual http://wmc.who.int/images/stories/pdf/combimanualVerCD.pdf Planning social mobilization and communication for dengue fever prevention and control http://apps.who.int/tdr/publications/training-guideline-publications/planning-social-mobilization-dengue-fever/pdf/planning_dengue.pdf WHO Mediterranean Centre for Vulnerability Reduction/COMBI programme website: http://wmc.who.int/index.php?option=content&task=view&id=43 Essentials for excellence: a research, monitoring and evaluation tool UNICEF, 2006). http://www.unicef.org/influenzaresources/files/EforE_RME_Guidelines_Dec_2006(1).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 (WHO, 2001) http://www.who.int/csr/resources/publications/surveillance/whocdscsrisr20012.pdf WHO report on global surveillance of epidemic-prone infectious diseases (WHO, 2000) http://www.who.int/csr/resources/publications/surveillance/WHO_CDS_CSR_ISR_2000_1/en/
Tetanus; Maternal and neonatal tetanus	Tetanus general information http://www.who.int/topics/tetanus/en/ 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 immunization 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 (WHO, 2008) http://www.who.int/ith/en/

Subject	Information source
Typhoid (see also diarrhoeal diseases)	Background document: the diagnosis, treatment, and prevention of typhoid fever (WHO, 2003) http://whqlibdoc.who.int/hq/2003/WHO_V&B_03.07.pdf Water-related diseases http://www.who.int/water_sanitation_health/diseases/typhoid/en/
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/immunisation/en/ WHO vaccine-preventable diseases: monitoring system – 2007 global summary http://www.who.int/immunisation/documents/WHO_IVB_2007/en/index.html Immunisation against diseases of public health importance http://www.who.int/immunisation_delivery/en/index.html Linking vaccines with other interventions http://www.who.int/immunisation_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/immunisation/documents/positionpapers/en/index.html WHO documents on vaccines and immunisation: http://www.who.int/immunisation/documents/en/ http://www.who.int/vaccines-documents/ WHO immunological basis of immunisation series http://www.who.int/immunisation/documents/general/en/index.html WHO-recommended standards for surveillance of selected vaccine preventable diseases (WHO/V&B/03.01) http://www.who.int/immunisation/documents/WHO_VB_03.01/en/index.html
Vector control	Integrated vector management http://www.who.int/malaria/vector_control/ivm/en/index.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

Subject	Information source
Water, sanitation and health	Guidelines for drinking-water quality, 3rd ed., 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 Fact sheets on environmental sanitation http://www.who.int/water_sanitation_health/hygiene/emergencies/envsanfactsheets/en/print.html
World Health Organization	About WHO http://www.who.int/about/en/
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 South-East Asia (SEARO)	http://www.searo.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 Global Alert and Response (GAR)	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/

ANNEX 7. WHO CONTACTS

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Switzerland
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