**Infectious Diseases (Complex Infections: HIV, Tropical Diseases)**

**Infectious diseases** (**ID**), also known as **infectiology**, is a [medical specialty](https://en.wikipedia.org/wiki/Medical_specialty) dealing with the diagnosis and treatment of infections. An infectious diseases specialist's practice consists of managing nosocomial ([healthcare-acquired](https://en.wikipedia.org/wiki/Hospital-acquired_infection)) infections or community-acquired infections.[[1]](https://en.wikipedia.org/wiki/Infectious_diseases_(medical_specialty)#cite_note-1) An ID specialist investigates and determines the cause of a disease (bacteria, virus, parasite, fungus or [prions](https://en.wikipedia.org/wiki/Prion)). Once the cause is known, an ID specialist can then run various tests to determine the best drug to treat the disease.[[2]](https://en.wikipedia.org/wiki/Infectious_diseases_(medical_specialty)#cite_note-:13-2) While infectious diseases have always been around, the infectious disease specialty did not exist until the late 1900s after scientists and physicians in the 19th century paved the way with research on the sources of infectious disease and the development of vaccines.

**INTRODUCTION**

Infectious diseases are illnesses caused by pathogenic microorganisms—such as bacteria, viruses, parasites, or fungi—that invade the body, multiply, and interfere with normal body functions. They can be acute or chronic, and simple or complex, depending on the pathogen involved, the host’s immune response, and treatment availability.

A complex infectious disease refers to an infection that:

* Has multiple stages or long latency periods
* Interacts with host immunity in complicated ways
* Can involve co-infections or opportunistic infections
* May be resistant to standard treatments
* Often needs multidisciplinary care

The landscape of infectious diseases has evolved dramatically in recent decades, with complex interactions between pathogens, host immune responses, and environmental factors driving global health crises. Tropical diseases exemplify this complexity, where biological synergies,socioeconomic disparities, and ecological disruptions amplify morbidity and mortality. Emerging research underscores the critical need for integrated frameworks that address not only pathogen biology but also the structural determinants of disease transmission and outcomes[.](https://pubmed.ncbi.nlm.nih.gov/15567014/) This report examines the pathogenesis, syndemic interactions, and multidisciplinary strategies required to mitigate these challenges.

# **What Is the Role of an Infectious Disease Specialist?**

Since the 1970s, specialists in infectious (also known as communicable) diseases have been researching pathogens, diagnosing diseases, and administering newly developed medications and vaccines. An infectious disease specialist, also known as an infectious disease doctor, is a physician who specializes in both chronic and acute diseases. Prospective specialists should learn more about the role and its responsibilities, [education requirements](https://publichealth.tulane.edu/online-mph/), and career outlook to determine if this career track is right for them.

## **The Role of an Infectious Disease Specialist**

Infectious disease specialists investigate and diagnose diseases caused by infectious microorganisms, from bacteria and viruses to fungi and protozoa. While their exact responsibilities vary, infectious disease specialists may diagnose and treat patients with the following conditions:

* Bone infections
* Pneumonia
* HIV
* Viral hepatitis
* Lyme disease
* Streptococcal infections

These are just some examples of the known diseases these specialists research and treat. Many practitioners focus on researching current and emerging diseases. Like other specialists, most infectious disease doctors work with a team of healthcare professionals. A primary care physician or specialist in a related area refers most patients to them.

## **Responsibilities of Infectious Disease Specialists**

Because they are specialized professionals, infectious disease specialists can work in both clinical and research capacities. Here are a few typical responsibilities of these specialists:

* Diagnosing acute and chronic diseases
* Managing treatment plans for patients
* Researching communicable diseases
* Tracking population data and community health statistics
* Applying research and expertise to public health
* Communicating disease prevention strategies
* Recommending public health initiatives based on research

Those who work in communicable diseases and public health may still work with individual patients. Still, they typically specialize in tracking community data, researching new strains of diseases, and providing education to improve public health initiatives.

### **Work Environment**

Some infectious disease specialists work primarily in clinical settings. These professionals treat and monitor patients with communicable diseases, whether in hospitals or specialist clinical facilities. They will likely have more face-to-face interaction with patients and other healthcare professionals.

On the other hand, infectious disease doctors specializing in public health typically work in lab and research settings. These professionals actively research new strains of diseases, track population data, and communicate findings with the scientific community and the greater population.

**Scope**

Infectious diseases specialists typically serve as consultants to other physicians in cases of complex infections, and often manage patients with [HIV/AIDS](https://en.wikipedia.org/wiki/HIV/AIDS) and other forms of immunodeficiency.[[6]](https://en.wikipedia.org/wiki/Infectious_diseases_(medical_specialty)#cite_note-IDSA_What_is-6)[[7]](https://en.wikipedia.org/wiki/Infectious_diseases_(medical_specialty)#cite_note-7) Although many common infections are treated by physicians without formal expertise in infectious diseases, specialists may be consulted for cases where an infection is difficult to diagnose or manage. They may also be asked to help determine the cause of a fever of unknown origin.[[6]](https://en.wikipedia.org/wiki/Infectious_diseases_(medical_specialty)#cite_note-IDSA_What_is-6)[[8]](https://en.wikipedia.org/wiki/Infectious_diseases_(medical_specialty)#cite_note-8)

Specialists in infectious diseases can practice both in hospitals (inpatient) and clinics (outpatient). In hospitals, specialists in infectious diseases help ensure the timely diagnosis and treatment of acute infections by recommending the appropriate diagnostic tests to identify the source of the infection and by recommending appropriate management such as prescribing antibiotics to treat bacterial infections. For certain types of infections, involvement of specialists in infectious diseases may improve patient outcomes.[[9]](https://en.wikipedia.org/wiki/Infectious_diseases_(medical_specialty)#cite_note-9) In clinics, specialists in infectious diseases can provide long-term care to patients with chronic infections such as HIV/AIDS.[[*citation needed*](https://en.wikipedia.org/wiki/Wikipedia:Citation_needed)]

**History**

*See also:* [*1854 Broad Street cholera outbreak*](https://en.wikipedia.org/wiki/1854_Broad_Street_cholera_outbreak)

Infectious diseases are historically associated with [hygiene](https://en.wikipedia.org/wiki/Hygiene) and [epidemiology](https://en.wikipedia.org/wiki/Epidemiology) due to periodic outbreaks ravaging countries, especially in the cities before the advent of [sanitation](https://en.wikipedia.org/wiki/Sanitation), but also with [travel medicine](https://en.wikipedia.org/wiki/Travel_medicine) and [tropical medicine](https://en.wikipedia.org/wiki/Tropical_medicine), as many diseases acquired in tropical and subtropical areas are infectious in nature.[[10]](https://en.wikipedia.org/wiki/Infectious_diseases_(medical_specialty)#cite_note-10)

Western innovations for treating infectious diseases originated in Ancient Greece, and before infectious disease was even conceptualized, a Greek Physician named Hippocrates formed the [Hippocratic Corpus](https://en.wikipedia.org/wiki/Hippocratic_Corpus). Included in this collection of 70 documents was a text that contained illness-causing infectious diseases. This text, called the Epidemiai volumes, played a key role in forming the western approach to infectious disease. A physician during the Roman empire, [Galen of Pergamon](https://en.wikipedia.org/wiki/Galen_of_Pergamon), also made great impacts on the western perception of infectious disease with his multiple treatises.[[4]](https://en.wikipedia.org/wiki/Infectious_diseases_(medical_specialty)#cite_note-:02-4) These treatises gave insight into the [Antonine Plague](https://en.wikipedia.org/wiki/Antonine_Plague) which we now recognize as smallpox based on the description in Galen's treatises.[[11]](https://en.wikipedia.org/wiki/Infectious_diseases_(medical_specialty)#cite_note-11)

**Between the 16th and 18th centuries**, medical professionals were educating more people, learning more from their research, and gaining access to information from other professionals in the field due to the use of printers like Gutenberg and the mass production of medical books. These books, now in the hands of many, included observations of infectious diseases. Such as syphilis, malaria, and smallpox. In the late 18th century we start to see vaccinations forming and the first vaccination for smallpox was established. Although there were records of individual infectious diseases spread out over medical documents, a combined perception of infectious disease as an area of medicine did not exist at that time.

**During the 19th century**, modern medicine began to develop and the sources of infectious diseases became more clear. [Robert Koch](https://en.wikipedia.org/wiki/Robert_Koch), a German physician who studied pathogens, discovered three major pathogens that were the cause of Anthrax, Tuberculosis, and Cholera.[[4]](https://en.wikipedia.org/wiki/Infectious_diseases_(medical_specialty)#cite_note-:02-4) [Louis Pasteur](https://en.wikipedia.org/wiki/Louis_Pasteur) was a pioneer in the creation of vaccines for infectious diseases, one being a vaccine for Anthrax. He also developed the [germ theory](https://en.wikipedia.org/wiki/Germ_theory) of infectious diseases which influenced [Joseph Lister](https://en.wikipedia.org/wiki/Joseph_Lister) to practice methods during surgery that reduce the growth of pathogens that cause infectious disease.[[5]](https://en.wikipedia.org/wiki/Infectious_diseases_(medical_specialty)#cite_note-:32-5) Although infectious disease started to become a more collective concept in the 19th-century it was not considered a medical specialty until the 1970s due to a number of newly discovered diseases and vaccines.[[3]](https://en.wikipedia.org/wiki/Infectious_diseases_(medical_specialty)#cite_note-:22-3)

**Investigations**

When diagnosing, a medical professional must first determine if a patient has an infectious disease or another condition not caused by infection but exhibits similar symptoms. Once the illness is confirmed to be caused by an infection, Infectious diseases specialists employ a variety of diagnostic tests to help identify the pathogen that is causing an infection. Common tests include [staining](https://en.wikipedia.org/wiki/Staining), [culture tests](https://en.wikipedia.org/wiki/Blood_culture), [serological tests](https://en.wikipedia.org/wiki/Serological_test), [susceptibility tests](https://en.wikipedia.org/wiki/Serology), [genotyping](https://en.wikipedia.org/wiki/Genotyping), [nucleic acid-base test](https://en.wikipedia.org/wiki/Genotyping), and [polymerase chain reaction](https://en.wikipedia.org/wiki/Polymerase_chain_reaction). Seeing as samples of bodily fluid or tissue are used in these tests, a specialist will have to distinguish between the non-disease-causing bacteria and disease-causing bacteria inhabiting the body to effectively identify and treat the infection.[[2]](https://en.wikipedia.org/wiki/Infectious_diseases_(medical_specialty)#cite_note-:13-2)

**Staining** is a method of testing that uses a special dye to change the color of pathogens and a microscope to view them. The change in color helps doctors distinguish the pathogen from its surrounding and identify what it is. This method is only successful with large and plentiful pathogens present. Therefore, this method is unsuccessful with viruses because they can not be viewed under a microscope due to their small size. Staining has more of an effect on bacteria where a violet colored stain is used, this is called [gram staining](https://en.wikipedia.org/wiki/Gram_stain). If the bacteria appears blue it is considered gram positive and if it appears red it is gram negative.[[2]](https://en.wikipedia.org/wiki/Infectious_diseases_(medical_specialty)#cite_note-:13-2)

**Culture tests** are done when there is not enough of the pathogen to be seen through other tests. ID specialists will grow the pathogen in the lab until they have enough to work with. Although cultures work on some pathogens, such as the bacteria that causes strep throat, it is ineffective on many others, such as syphilis. A test to identify the pathogen, such as staining, would take place after culture tests.

**Susceptibility tests** are done by ID specialists to discover which antimicrobial drug would be most effective at killing the pathogen. Cultures can also be used as a form of susceptibility testing by adding the drug to the cultured pathogens and observing whether or not it kills the pathogen and how much of the drug is needed to kill it.

**Nucleic acid-base tests** are used to detect genetic material. For pathogens that can't be cultured, ID specialists can identify them by looking for specific DNA or RNA. **Polymerase chain reaction (PCR)**, a type of nucleic acid-base test, is similar to culture tests in that genes from the pathogen are duplicated. This method is mainly used when a specific pathogen is suspected.

**Treatments**

Infectious diseases specialists employ a variety of antimicrobial agents to help treat infections. The type of antimicrobial depends on the organism that is causing the infection. Antibiotics are used to treat bacterial infections; antiviral agents treat viral infections; and antifungal agents treat fungal infections.

# **Infectious Disease Doctor**

Infectious disease doctors are healthcare providers who specialize in diagnosing and treating conditions caused by bacteria, parasites, viruses and fungi. In most cases, another healthcare provider refers you to see an infectious disease doctor.

### **What is an infectious disease doctor?**

An infectious disease (ID) doctor or infectious disease specialist is a physician who specializes in [infectious diseases](https://my.clevelandclinic.org/health/diseases/17724-infectious-diseases).

Infectious diseases are illnesses caused by harmful organisms that get into your body. The most common causes of infectious diseases are viruses, bacteria, fungi and parasites. These organisms are everywhere. Most of the time, we coexist with some of them without them ever causing a problem. But they can also cause diseases that range from mild to deadly.

Infectious diseases usually spread from person to person, through contaminated food, water and soil, or through insect and animal bites. Infectious diseases can affect your skin, [urinary tract](https://my.clevelandclinic.org/health/diseases/9135-urinary-tract-infections), [lungs](https://my.clevelandclinic.org/health/body/8960-lungs), blood and virtually any area of your body.

Infectious diseases are extremely common worldwide, but some are more common than others. The [flu](https://my.clevelandclinic.org/health/diseases/4335-influenza-flu), measles, [common cold](https://my.clevelandclinic.org/health/diseases/12342-common-cold), [strep throat](https://my.clevelandclinic.org/health/diseases/4602-strep-throat), [COVID-19](https://my.clevelandclinic.org/health/diseases/21214-coronavirus-covid-19) and [*salmonella*](https://my.clevelandclinic.org/health/diseases/15697-salmonella) are all examples of infectious diseases.

You don’t need to see an infectious disease doctor anytime you have a virus or infection. Most healthcare providers can treat common infections and viruses. An infectious disease doctor is an expert in diagnosing, managing and treating rare, complex, serious or chronic infections. In some ways, they’re like detectives of organisms in the human body, considering tiny details of a person’s medical history or laboratory results to try to understand and control an infectious disease.

#### **What does an infectious disease doctor do?**

An infectious disease doctor is an expert in diagnosing, managing and treating acute (sudden) and chronic (present for a long time) diseases caused by bacteria, viruses, fungi, parasites and prions. They often work alongside other physicians and specialists to diagnose and treat conditions or determine the cause of a specific symptom. They spend many hours conducting research on how organisms affect different parts of your body and how these organisms can affect our society as a whole.

They also work to understand:

* New or emerging infections or infections that change over time.
* Infections that spread mainly due to international travel.
* [Antibiotics](https://my.clevelandclinic.org/health/drugs/16386-antibiotics), [antivirals](https://my.clevelandclinic.org/health/drugs/21531-antivirals) and [vaccinations](https://my.clevelandclinic.org/health/treatments/24135-vaccines).

### **Why would you see an infectious disease doctor?**

An infectious disease doctor works with many other types of physicians and specialists. Most of the time, but not always, a [primary care physician (PCP)](https://my.clevelandclinic.org/health/articles/23467-primary-care-physician) or another specialist refers you to an infectious disease doctor. It’s typically because they need additional help diagnosing and treating an infection or virus because it’s uncommon, severe, chronic or unknown. They may refer you to an ID doctor to rule out certain infections as the cause of a medical condition.

For example, a [dermatologist](https://my.clevelandclinic.org/health/articles/12165-dermatologists-skin-care-doctors) may ask an infectious disease doctor for help with rare skin conditions and a [pulmonologist](https://my.clevelandclinic.org/health/articles/22210-pulmonologist) may consult with an infectious disease specialist if a person has chronic or untreatable pneumonia.

Some of the ways infectious disease specialists help other healthcare providers are by:

* Diagnosing rare or complex infections or viruses.
* Identifying an organism causing a person’s symptoms.
* Interpreting laboratory tests like blood work.
* Treating an infection due to [antibiotic resistance](https://my.clevelandclinic.org/health/articles/21655-antibiotic-resistance) (antibiotics aren’t working).
* Pinpointing reasons for symptoms like unexplained high fevers or [high white blood cell count](https://my.clevelandclinic.org/health/diagnostics/17704-high-white-blood-cell-count).
* Providing long-term, specialized care for chronic infections like HIV/AIDS or hepatitis.
* Consulting on guidelines for antibiotics and vaccines or deciding what types of disinfectants or PPE (personal protective equipment) to use to minimize infections in healthcare settings.

### **What conditions do infectious disease doctors treat?**

There are several dozen infectious diseases in the world. Some of the more common conditions that an infectious disease doctor would treat include:

* [*Clostridium difficile (C. diff)*](https://my.clevelandclinic.org/health/diseases/15548-c-diff-infection).
* [*E. coli*](https://my.clevelandclinic.org/health/diseases/16638-e-coli-infection).
* [Fevers](https://my.clevelandclinic.org/health/symptoms/10880-fever).
* [Hepatitis](https://my.clevelandclinic.org/health/diseases/4245-hepatitis-viral-hepatitis-a-b--c).
* [HIV/AIDS](https://my.clevelandclinic.org/health/diseases/4251-hiv-aids).
* [Leukocytosis](https://my.clevelandclinic.org/health/diagnostics/17704-high-white-blood-cell-count) (high white blood cell count).
* [Lyme disease and other tick-borne diseases](https://my.clevelandclinic.org/health/diseases/11586-lyme-disease).
* [Measles](https://my.clevelandclinic.org/health/diseases/8584-measles).
* [Meningococcal disease](https://my.clevelandclinic.org/health/diseases/22442-meningococcal-disease).
* [Mpox](https://my.clevelandclinic.org/health/diseases/22371-monkeypox).
* [Mumps](https://my.clevelandclinic.org/health/diseases/15007-mumps).
* [Rubella](https://my.clevelandclinic.org/health/diseases/17798-rubella).
* [Sexually transmitted infections (STIs)](https://my.clevelandclinic.org/health/diseases/9138-sexually-transmitted-diseases--infections-stds--stis).
* [Streptococcal infections](https://my.clevelandclinic.org/health/articles/5911-group-a-streptococcal-infections).
* [Tuberculosis (TB)](https://my.clevelandclinic.org/health/diseases/11301-tuberculosis).
* [Whooping cough (pertussis)](https://my.clevelandclinic.org/health/diseases/15661-whooping-cough-pertussis).

### **What should I expect from an appointment with an infectious disease doctor?**

Infectious disease doctors perform [physical exams](https://my.clevelandclinic.org/health/diagnostics/17366-physical-examination) and take your full medical history. They’ll ask you lots of questions about your symptoms, medications and your environment. Environmental questions could include things like what pets you have, if you traveled out of the country or if you spend a lot of time outdoors or around chemicals.

Then, they’ll likely order some of the following tests to help diagnose a condition:

* [Blood tests](https://my.clevelandclinic.org/health/diagnostics/24508-blood-tests).
* [Urine tests](https://my.clevelandclinic.org/health/diagnostics/17893-urinalysis).
* Stool (poop) samples.
* Throat swabs.
* [Tissue biopsies](https://my.clevelandclinic.org/health/diagnostics/21857-skin-biopsy).
* Imaging scans like [X-Ray](https://my.clevelandclinic.org/health/diagnostics/21818-x-ray), [MRI](https://my.clevelandclinic.org/health/diagnostics/4876-magnetic-resonance-imaging-mri), [CT scans](https://my.clevelandclinic.org/health/diagnostics/4808-ct-computed-tomography-scan), etc.
* [Spinal taps (lumbar puncture)](https://my.clevelandclinic.org/health/diagnostics/12544-lumbar-puncture-spinal-tap).

### **How do you become an infectious disease doctor?**

Infectious disease doctors have a similar medical background as other doctors. But they spend many additional years understanding and learning about immunology and epidemiology. Immunology is the study of a person’s [immune system](https://my.clevelandclinic.org/health/articles/21196-immune-system) and how organisms affect it. Epidemiology is a science that investigates factors that determine why diseases and disorders exist or don’t exist.

Infectious disease specialists have extensive knowledge of how and why viruses, parasites, bacteria and fungi affect your body.

This requires years of training. Infectious disease doctors complete the following:

* Undergraduate degree.
* Medical school (four years).
* Residency in internal medicine (three years).
* Fellowship in infectious diseases (usually two years).
* Specialization in a specific area like transplant infectious disease (optional one-year program).
* Board certification.

#### **Where do infectious disease doctors work?**

Some infectious disease doctors focus on research and may spend time working in public health, such as for the Centers for Disease Control and Prevention (CDC). Other infectious disease specialists are more clinical, which means they work in a hospital or community practice and visit patients regularly.

### **A note**

Your primary care physician (PCP) may refer you to an infectious disease specialist if you have a specific infectious disease that may be challenging to diagnose or treat. Infectious disease doctors are experts in infections that happen due to bacteria, viruses, fungi or parasites. They work with your PCP and other healthcare providers to manage your condition and get you feeling well again.

# **Tropical Disease**

**tropical disease**, any [disease](https://www.britannica.com/science/disease) that is [indigenous](https://www.merriam-webster.com/dictionary/indigenous) to tropical or subtropical areas of the world or that occurs principally in those areas. Examples of tropical diseases include [malaria](https://www.britannica.com/science/malaria), cholera, Chagas disease, [yellow fever](https://www.britannica.com/science/yellow-fever), and dengue.

## **Historical overview of tropical diseases**

Diseases of the tropics and subtropics have been known since ancient times. For example, ancient physicians, including Greek physician [Hippocrates](https://www.britannica.com/biography/Hippocrates) and Roman medical writer [Aulus Cornelius Celsus](https://www.britannica.com/biography/Aulus-Cornelius-Celsus), wrote about malarial diseases, and modern molecular analyses of Egyptian mummies have suggested that [malaria](https://www.britannica.com/science/malaria) was present in [ancient Egypt](https://www.britannica.com/place/ancient-Egypt). Other tropical diseases were recognized later. For example, after the Spanish conquest in the 16th century, Europeans discovered [yellow fever](https://www.britannica.com/science/yellow-fever), a disease present in tropical [Africa](https://www.britannica.com/place/Africa) and [South America](https://www.britannica.com/place/South-America).

Scientific interest in the identification and [classification](https://www.britannica.com/dictionary/classification) of tropical diseases emerged in the 19th century, when increasing numbers of Europeans and Americans, as a result of exploration and colonial expansion, were brought into contact with infectious diseases in tropical and subtropical climates. The study of tropical diseases formed the basis of [tropical medicine](https://www.britannica.com/science/tropical-medicine). Among the first diseases to be investigated were [filariasis](https://www.britannica.com/science/filariasis), malaria, and yellow fever. In the late 19th and early 20th centuries, many tropical diseases were found to be transmitted by vectors, such as [mosquitoes](https://www.britannica.com/animal/mosquito-insect), [fleas](https://www.britannica.com/animal/flea), [lice](https://www.britannica.com/animal/louse), [snails](https://www.britannica.com/animal/snail), and other animals, and some diseases were linked to contaminated food or water. Eventually, the pathogens (disease-causing organisms) for many tropical diseases were identified; they include [bacteria](https://www.britannica.com/science/bacteria), [viruses](https://www.britannica.com/science/virus), and [parasites](https://www.britannica.com/science/parasitism).

In the late 20th and early 21st centuries, the significance of tropical diseases grew. Whereas some diseases had been largely controlled through improved awareness and advances in prevention and treatment, others increased in [incidence](https://www.britannica.com/science/incidence-epidemiology) as a result of [population growth](https://www.britannica.com/science/population-growth), large-scale [human migration](https://www.britannica.com/topic/human-migration) and displacement, the deterioration of [public health](https://www.britannica.com/topic/public-health) [infrastructure](https://www.merriam-webster.com/dictionary/infrastructure), and [tourism](https://www.britannica.com/topic/tourism). In addition, some tropical diseases that had been largely controlled, such as [cholera](https://www.britannica.com/science/cholera), [dengue](https://www.britannica.com/science/dengue), and meningococcal meningitis, reemerged. And new diseases, such as [Ebola](https://www.britannica.com/science/Ebola), appeared. Some tropical diseases began to spread into temperate climates as a result of increased human travel and climate-driven migration of vectors. The impact of a large number of tropical diseases was influenced by factors such as [poverty](https://www.britannica.com/topic/poverty), lack of clean water, and lack of medical care.

## **Neglected tropical diseases**

Numerous tropical diseases have been described, and they collectively affect hundreds of millions of people worldwide each year. However, while many tropical diseases have been eliminated from more-developed countries, some of those diseases have remained major sources of illness and mortality in poor, [marginalized](https://www.merriam-webster.com/dictionary/marginalized), and rural regions. Those diseases, known as neglected tropical diseases, affect roughly one billion people globally. Examples of neglected tropical diseases include [African sleeping sickness](https://www.britannica.com/science/sleeping-sickness), [Chagas disease](https://www.britannica.com/science/Chagas-disease), dengue, [guinea worm disease](https://www.britannica.com/science/guinea-worm-disease), [leishmaniasis](https://www.britannica.com/science/leishmaniasis), [leprosy](https://www.britannica.com/science/leprosy), lymphatic filariasis, [onchocerciasis](https://www.britannica.com/science/onchocerciasis), [rabies](https://www.britannica.com/science/rabies), [schistosomiasis](https://www.britannica.com/science/schistosomiasis), [trachoma](https://www.britannica.com/science/trachoma), and [yaws](https://www.britannica.com/science/yaws).

Efforts to prevent and control neglected tropical diseases have been challenged by the limited international visibility of the diseases, by the significant economic and social problems that face afflicted regions, by a lack of medical access in those regions, and by a lack of local education about the diseases. In the early 21st century, however, increased international attention led to improved research funding and accessibility to medical care in some affected areas. Although drugs were available for only a few neglected tropical diseases, so-called mass drug administration, in which drugs were made available to large numbers of people, and other interventions, such as [vector](https://www.britannica.com/dictionary/vector) control and sanitation and [hygiene](https://www.britannica.com/science/hygiene) improvements, proved highly effective against the diseases.

## Key Points.

* •
* The term tropical diseases encompasses all communicable and non-communicable diseases that occur principally in the tropics.
* •
* Approximately 15 million people die each year because of tropical infectious and parasitic diseases.
* •
* Tropical diseases are not restricted to the tropics. Increasing migration, international air travel, tourism, and work visits to tropical regions have contributed to an increased incidence of such diseases being seen in the United States, United Kingdom, and Europe.
* •
* Classification of tropical diseases is useful for microbiologists, pathologists, laboratory staff and practicing infectious diseases physicians.
* •
* This article gives an overview of the definition, geographical distribution, transmission and practical classification of tropical infectious diseases.

## Classification of tropical diseases

The number and range of tropical and infectious diseases prevalent globally is extremely large and broad ranging.[1](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/#bib1), [2](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/#bib2), [3](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/#bib3) Thus, for practical purposes, specific listings and classifications are useful for streamlining the microbiological and clinical assessment of the patient's illness. Classification of tropical diseases can also serve as aide-mémoires or checklists for guiding clinicians, microbiologists, pathologists, and laboratory staff. For the practicing infectious diseases physician, there are several ways in which tropical/infectious diseases are presented in century-old classic tropical diseases textbooks like *Manson's Tropical Diseases* or other major treatises that present the classification of tropical diseases with a combination of clinical and microbiological approaches. The classification of infectious and tropical diseases, and their treatment, control, and prevention, have historically involved the joint efforts of epidemiologists, microbiologists, and clinicians.

[Table 1](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/#tbl1) gives a basic classification of common infectious pathogens for clinical use. Physicians also tend to classify infectious diseases according to the most important organ or organ system to be affected, or the important clinical manifestations of the specific disease ([Table 2](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/#tbl2) ).[13](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/#bib13), [14](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/#bib14) Microbiologists tend to prefer classifying infectious diseases according to the classic microbiological nomenclature codes of kingdom, phylum, class, order, family, genus, and species and have large standard textbooks that give detailed classification and nomenclature.[15](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/#bib15) They relate information according to microscopic appearance after staining or culture characteristics, to advise the clinician on the most appropriate antibiotic therapy and management. However, with advances in molecular technology, microorganisms are frequently being reclassified and renamed. For example *Rickettsia tsutsugamushi*, the causal agent for scrub typhus, has been reclassified into the genus *Orientia*. DF-2 is now known as *Capnocytophaga canimorsus*.[16](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/#bib16) Epidemiologists usually describe tropical disease in terms of person, place, time, and exposure, with a view to developing control and prevention strategies to limit the spread of the diseases in the community. They often classify infectious diseases according to their distribution, their means of transmission, and according to their reservoirs in nature. Such classifications use the routes of transmission or acquisition of the infectious disease ([Table 3](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/#tbl3) ).

### Table 1.

Basic microbiological classification of common infectious pathogens for clinicians

| **Microbiological or Clinical Grouping** | **Parasitologic Grouping and Examples** |
| --- | --- |
| Bacteria  * Morphologic descriptions   + Cocci, bacilli, vibrios * Gram staining   + Gram-positive (high or low GC)   + Gram-negative * Oxygen requirements   + Aerobes and anaerobes  Chlamydia  * *Chlamydia pneumoniae* * *Chlamydia trachomatis*  Mycoplasma  * *Mycoplasma pneumoniae* * *Mycoplasma arthritidis* * *Mycoplasma genitalium*  Spirochetes  * *Treponema* spp (*Treponema pallidum*, *Treponema pertenue*, *Treponema carateum*) * *Leptospira* spp (*Leptospira icterohaemorrhagica*, *Leptospira canicola*) * *Borrelia* spp (*Borrelia recurrentis*, *Borrelia burgdorferi*) * *Spirillum minus*  Rickettsia  * *Rickettsia* spp * Spotted fever group * Typhus group * Scrub typhus group (now *Orientalis*)  Viruses  * DNA viruses   + Group 1: double-stranded DNA (pox, herpes, papova, hepadna)   + Group II: single-stranded DNA (parvo) * RNA viruses   + Group III: double-stranded (reo)   + Group IV: single-stranded (positive sense: orthomyxo, rhabdo, picorna, toga)   + Group V: single-stranded (negative sense: Ebola, Marburg)  Fungi  * Ascomycetes (sac fungi) * Basidiomycetes (club fungi) * Zygomycetes (mucor fungi) * Phycomycetes (algal fungi) * Morphology   + Unicellular (*Candida* spp, *Histoplasma* spp)   + Multicellular (*Aspergillus* spp, *Rhizopus* spp, *Fusarium* spp)   + Dimorphic (*Penicillium marneffei*) | Protozoa  * Flagellates   + i.   + *Trypanosoma* spp *(T cruzi*, *T brucei rhodesiense*, *T brucei gambiense*, *T rangeli*)   + ii.   + *Giardia lamblia*   + iii.   + *Leishmania* spp   + iv.   + *Trichomonas* spp * Ameboids   + i.   + *Entamoeba histolytica*   + ii.   + *Acanthamoeba* spp   + iii.   + *Naegleria fowleri* * Ciliates   + i.   + *Balantidium coli* * Sporozoans   + i.   + *Plasmodium* spp (*Plasmodium falciparum*, *Plasmodium malariae*, *Plasmodium vivax*, *Plasmodium ovale*)   + ii.   + *Babesia microti*   + iii.   + *Toxoplasma gondii*   + iv.   + *Microsporidium* spp   + v.   + *Cryptosporium* spp  Helminths  * Nematodes (roundworms, pin/threadworms, whipworms, hookworms)   + i.   + Gut nematodes (*Ascaris lumbricoides*, *Enterobius vermicularis*, *Trichuris trichiuria*, *Ancylostoma* spp, *Necator americanus*)   + ii.   + Tissue/muscle nematode (*Dracunculus medinensis*, *Trichinella spiralis*, *Gnathostoma spinigerum*, *Linguatella serrata*, *Armillifer armillatus*)   + iii.   + Central nervous system nematodes (*Angiostrongylus cantonensis*) * Trematodes (flatworms/flukes)   + i.   + Liver flukes (*Fasciola hepatica*, *Fasciolopsis buski*, *Clonorchis sinensis*, *Opisthorchis* spp)   + ii.   + Blood flukes (*Schistosoma haematobium*, *Schistosoma mansoni*, *Schistosoma japonicum*, *Schistosoma intercalatum*, *Schistosoma mekongi*)   + iii.   + Lung flukes (*Paragonimus westermani*) * Cestodes (tapeworms)   + i.   + Intestinal tapeworms (*Taenia solium*, *Taenia saginata*, *Diphyllobothrium latum*, *Hymenolepis nana*)   + ii.   + Intestinal tapeworm larval infections in organs:     - a.     - Cysticercosis (*Taenia solium* larvae)     - b.     - Echinococcosis (larvae of dog tapeworms *Echinococcus granulosus*, and *Echinococcus multilocularis*) |

[Open in a new tab](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/table/tbl1/)

*Abbreviation:* GC, guanine and cytosine.

### Table 2.

Some examples of tropical infectious diseases by main organ system involved

| **Main Organ System Involved** | **Common Pathogens** |
| --- | --- |
| Gastrointestinal | * Bacterial: all gastroenteritides, tuberculosis * Protozoal: Chagas disease, amebiasis, *Giardia*, coccidia * Helminthic: multiple |
| Hepatic | * Bacterial: leptospirosis, polymicrobial, anaerobes * Protozoal: amoebic hepatitis/abscess, malaria, trypanosomiasis * Helminthic: schistosomiasis, liver trematodes, hydatidosis * Viral: hepatitis A–E, yellow fever, herpes viruses |
| Respiratory | * Bacterial: tuberculosis, pneumococcal pneumonia, legionnaires, mycoplasma pneumonia * Fungal: aspergillosis, histoplasmosis, coccidioidomycosis, blastomycosis * Helminthic: paragonimiasis, strongyloides hyperinfection, hydatid, tropical pulmonary eosinophilia * Protozoal: *Plasmodium falciparum* |
| Cardiovascular | * Bacterial: endocarditis, rheumatic fever, tuberculosis, syphilis * Protozoal: Chagas disease * Helminthic: schistosomiasis |
| Renal tract | * Bacterial: poststreptococcal, tuberculosis * Helminthic: schistosomiasis * Protozoal: *Plasmodium falciparum* |
| Neurologic | * Bacterial: *Neisseria meningitidis* and other bacterial meningitis, leprosy, botulism, diphtheria * Protozoal: *Naegleria fowleri*, Acanthamoebae, trypanosomiasis, *Plasmodium falciparum* * Helminthic: cysticercosis, hydatid, *Angiostrongylus cantonensis*, gnathostomiasis * Viral: HIV, HTLV-1, Japanese encephalitis, enteroviruses, rabies |
| Dermatologic | * Bacterial: tropical ulcers, syphilis, mycobacteria (eg, leprosy, tuberculosis, *Mycobacterium ulcerans*), anthrax * Fungal: sporotrichosis, mycetoma, *Penicillium* * Protozoal: leishmaniasis * Helminthic: acute schistosomiasis, *Loa loa*, *Gnathostoma*, onchocerciasis, cutaneous larva migrans, larva currens * Arthropods: bites and stings, scabies, myiasis, tungiasis |
| Musculoskeletal | * Pyomyositis, trichinosis, cysticercosis, tuberculosis, hydatid |

[Open in a new tab](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/table/tbl2/)

### Table 3.

Main routes of transmission of tropical and parasitic diseases

| **Route/Mode of Transmission** | **Disease (Examples)** |
| --- | --- |
| Mother to child | |
| Congenital/vertical |  |
| Transplacental transmission via blood | TORCHES group of infections (toxoplasmosis, rubella, cytomegalovirus, *Herpes simplex*, syphilis), HIV, hepatitis viruses, malaria, trypanosomiases, bacterial infections |
| Perinatal |  |
| Vaginal/cervical contact during delivery | Bacterial, viral, fungal infections |
| Contact via breast milk | Sexually transmitted diseases |
| Airborne/inhalational | |
| Inhalation of air, aerosol, fomite contaminated by microbes | RTIs caused by bacteria, viruses, fungi, *Chlamydia* spp and *Mycoplasma* spp (eg, lobal pneumonia, influenza, pneumonic plague, tuberculosis) |
| Contact of skin/mucosa | |
| Direct (touching, kissing, sex) | Sexually transmitted diseases, mycosis, scabies, MRSA |
| Indirect (indirect contact with infected fomite, body fluid, secretions, stool, blood, plasma, or pus) | Boils, MRSA, sexually transmitted diseases, respiratory infections, *C difficile* and so forth |
| Ingestion | |
| Ingestion of any food or water contaminated with: |  |
| Microorganisms | Infections caused by bacteria (eg, typhoid, cholera, dysentery), viruses (eg, hepatitis A, B, and C), mycobacteria (eg, *Mycobacterium xenopi*), protozoa (eg, *Entamoeba histolytica*, *Cryptosporidium* spp) |
| Toxins | Staphylococcal, botulism, *Bacillus cereus*, scrombrotoxin, mushroom (*Amanita phalloides*) |
| Parasite ova/cysts | Infections caused by nematodes, trematodes, cestodes, protozoa (*Entamoeba histolytica*, *Cryptosporidium* spp) |
| Insect/arthropod-borne injection through skin penetration | |
| Mosquitoes and disease transmission |  |
| *Anopheles* spp | Malaria (all *Plasmodium* spp), bancroftian filariasis (*Wuchereria bancrofti*) |
| *Culicine* spp | Arbovirus encephalitis (eg, Japanese B encephalitis, St Louis encephalitis, West Nile virus) |
| *Aedes* spp | Yellow fever, filariasis (bancroftian) |
| Sandfly and disease transmission (*Phlebotomus* spp, *Lutzomyia* spp) | Leishmaniasis (all forms), sandfly fever (or Pappataci 3 day fever; Toscana, Sicilian, and Naples virus infections), bartenellosis (*Bartonella bacciliformis*) |
| Tsetse flies and disease transmission (*Glossina* spp) | Sleeping sickness (*Trypanosoma brucei rhodesiense*, *T brucei gambiense*) |
| Black flies (*Simulium* spp) | Onchocerciases (river blindness) (*Onchocerca volvulus*) |
| Horse/deer flies (*Chrysops* spp) | Filariasis (*Loa loa*), tularemia (*Francisella tularensis*) |
| Lice | Pediculosis  Trench fever, bacillary angiomatosis and endocarditis (*Bartonella quintana*), epidemic typhus (*Rickettsia prowazekii*), louse-borne relapsing fever (*Borrelia recurrentis*) |
| Fleas | Plague (*Yersinia pestis*), endemic/murine typhus (*Rickettsia typhi*), bartonellosis, and cat scratch disease (*Bartonella henselae*), dwarf tapeworm (*Hymenolepis nana*) |
| Arachnids | |
| Mites | Chiggers, scrub typhus (*Orientia tsutsugamushi*)  Scabies |
| Ticks | Lyme disease (*Borrelia burgdorferi*), tick typhus (Rocky Mountain spotted fever), ehrlichiosis (*Anaplasma phagocytophilum*), relapsing fever (*Borrelia recurrentis*), tularemia (*Francisella tularensis*), arboviruses (eg, Crimean-Congo hemorrhagic fever, Omsk hemorrhagic fever, babesiosis (*Babesia microti*) |
| Insect feces rubbed into skin | |
| Reduvid bugs (*Rhodnius* spp, *Triatoma* spp, *Panstrongylus* spp) | Chagas disease: feces of reduvid bugs with *T cruzi* spp are rubbed into skin by scratching) |
| Direct penetration through skin | |
| Helminth larvae | Helminth larvae penetration into subcutaneous tissue: swimmers itch (*Schistosoma* spp), hookworm and roundworm larvae |
| Fly larvae | Fly (bots and warbles) larvae (cutaneous myiases) |
| Innoculation or injection | |
| Breach of skin or mucous membrane caused by needles, tattoos, ear piercing, acupuncture, cupping, traditional scarification via blades | Viruses, bacteria, or fungal infections |
| Animal and human bites | Viruses (rabies, HIV, hepatitis B, hepatitis C, *Herpes* spp), bacterial infections (anaerobic and aerobic) including tetanus, actinomycosis, rat bite fever (*Spirillum minus*), *Pasteurella multocida*, *Capnocytophaga canimorsus* |
| Multiple modes of transmission | |
| Insect bites and airborne | eg, Plague: *Y pestis* flea bite (bubonic plague), airborne (pneumonic plague) |
| Direct contact, airborne, and ingestion of contaminated meat | eg, Anthrax: *Bacillus anthracis* skin contact with animal hides (cutaneous anthrax), airborne (pulmonary anthrax), ingestion of contaminated meat (gastrointestinal anthrax) |
| Insect bites, blood transfusion, needles, and congenital | eg, Malaria: *Plasmodium* spp |
| Skin/mucosa contact, needles, blood transfusion | eg, HIV, hepatitis B |

[Open in a new tab](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/table/tbl3/)

Many tropical infectious diseases are characterized by chronic inflammation as the battle between the host and pathogen becomes protracted.

Pathologic reports often describe the presence of a granuloma in biopsy tissue and the tissue may be processed with special stains, molecular methods, or culture to try to identify further. A granuloma[17](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/#bib17), [18](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/#bib18), [19](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/#bib19) is defined as a chronic, compact collection of inflammatory cells in which mononuclear cells predominate, usually formed as a result of an undegradable product, in the case of tropical infectious diseases; examples are given in [Table 4](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/#tbl4) . Some of the organisms contained within the granuloma remain viable, and these can reactivate to cause active disease when the patient becomes immunosuppressed from HIV or immunosuppressive therapy. Tuberculosis in HIV-infected individuals or in those on anti-TNF-α therapy, and Chagas disease in transplant recipients, are classic examples. Infectious diseases transmitted through medical procedures (eg, transfusion of blood or blood-related products[20](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/#bib20) and via transplantation) can also be classified microbiologically according to the type of microorganism ([Box 1](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/#tbox1) ).

### Table 4.

Infectious causes of granulomas

| **Class of Organism** | **Examples** | **Clinical Disease and Site of Granulomas** |
| --- | --- | --- |
| Bacteria | | |
| *Mycobacteria* spp | *Mycobacterium tuberculosis*  *Mycobacterium leprae*  *Mycobacterium kansasii*  *Mycobacterium marinum*  *Mycobacterium bovis* | Tuberculosis (any organ)  Leprosy (skin and nerves)  Pneumonia (lung)  Fish tank granuloma (skin)  BCGiosis (skin) |
| *Brucella* spp | *Brucella abortus*, *Brucella mellitensis*, *Brucella suis* | Brucellosis (any organ) |
| *Yersinia* spp | *Y pestis* | Plague (skin, lung) |
| *Listeria* spp | *Listeria monocytogenes* | Listerioses (brain) |
| Spirochetes | *Treponema pallidum*  *Treponema carateum* | Primary syphilis (skin)  Yaws (skin/mucous membranes) |
| Fungi | *Histoplasma capsulatum*  *Coccidioides immitis*  *Aspergillus fumigatus*  *Cryptococcus neoformans* | Histoplasmosis (any organ)  Cocciodomycoses (any organ)  Pulmonary aspergillosis (lung)  Cryptococcosis (any organ) |
| Protozoa | *Toxoplasma gondii*  *Leishmania* spp | Toxoplasmosis (eye or brain)  Leishmaniases (skin, mucous membranes, spleen, liver) |
| Helminth ova/larvae | | |
| Trematodes | *Schistosoma* spp  *Fasciola* spp, *Opisthorchis* spp | Granulomas (any organ)  Granulomas (liver, bile duct) |
| Cestodes | *Clonorchis sinensis*  *Taenia solium* | Granuloma around cysticerci (muscle, brain, subcutaneous tissue) |
| Helminth larvae | *Ascaris lumbricoides*, *Ancylostoma* spp, *Necator americanus* | Granulomas (cutaneous and visceral) around dead larvae |

[Open in a new tab](https://pmc.ncbi.nlm.nih.gov/articles/PMC7135174/table/tbl4/)

### Box 1. Classification of infections related to transfusion (of blood, platelet, immunoglobulin, clotting factors, or plasma).

#### Parasites

* *Plasmodium* spp
* *Babesia microti* ssp
* *Trypanosma cruzi*
* *Trypanosoma brucei* ssp
* *Leishmania donovani*
* *Toxoplasma gondii*

#### Viruses

* HIV-1, HIV-2
* Human T-lymphotropic virus (HTLV) type I, HTLV type II
* Hepatitis A, B, C, D, E
* Epstein B virus, cytomegalovirus
* Kaposi sarcoma herpesvirus (HHV-8)
* Parvovirus
* West Nile virus
* Severe acute respiratory syndrome

#### Bacteria

* Gram-negative bacteria (eg, *Pseudomonas* spp, *Yersinia* spp, *Salmonella* spp)
* Gram-positive bacteria (eg, *Staphylococcus* spp, *Streptococcus* spp, *Brucella* spp)

#### Spirochetes

* Spirochetes (eg, *Treponema pallidum*, *Leptospira* spp, *Borrelia burgdorferi*)
* Ehrlichia

#### Fungi

* *Candida* spp

#### Other

* New variant Creutzfeldt-Jakob disease prion

## **What are tropical diseases?**

[Tropical diseases](https://www.topdoctors.co.uk/doctor/tropical-diseases/) encompass all diseases that occur in the tropics and the term refers to infectious diseases that thrive in hot and humid conditions. The main reasons that infectious diseases spread in such regions are due to both environmental and biological factors that support high levels of biodiversity of pathogens and vectors, and hosts.

Social factors also play a role as to why the infections spread because efforts to control these diseases are undermined. Tropical diseases include malaria, leishmaniasis, schistosomiasis, onchocerciasis and African trypanosomiasis.

## **What are the most common typed of tropical diseases and infections?**

The following are the most common types of tropical diseases:

* **Tuberculosis** – this infection represents the leading cause of death associated with infectious diseases globally, especially in developing countries. It is a **chronic bacterial disease** caused by Mycobacterium tuberculosis. The disease develops slowly and the illness is prolonged. Symptoms of TB include coughing for three weeks or more, coughing up blood and [chest pain](https://www.topdoctors.co.uk/medical-dictionary/chest-pain).
* **Malaria** – this is an **infectious, hematologic disease** caused by a Plasmodium parasite. It is a life-threatening mosquito-borne disease transmitted to humans through the bite of the Anopheles mosquito. Symptoms include shaking chills, high fever, sweating, [headache](https://www.topdoctors.co.uk/medical-dictionary/headache), nausea and vomiting.
* **Diarrhoea** – rotavirus is one of the most common diseases that can affect young children. Rotavirus is found in countries such as Bangladesh, Somalia, Rwanda and Nepal. More serious epidemics can cause **dysentery** due to the bacteria Shigella dysenteriae. Symptoms include watery [diarrhoea](https://www.topdoctors.co.uk/medical-dictionary/diarrhoea), [fever](https://www.topdoctors.co.uk/medical-dictionary/fever), vomiting and [abdominal pain](https://www.topdoctors.co.uk/medical-dictionary/abdominal-pain).
* **Leishmaniasis** – this disease is caused by parasites of the Leishmania type and is spread through the bite of certain types of sandflies. It can be present in three main forms. The first, **visceral leishmaniasis** is the most deadly if left untreated in over ninety-five per cent of cases. Most cases occur in Brazil, East Africa and South-East Asia. Symptoms include fever, [weight loss](https://www.topdoctors.co.uk/medical-dictionary/unexplained-weight-loss), [anaemia](https://www.topdoctors.co.uk/medical-dictionary/anaemia) and enlargement of the spleen and liver. The most common type is **cutaneous leishmaniasis**, which causes skin lesions, mainly ulcers on exposed parts of the body. These can leave lifelong scars and serious disabilities. The third strain of leishmaniasis is **Mucocutaneous leishmaniasis**, which leads to partial or total destruction of mucous membranes of the nose, mouth and throat. This is usually contracted in Bolivia, Brazil, Ethiopia and Peru.

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## **How are tropical diseases diagnosed?**

The doctor will order laboratory tests that will check the [blood](https://www.topdoctors.co.uk/medical-dictionary/blood-test), urine and throat and also ask for stool samples. There may also take a spinal tap, which is a procedure that obtains a sample of the cerebrospinal fluid using a needle that is inserted between the bones of the lower spine. Imaging scans such as X-rays, computerised tomography and [MRI scans](https://www.topdoctors.co.uk/medical-dictionary/mri) can also help in diagnosis. Biopsies may be required to take a sample of tissue from an internal organ for testing.

## 

## **How are they treated?**

Treatment depends on what type of bacteria and type of tropical disease that the patient has. It could range from the following options:

* **Antibiotics**
* **Antivirals** – drugs that treat some, but not all, viruses including [HIV](https://www.topdoctors.co.uk/medical-dictionary/hiv)**/**[AIDS](https://www.topdoctors.co.uk/medical-dictionary/aids) and [Hepatitis B and C](https://www.topdoctors.co.uk/medical-dictionary/viral-hepatitis)
* **Antifungals** – used to treat skin or nail infections caused by fungi.
* **Antiparasitics** – to treat some diseases caused by parasites, however, some varieties have resistance to the medication.

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**LIST OF INFECTIOUS DISEASE (COMPLEX INFECTION: HIV, TROPICAL DISEASES)**

* Snakebite envenoming
* ‎Malaria – Plasmodium spp., transmitted by Anopheles mosquitoes
* ‎Leishmaniasis – Leishmania spp., transmitted by sandflies
* HIV
* ‎African Trypanosomiasis (Sleeping Sickness) – Trypanosoma brucei, tsetse fly vector
* ‎Chagas Disease (American Trypanosomiasis) – Trypanosoma cruzi, kissing bug vector
* ‎Amoebiasis – Entamoeba histolytica, via contaminated food/water
* ‎Giardiasis – Giardia lamblia, waterborne
* ‎Cryptosporidiosis – Cryptosporidium spp., waterborne
* ‎Helminthic (Worm) Infections
* ‎Schistosomiasis (Bilharzia) – Blood flukes, snail intermediate host
* ‎Lymphatic Filariasis (Elephantiasis) – Wuchereria bancrofti, mosquito vector
* ‎Onchocerciasis (River Blindness) – Onchocerca volvulus, blackfly vector
* ‎Soil-transmitted helminths
* Blastomycosis – Blastomyces dermatitidis
* ‎Talaromycosis (Penicilliosis) – Talaromyces marneffei (HIV-associated)
* ‎Candidiasis – common in immunocompromised patients
* Monkeypox (now Mpox) – contact with infected animals or humans
* **‎**Ascariasis – Ascaris lumbricoides
* ‎Trichuriasis – Trichuris trichiura
* ‎Hookworm – Necator americanus, Ancylostoma duodenale
* ‎Taeniasis and Cysticercosis – Taenia solium (pork tapeworm)
* ‎Echinococcosis (Hydatid disease) – Echinococcus spp.
* ‎Strongyloidiasis – Strongyloides stercoralis
* ‎Dracunculiasis (Guinea Worm Disease) – Dracunculus medinensis (almost eradicated)
* ‎Leprosy (Hansen's disease) – Mycobacterium leprae
* ‎Tuberculosis (TB) – Mycobacterium tuberculosis (prevalent in tropical areas)
* ‎Cholera – Vibrio cholerae, via contaminated water
* ‎Typhoid Fever – Salmonella typhi
* ‎Buruli Ulcer – Mycobacterium ulcerans
* ‎Hepatitis A and E – waterborne
* ‎Rabies – bites from infected animals
* ‎Histoplasmosis – Histoplasma capsulatum
* ‎Coccidioidomycosis (Valley Fever) – Coccidioides spp.
* ‎Taeniasis/cysticercosis
* **‎**Yaws – Treponema pallidum pertenue
* ‎Relapsing Fever – Borrelia spp., lice or ticks
* ‎Trachoma – Chlamydia trachomatis, causes blindness
* ‎Melioidosis – Burkholderia pseudomallei, soil and water exposure
* ‎Rickettsial diseases – e.g., Scrub typhus, Murine typhus
* Dengue Fever – Dengue virus, Aedes mosquitoes
* ‎Yellow Fever – Yellow fever virus, Aedes/Aedes aegypti mosquitoes
* ‎Zika Virus – Zika virus, Aedes mosquitoes
* ‎Chikungunya – Chikungunya virus, Aedes mosquitoes
* ‎Rift Valley Fever – Phlebovirus, mosquitoes and livestock
* ‎Crimean-Congo Hemorrhagic Fever (CCHF) – Ticks
* Ebola Virus Disease – contact with infected body fluids
* ‎Marburg Virus Disease – similar to Ebola
* ‎Lassa Fever – Arenavirus, rodent vector
* ‎Japanese Encephalitis – Flavivirus, Culex mosquitoes
* ‎West Nile Virus – mosquitoes
* Buruli ulcer
* ‎Chagas disease
* ‎Dengue and Chikungunya
* ‎Dracunculiasis
* ‎Echinococcosis
* ‎Foodborne trematodiases
* ‎Human African trypanosomiasis
* ‎Leishmaniasis
* ‎Leprosy
* ‎Lymphatic filariasis
* ‎Mycetoma, chromoblastomycosis and other deep mycoses
* ‎Onchocerciasis
* ‎Rabies
* ‎Scabies and other ectoparasites
* ‎Schistosomiasis
* ‎Soil-transmitted helminthiases
* Nipah Virus Infection – bats and pigs
* ‎Hantavirus – rodent droppings
* ‎Scrub Typhus – Orientia tsutsugamushi, chiggers

**SNAKEBITE ENVENOMING**

Snakebite envenoming is a potentially life-threatening disease caused by toxins in the bite of a venomous snake. Envenoming can also be caused by having venom sprayed into the eyes by certain species of snakes that have the ability to spit venom as a defence measure.

Inadequate past efforts to control snakebite envenoming has produced fragmented, inaccurate epidemiological data. Many victims do not attend health centres or hospitals and instead rely on traditional treatments. However, available data show 4.5–5.4 million people get bitten by snakes annually. Of this, 1.8–2.7 million develop clinical illness and 81 000 to 138 000 die from complications.

Snakebite envenoming represents a significant global health challenge that disproportionately affects rural communities in developing regions. This review examines current knowledge about this potentially fatal condition, from its definition to epidemiological patterns. According to the World Health Organization, approximately 4.5-5.4 million people suffer snakebites annually, with 1.8-2.7 million developing clinical illness and 81,000-138,000 deaths resulting from complications. Most deaths occur in Southeast Asia, India, Brazil, and Africa, with the majority being preventable through improved access to quality antivenoms and healthcare services.

**DESCRIPTION**

Snakebite envenoming is a potentially life-threatening disease caused by toxins introduced through the bite of a venomous snake. Envenoming can also occur when certain snake species spray venom into the eyes as a defensive mechanism. Snake venom is a complex mixture containing up to twenty or more toxins, primarily consisting of enzymes, non-enzyme peptide toxins, and non-toxic proteins.

Different snake families produce venoms with varying compositions and effects: cobra and krait venoms (elapids) are predominantly neurotoxic and cardiotoxic, while viper venoms are vasculotoxic with severe necrotizing local effects. The neurotoxins from elapids and sea snakes are absorbed rapidly into the bloodstream, causing quick systemic effects, whereas the larger molecules in viper venom travel more slowly through the lymphatics, resulting in more severe local effects. Most venoms do not cross the blood-brain barrier.

**CAUSES AND RISK FACTORS**

Snakebite envenoming occurs when a venomous snake injects toxins into tissue through specialized fangs. Several populations face heightened risk, including rural agricultural workers, herders, fishermen, hunters, and working children. People living in poorly constructed houses and those with limited access to education and healthcare also experience increased vulnerability.

Children represent a particularly high-risk group due to their smaller body mass compared to adults, which means they receive proportionally larger venom doses. This physiological factor contributes to their higher case fatality rates. Gender also plays a role in risk assessment; women in some cultures experience increased barriers to accessing medical care, and pregnant women are especially vulnerable to snakebite complications.

Environmental and behavioural factors significantly influence exposure risk. Those working barefoot in agricultural settings or walking through tall grass without proper footwear face increased chances of encountering venomous snakes. Additionally, people living in regions with fragmented healthcare infrastructure often lack timely access to antivenom, which exacerbates the consequences of envenoming.

**SYMPTOMS**

The clinical manifestations of snakebite envenoming vary considerably depending on the snake species, amount of venom injected, bite site, and victim characteristics. Even in "dry bites" where minimal or no venom is injected, psychological symptoms commonly occur, including flushing, breathlessness, palpitations, dizziness, chest tightness, sweating, and acroparesthesiae due to anxiety and sympathetic overactivity.

Local Symptoms

Local symptoms at the bite site may include:

* Pain, bleeding, bruising, swelling, or tenderness
* Progressive swelling that may involve an entire limb
* Blistering and tissue necrosis, particularly with viper bites
* Fang marks or scratch-like teeth marks on the skin

**Systemic Symptoms**

Systemic manifestations vary by snake type:

**Elapid bites** (cobras, kraits, mambas) typically present with:

* Vomiting, heaviness of eyelids, blurred vision, hypersalivation, and conjunctival congestion
* Neurotoxic effects including ptosis (drooping eyelids), ophthalmoplegia (fixed eyeballs), poor mouth opening, weak voice, pooling of saliva, and difficulty swallowing
* Progressive symmetric limb weakness and potential respiratory paralysis

**Viper bites** commonly exhibit:

* Hemorrhagic manifestations including bleeding from gums, hemoptysis, hematemesis, and ecchymosis
* Incoagulable blood and hypovolemic shock
* Abdominal tenderness and enlarged regional lymph nodes

**Sea snake envenomation** typically causes:

* Headache, thick feeling of the tongue, thirst, sweating, and vomiting
* Rhabdomyolysis (muscle breakdown) leading to muscle pain, tenderness, weakness, and dark urine

Nausea and vomiting are common symptoms across all severe envenomations. Additional symptoms may include seizures, diarrhea, fainting, fever, increased thirst, loss of muscle coordination, and rapid pulse.

**DIAGNOSIS METHODS**

Accurate and timely diagnosis of snakebite envenoming is crucial for appropriate treatment and can be approached through both clinical and laboratory methods.

**Clinical Diagnosis**

The clinical diagnosis relies on recognizing specific envenomation signs, including:

* Identification of snake species (when possible)
* Assessment of fang or bite marks, which can help differentiate between venomous and non-venomous snakebites
* Evaluation of local symptoms (swelling, blistering, necrosis)
* Assessment of systemic manifestations characteristic of specific snake families

Research indicates that examination of bite marks can be valuable for differential diagnosis-isolated fang marks have a sensitivity of 100%, specificity of 56%, and a predictive value of 89% for venomous snakebites, while multiple scratch-like teeth marks have a 100% predictive value for non-venomous snakebites.

**DIAGNOSIS**

Laboratory tests are essential for confirming envenomation and monitoring treatment response:

* The 20-minute whole blood clotting test (WBCT20) is a simple bedside test to assess coagulopathy
* Complete blood count to detect thrombocytopenia and changes in red and white blood cell counts
* Enzyme tests, particularly creatine phosphokinase, to assess muscle damage
* Urinalysis to detect myoglobinuria in cases of rhabdomyolysis
* Immunodiagnostic techniques such as enzyme immunoassay (EIA) or enzyme-linked immunosorbent assay (ELISA) to identify specific venom antigens in the victim's blood

These laboratory methods not only help confirm envenomation but also assist in identifying the responsible snake species when this information is unavailable, which is particularly valuable in regions where only monospecific antivenoms are available.

**TREATMENT**

The cornerstone of snakebite envenoming treatment is the timely administration of appropriate antivenom, which remains the only effective therapy for preventing or reversing most venomous effects. Snake antivenoms are included in the WHO list of essential medicines and work by neutralizing circulating venom components.

First Aid Measures

Proper first aid can significantly impact outcomes:

* Immediately move away from the snake's vicinity
* Remove constrictive items (jewelry, tight clothing) from the affected limb
* Immobilize the bitten limb to slow venom spread
* Transport the victim to a healthcare facility as quickly as possible
* Apply a pressure pad at the bite site in certain cases

Importantly, traditional first aid methods like cutting, sucking, or applying tourniquets should be avoided as they can worsen outcomes. Paracetamol may be given for pain management, and patients should be positioned on their left side (recovery position) in case vomiting occurs.

**Hospital Management**

In a healthcare setting, treatment typically includes:

* Antivenom administration based on clinical presentation and, when possible, snake identification
* Supportive care including airway management, respiratory support, and fluid resuscitation
* Treatment of complications such as acute kidney injury, coagulopathy, or compartment syndrome
* Wound care and tetanus prophylaxis
* Antibiotic therapy when secondary infection is present or suspected

Antivenom administration requires careful monitoring for adverse reactions, particularly anaphylaxis, and appropriate facilities for managing such emergencies should be available.

**RECOMMENDATION AND PREVENTION**

Preventing snakebite envenoming requires both individual behavioral changes and community-level interventions:

Individual Measures

* Wear protective footwear, especially when walking in grassy or wooded areas
* Use a light source when walking at night
* Avoid placing hands in locations not clearly visible
* Sleep under mosquito nets, particularly in areas with nocturnal snake species like kraits
* Clear vegetation and debris around dwellings to reduce snake habitats

Community Approaches

* Implement educational programs about snake behavior and bite prevention
* Improve housing construction to prevent snake entry
* Create proper waste management systems that reduce rodent populations (snake prey)
* Ensure access to transportation and healthcare facilities for rapid response to envenomation cases

The combination of these preventive strategies, along with raising awareness among communities and health workers about primary prevention methods, represents the most effective approach to reducing snakebite incidence and severity.

**PROGNOSIS**

The prognosis for snakebite envenoming varies significantly based on several factors including the snake species involved, venom dose, time elapsed before treatment, quality of care received, and individual patient characteristics. With prompt medical attention and appropriate antivenom administration, many snake bites will not result in serious long-term effects.

The outcome is particularly favorable when patients receive species-specific antivenom within the first few hours after envenomation. However, delayed presentation to healthcare facilities-common in rural and remote areas-significantly worsens prognosis. Children face higher mortality risks due to their smaller body mass relative to venom load.

Recovery time depends on the extent of envenomation and type of toxins involved. Neurotoxic effects may resolve within days to weeks, while local tissue damage from cytotoxic venoms can require months of healing and may result in permanent disability. Most deaths from snakebite are entirely preventable with timely access to quality antivenoms.

**POSSIBLE COMPLICATIONS**

Snakebite envenoming can lead to numerous complications affecting multiple organ systems, with severity dependent on the snake species, venom composition, and treatment timing.

Acute Complications

* Respiratory failure from neurotoxic paralysis, particularly affecting bulbar and respiratory muscles
* Cardiovascular collapse and shock due to vasodilation, increased vascular permeability, or direct cardiotoxicity
* Acute kidney injury resulting from direct nephrotoxicity, rhabdomyolysis, or hypotension
* Coagulopathy leading to systemic hemorrhage and potential cerebrovascular accidents
* Compartment syndrome in severely swollen limbs

Long-term Complications

* Permanent disability from tissue necrosis and subsequent scarring
* Limb amputation in cases of severe necrosis or gangrene
* Chronic kidney disease following acute kidney injury
* Psychological sequelae including post-traumatic stress disorder and depression
* Secondary bacterial infections at the bite site, particularly from oral flora present in snake mouths

Children may experience more severe complications due to their smaller body size and higher venom-to-body mass ratio. Additionally, pregnant women face risks of pregnancy loss, premature labor, placental abruption, and fetal death.

**WHEN TO CONSULT A DOCTOR**

All suspected snakebites should be considered medical emergencies requiring immediate professional attention, regardless of whether the snake was identified or symptoms are immediately apparent. Even seemingly minor bites can progress to life-threatening envenomation within hours.

Seek Emergency Care Immediately If:

* Any snake bite occurs, venomous or not
* Bite marks, pain, or swelling are present at a potential bite site
* Any systemic symptoms develop, including nausea, vomiting, dizziness, or weakness
* Bleeding occurs from the bite site or elsewhere (gums, wounds, etc.)
* Difficulty breathing, swallowing, or speaking develops
* Drooping eyelids or blurred vision occurs
* The bitten limb becomes increasingly swollen or painful

The critical window for effective antivenom administration is typically within the first few hours after envenomation, making rapid medical assessment essential. Even in cases where initial symptoms appear mild, monitoring in a healthcare setting is crucial as envenomation effects can develop progressively.

**DIAGNOSIS**

The differential diagnosis of snakebite envenoming requires distinguishing it from other conditions with similar presentations and determining whether a bite came from a venomous or non-venomous species.

Venomous vs. Non-venomous Bites

Examination of bite marks provides valuable diagnostic information:

* Isolated fang punctures strongly suggest venomous snakebite (sensitivity 100%, specificity 56%, predictive value 89%)
* Multiple scratch-like teeth marks reliably indicate non-venomous snakebite (predictive value 100%)

Other Conditions to Consider

* Spider bites, particularly from widow spiders (causing severe pain) or recluse spiders (causing local necrosis)
* Hymenoptera stings (bees, wasps) causing local reactions or anaphylaxis
* Scorpion stings, particularly those causing neurotoxic effects
* Allergic reactions to insects or plants
* Infections causing cellulitis or necrotizing fasciitis
* Acute abdominal conditions in cases presenting with abdominal pain (appendicitis, pelvic inflammatory disease)

When symptoms include abdominal tenderness, distinguishing from acute abdomen conditions is important-snake envenomation typically presents without rebound tenderness or rigidity.

Statistics and Epidemiology

Snakebite envenoming represents a significant global health challenge disproportionately affecting rural populations in tropical and subtropical regions. Current epidemiological data indicates approximately 4.5–5.4 million snakebites occur annually worldwide, resulting in 1.8–2.7 million cases of envenoming and 81,000–138,000 deaths. However, these figures likely underestimate the true burden due to fragmented reporting systems and the prevalence of victims seeking traditional healers rather than formal medical care.

**GEOGRAPHIC DISTRIBUTION**

The highest burden of snakebite mortality occurs in:

* Southeast Asia
* India
* Brazil
* Areas of Africa

These regions combine high venomous snake density with limited healthcare infrastructure and antivenom accessibility.

**Demographic Patterns**

Several demographic factors influence snakebite epidemiology:

* Age: Children and young adults suffer higher case fatality rates
* Occupation: Agricultural workers, herders, fishermen, and hunters face elevated risk due to environmental exposure
* Socioeconomic status: People with limited education and healthcare access experience greater vulnerability
* Gender: In some cultures, women face additional barriers to accessing medical care following snakebite

Notably, the socioeconomic impact extends beyond immediate health consequences, as victims often face catastrophic financial burdens from treatment costs and lost productivity, perpetuating cycles of poverty in affected communities.

Conclusion

Snakebite envenoming remains a significant yet neglected public health challenge affecting primarily rural communities in resource-limited settings. While substantial knowledge exists regarding its clinical manifestations, diagnosis, and treatment, considerable gaps persist in healthcare delivery and antivenom accessibility. The burden falls disproportionately on vulnerable populations, including children, agricultural workers, and those with limited healthcare access.

Effective management requires a comprehensive approach encompassing prevention strategies, prompt medical response, appropriate antivenom administration, and supportive care. Improvements in snake antivenom production, distribution, and affordability represent critical priorities for reducing mortality and morbidity. Additionally, community education about prevention measures and first aid, combined with strengthened healthcare systems capable of managing envenomation cases, could substantially reduce the global impact of this condition.

Looking forward, increased research, improved surveillance systems, and greater international collaboration will be essential for addressing this neglected tropical condition and achieving better outcomes for the millions affected annually by snakebite envenoming.

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**Monkeypox**

Mpox is a zoonotic viral disease transmitted through close contact with infected humans or animals. It causes a characteristic rash and systemic symptoms and can lead to serious complications in vulnerable populations. Diagnosis relies on clinical and laboratory methods, while treatment is mainly supportive. Prevention through hygiene, protective measures, and vaccination is key to controlling spread. The recent global outbreak has highlighted the need for continued surveillance and research to better understand and manage this re-emerging infectious disease.

Monkeypox, now officially renamed **mpox**, is an emerging zoonotic viral disease caused by the monkeypox virus (MPXV), a member of the Orthopoxvirus genus in the Poxviridae family. This virus is closely related to the variola virus, which causes smallpox, but mpox generally causes a milder illness. The disease was first identified in 1958 in monkeys kept for research, hence the original name, but the natural reservoir is believed to be small mammals such as rodents and squirrels in Central and West Africa, where the disease is endemic.

**MEDICAL AND COMMON NAMES**

* **Medical name:** Mpox (formerly monkeypox)
* **Common name:** Monkeypox (used historically)  
  The World Health Organization renamed the disease "mpox" in 2022 to reduce stigma and avoid geographic or cultural offense, while the virus retains the historic name monkeypox virus.

**DESCRIPTION**

Mpox is an infectious viral disease characterized by a painful rash, fever, swollen lymph nodes, headache, muscle aches, back pain, and fatigue. The rash progresses through stages: macules, papules, vesicles, pustules, and scabs, lasting 2 to 4 weeks. It is a zoonosis, meaning it can spread from animals to humans, and also spreads between humans primarily through close contact.

**CAUSES AND RISK FACTORS**

The disease is caused by infection with the monkeypox virus, which has two main clades:

* **Clade I** (Central African clade) associated with more severe disease and higher fatality rates.
* **Clade II** (West African clade), including subclade IIb responsible for the 2022–2023 global outbreak, generally causes milder illness.

**TRANSMISSION**

* **Animal-to-human contact:** Through bites, scratches, or handling infected animals such as squirrels, Gambian pouched rats, dormice, and monkeys during hunting, skinning, cooking, or playing with carcasses. The exact animal reservoir remains unknown.
* **Human-to-human contact:** Close skin-to-skin contact, sexual contact, mouth-to-mouth or mouth-to-skin contact, respiratory droplets during face-to-face interactions, and contact with contaminated objects like bedding, clothing, or needles.
* **Vertical transmission:** From mother to fetus or newborn during pregnancy or birth, which can cause severe outcomes.

**RISK FACTORS**

* Close contact with infected individuals, especially in households or sexual networks.
* People with multiple sexual partners have higher risk.
* Healthcare workers exposed to infected patients or contaminated materials.
* Immunocompromised individuals, children, and pregnant people are at increased risk of severe disease.

**SYMPTOMS**

Symptoms typically develop within 1 to 21 days after exposure, most commonly within a week. The illness begins with a prodromal phase lasting 1 to 3 days, including:

* Fever
* Headache
* Muscle aches
* Back pain
* Swollen lymph nodes (a key distinguishing feature from smallpox)
* Fatigue
* Sore throat and cough.

Following this, a characteristic rash appears, often starting on the face and spreading to the rest of the body, including palms and soles, genital and anal areas, mouth, and throat. The rash evolves through stages: flat macules → raised papules → fluid-filled vesicles → pus-filled pustules → scabs/crusts that eventually fall off. Lesions can be painful or itchy and may leave scars.

Some patients experience only a few lesions, while others have hundreds. Additional symptoms can include painful rectal swelling (proctitis), difficulty urinating (dysuria), and painful swallowing. People remain contagious until all lesions have healed and new skin has formed.

**DIAGNOSIS**

Diagnosis is primarily clinical, based on characteristic symptoms and rash appearance. Confirmation requires laboratory testing:

* **PCR testing** of lesion swabs or fluid is the gold standard to detect monkeypox virus DNA.
* Other tests include electron microscopy, virus isolation, and serology but are less commonly used.
* Differential diagnosis includes chickenpox, herpes simplex, syphilis, smallpox (eradicated), hand-foot-and-mouth disease, and allergic reactions.

**Treatment Options**

There is no specific antiviral treatment universally approved for mpox. Management is mainly supportive:

* Control of fever and pain with analgesics and antipyretics.
* Maintaining hydration and nutrition.
* Careful skin hygiene to prevent secondary bacterial infections.
* Treatment of complications such as bacterial superinfection or co-infections like HIV.
* Tecovirimat, an antiviral approved for smallpox, is sometimes used off-label for mpox under clinical guidance.
* Vaccination with smallpox vaccines (e.g., Jynneos, ACAM2000) is used for prevention and post-exposure prophylaxis in high-risk groups.

**PREVENTION**

Preventive measures focus on reducing contact with infected humans or animals:

* Avoid close, direct contact with people who have mpox symptoms or lesions.
* Avoid contact with wild animals or products from animals in endemic areas.
* Use personal protective equipment (PPE) when caring for infected patients or handling animals.
* Practice good hand hygiene and disinfect contaminated surfaces and objects.
* Vaccination is recommended for high-risk individuals and post-exposure to reduce disease severity and transmission.

**PROGNOSIS**

Most people recover fully within 2 to 4 weeks without lasting effects. The clade IIb strain causing the recent global outbreak has a low fatality rate (<3.3%). Clade I infections historically had higher mortality (~10%). Severe illness and death are more likely in children, pregnant people, and immunocompromised individuals.

**POSSIBLE COMPLICATIONS**

Complications can include:

* Secondary bacterial skin infections leading to abscesses or scarring.
* Pneumonia, sepsis, encephalitis, myocarditis (inflammation of the heart).
* Corneal infection causing vision loss.
* Painful inflammation of the rectum, genital organs, or urinary tract.
* Pregnancy complications such as miscarriage, stillbirth, or neonatal death.

**WHEN TO CONSULT A DOCTOR**

Seek medical care if you develop symptoms consistent with mpox, especially after known exposure to infected persons or animals. Immediate consultation is crucial if:

* Rash is widespread, painful, or accompanied by difficulty breathing, swallowing, or severe systemic symptoms.
* You are pregnant or immunocompromised.
* You experience signs of secondary infection or complications such as severe pain or vision changes.

**DIAGNOSIS**

Mpox can be confused with other rash illnesses:

* Chickenpox (varicella) - mpox lesions appear simultaneously and progress uniformly, chickenpox lesions appear in crops.
* Herpes simplex virus infections.
* Syphilis.
* Allergic or drug reactions.
* Smallpox (eradicated but historically similar).

**STATISTICS AND EPIDEMIOLOGY**

Between 2022 and January 2025, over 123,000 confirmed mpox cases were reported globally from 127 countries and territories. The Americas accounted for 54.9% of cases, Europe 23.4%, and Africa 15.8%. The 2022–2023 global outbreak was caused by the clade IIb strain, with ongoing outbreaks of clades Ia and Ib in Central Africa, particularly the Democratic Republic of Congo.

Mpox affects all demographics but is more common in people with close contact to infected individuals or animals. Children, pregnant people, and immunocompromised individuals are at higher risk for severe disease and complications. The disease remains endemic in parts of Central and West Africa, with periodic outbreaks elsewhere linked to travel or animal importation.

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**Candidiasis**

**MEDICAL AND COMMON NAMES**  
Candidiasis, also known as candidosis, moniliasis, or oidiomycosis, is a fungal infection caused by yeasts of the genus *Candida*, most commonly *Candida albicans*. It is often referred to as a "yeast infection" in common parlance, especially when affecting the vagina or mouth.

**DESCRIPTION**  
Candidiasis is an infection resulting from an overgrowth of *Candida* species, which are normally harmless fungi living on the skin and mucous membranes such as the mouth, throat, gut, and vagina. Under certain conditions, these fungi multiply excessively and cause infection. Candidiasis can manifest as superficial infections like oral thrush or vaginal yeast infections, or as invasive candidiasis when the fungus enters the bloodstream or internal organs, which is more serious and typically occurs in hospitalized or immunocompromised patients.

**CAUSES AND RISK FACTORS**  
The primary cause of candidiasis is the overgrowth of *Candida* species, particularly *Candida albicans*, but other species such as *C. glabrata*, *C. tropicalis*, *C. parapsilosis*, *C. krusei*, and the emerging *C. auris* can also cause infection. Normally, *Candida* is kept in check by the body's immune system and the balance of native bacteria. Disruption of this balance-due to antibiotics, immunosuppression, or changes in the local environment-can trigger candidiasis.

Common risk factors include:

* **Immunosuppression:** Conditions like HIV/AIDS, cancer, or use of immunosuppressive drugs (e.g., corticosteroids, chemotherapy) increase susceptibility.
* **Antibiotic use:** Broad-spectrum antibiotics reduce normal bacterial flora, allowing yeast to overgrow.
* **Diabetes mellitus:** High blood sugar and impaired immunity favor fungal growth.
* **Pregnancy and hormonal changes:** These can alter vaginal flora and pH, increasing risk of vaginal candidiasis.
* **Hospitalization:** Use of central venous catheters, surgery, and prolonged ICU stays increase risk of invasive candidiasis.
* **Other factors:** Poor hygiene, wearing tight or damp clothing, and high moisture areas on the skin favor superficial infections.

**SYMPTOMS**  
Symptoms vary depending on the site of infection:

* **Vaginal candidiasis (yeast infection):** Itching, soreness, burning sensation, pain during intercourse or urination, and thick, white, "cottage cheese-like" vaginal discharge. Severe cases may have redness, swelling, and fissures.
* **Oral candidiasis (thrush):** White, curd-like patches on the tongue, inner cheeks, roof of the mouth, and throat. These patches may be painful or bleed if scraped. Patients may experience redness, soreness, a cotton-like feeling, loss of taste, and cracking at the corners of the mouth.
* **Esophageal candidiasis:** Pain and difficulty swallowing, chest pain behind the breastbone. Usually occurs in immunocompromised individuals and often coexists with oral thrush.
* **Cutaneous candidiasis:** Red, moist, weepy skin patches, often in skin folds such as under breasts, groin, or between fingers. May have small pustules.
* **Invasive candidiasis:** Fever and chills that do not improve with antibiotics, potentially leading to shock and multi-organ failure if untreated. Symptoms depend on which organs are affected (heart, brain, eyes, bones, joints).

**DIAGNOSIS**  
Diagnosis is primarily clinical, based on characteristic symptoms and examination of affected areas. Confirmation involves:

* Microscopic examination and culture of skin scrapings, oral swabs, vaginal discharge, or blood samples.
* Polymerase chain reaction (PCR) and other molecular tests may be used for invasive infections.
* Endoscopy with biopsy for esophageal candidiasis.
* Blood cultures for candidemia (invasive candidiasis).

**Treatment Options**  
Treatment depends on the infection type and severity:

* **Topical antifungals:** Clotrimazole, nystatin, miconazole creams or suppositories for vaginal and cutaneous candidiasis. Oral lozenges or rinses for mild oral thrush.
* **Oral antifungals:** Fluconazole is commonly used for vaginal yeast infections and oral/esophageal candidiasis. Itraconazole and ketoconazole are alternatives.
* **Intravenous antifungals:** For invasive candidiasis, drugs such as echinocandins (caspofungin, micafungin), amphotericin B, or high-dose fluconazole are used[5](https://www.cdc.gov/candidiasis/about/index.html)[7](https://www.drugs.com/health-guide/candidiasis.html).
* Duration and choice of treatment depend on the site, severity, and patient’s immune status.

**RECOMMENDATION AND PREVENT**

* Maintain good hygiene and keep skin dry, especially in folds.
* Avoid tight, damp clothing and change out of wet clothes promptly.
* Use antibiotics only when necessary and complete prescribed courses.
* Manage underlying conditions such as diabetes effectively.
* For recurrent vaginal yeast infections, avoid irritants like scented soaps and douches.
* In healthcare settings, rigorous infection control to prevent *Candida auris* spread.

**PROGNOSIS**  
Most superficial candidiasis cases respond well to treatment and resolve without complications. Oral and vaginal candidiasis rarely cause serious illness in healthy individuals. However, invasive candidiasis can be life-threatening with high mortality rates, especially in immunocompromised or critically ill patients.

**POSSIBLE COMPLICATIONS**

* Secondary bacterial infections of affected skin or mucosa.
* Chronic or recurrent infections, especially in immunocompromised hosts.
* Esophageal candidiasis can cause strictures or perforation if untreated.
* Invasive candidiasis may cause endocarditis, meningitis, osteomyelitis, or multi-organ failure.

**WHEN TO CONSULT A DOCTOR**

* Persistent or severe symptoms of oral or vaginal candidiasis.
* Symptoms of esophageal candidiasis such as painful swallowing.
* Fever and chills unresponsive to antibiotics, especially in hospitalized or immunocompromised patients.
* Recurrent infections or infections not responding to over-the-counter treatments.

**DIAGNOSIS**

* Bacterial vaginosis or other sexually transmitted infections for vaginal symptoms.
* Herpes simplex virus or aphthous ulcers for oral lesions.
* Dermatophytosis or eczema for skin rashes.
* Other causes of fever and rash in invasive candidiasis cases.

**STATISTICS AND EPIDEMIOLOGY**

* Vaginal yeast infections affect approximately 75% of women at least once in their lifetime, with 40-45% experiencing recurrent episodes.
* Oral candidiasis occurs in about 6% of newborns and is common in immunocompromised adults.
* Invasive candidiasis is a significant cause of bloodstream infections in hospitalized patients, with mortality rates ranging from 30% to 60% depending on the population and promptness of treatment.
* *Candida auris*, first identified in 2009, has caused outbreaks in healthcare settings worldwide due to its multidrug resistance and persistence on surfaces.
* The incidence of candidiasis is rising globally, partly due to increased use of immunosuppressive therapies and invasive medical devices.

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**TALAROMYCOSIS (PENICILLIOSIS) -TALAROMYCE MARNEFFEI (HIV-ASSOCIATED)**

**Talaromycosis**, formerly known as penicilliosis, is a life-threatening fungal infection caused by *Talaromyces marneffei*. Primarily affecting immunocompromised individuals, especially those with HIV/AIDS, it is endemic in Southeast Asia, southern China, and northeastern India. Below is a comprehensive overview structured by key clinical and epidemiological aspects. Talaromycosis is a neglected tropical disease with high mortality in untreated HIV patients. Early diagnosis via antigen/DNA testing and prompt antifungal therapy coupled with ART are vital to improving outcomes. Public health efforts must prioritize HIV management and environmental risk reduction in endemic regions.

**Medical and Common Names**

* **Medical name:** Talaromycosis (replaced "penicilliosis" in 2014 to reflect taxonomic reclassification).
* **Common names:** Penicilliosis (historical), *Talaromyces marneffei* infection.

**DESCRIPTION**

Talaromycosis is an opportunistic systemic fungal infection characterized by fever, weight loss, anemia, and distinctive skin lesions. The fungus exists in two forms:

**Environmental mold:** Found in soil and decomposing organic matter in tropical regions.

**Parasitic yeast:** Transforms upon infecting warm-blooded hosts, disseminating via the bloodstream to organs like the skin, lungs, liver, and bone marrow.  
The disease is almost exclusively seen in immunocompromised individuals, with HIV-associated cases accounting for ~90% of infections.

Talaromycosis is an invasive fungal infection found in Southeast Asia, caused by breathing in a type of fungal pathogen called *Talaromyces marneffei*. Healthy people are unlikely to develop the disease, but for individuals with a weakened immune system caused by medical conditions, such as human immunodeficiency virus (HIV), it can be fatal. Up to one in three individuals with talaromycosis will die of the disease, even after receiving treatment.

Unfortunately, early symptoms are non-specific, often mild and difficult to spot, and methods of diagnosing talaromycosis take so long that the disease has often spread throughout the body by the time a patient has been diagnosed.

**CAUSES AND RISK FACTORS**

**Causative agent:**

* *Talaromyces marneffei*, a thermally dimorphic fungus.

**Transmission:**

* Inhalation of fungal spores from contaminated soil or organic matter.
* No evidence of human-to-human or animal-to-human transmission.

**Risk factors:**

* **HIV/AIDS** (CD4+ count <100 cells/μL).
* Immunosuppressive therapies (e.g., corticosteroids, organ transplant drugs).
* Autoimmune diseases, malnutrition, or cancer.
* Occupational exposure (e.g., farming, bamboo rat handling in endemic areas).

**SYMPTOMS**

Fever

General discomfort

Weight loss

Cough or shortness of breath

Swollen lymph nodes, liver, or spleen

Diarrhea

Abdominal pain

Symptoms vary between HIV-positive and HIV-negative individuals:

| **Symptom** | **HIV-Positive Patients** | **HIV-Negative Patients** |
| --- | --- | --- |
| **Skin lesions** | Painless papules with central umbilication. | Smooth, non-umbilicated nodules. |
| **Systemic involvement** | Disseminated disease (bloodstream, spleen, liver). | Localized infections (lungs, oral mucosa). |
| **Other symptoms** | Fever, weight loss, cough, diarrhea, anemia. | Bone lesions, lymphadenopathy, hepatomegaly. |

Symptoms typically appear weeks to years after exposure, with advanced HIV patients showing rapid progression.

**DIAGNOSIS METHODS**

Talaromycosis can be diagnosed by using a sample from the body part that is affected. For example:

* Bone marrow
* Blood
* Fluid in and around the lungs
* Lymph node
* Skin

The sample is sent to a laboratory for a fungal culture or to be examined under the microscope. The sample can also be tested for the presence of a protein or DNA of the fungus.

1. **Microscopy/Culture:**
   * Gold standard but slow (up to 4 weeks for culture).
   * Microscopic identification of yeast cells in biopsy specimens (e.g., bone marrow, skin).
2. **Molecular Methods:**
   * PCR to detect fungal DNA in blood or tissue.
3. **Antigen Detection:**
   * Galactomannan enzyme immunoassay (GM-EIA) for rapid diagnosis.
4. **Imaging:**
   * Chest X-rays showing diffuse reticulonodular shadows.

**DIAGNOSIS:** Tuberculosis, histoplasmosis, cryptococcosis, and lymphoma.

**Treatment Options**

1. **Initial Therapy:**
   * **Amphotericin B** (0.7 mg/kg/day IV) for 2 weeks.
2. **Maintenance Therapy:**
   * **Itraconazole** (200 mg/day orally) for 10 weeks.
3. **Antiretroviral Therapy (ART):**
   * Critical for HIV patients to restore immune function.
4. **Adjunctive Care:**
   * Manage anemia, secondary infections, and nutritional support.

**RECOMMENDATION AND PREVENTION**

* **Antifungal Prophylaxis:** Itraconazole (200 mg/day) for HIV patients with CD4+ <100 cells/μL in endemic regions.
* **Environmental Avoidance:** Limit exposure to soil during rainy seasons.
* **Early ART Initiation:** Reduces risk of opportunistic infections.

**PROGNOSIS**

* **Untreated mortality:** >90%.
* **Treated mortality:** 16.2–30%, with higher death rates in HIV patients during initial hospitalization.
* Poor prognostic factors: Leukopenia, elevated LDL, delayed diagnosis.

**POSSIBLE COMPLICATIONS**

* Disseminated intravascular coagulation (DIC).
* Respiratory failure, hepatic dysfunction.
* Secondary bacterial infections (e.g., sepsis).
* Relapse in 30% of cases without maintenance therapy.

**WHEN TO CONSULT DOCTOR**

* HIV patients in endemic regions with unexplained fever, weight loss, or skin lesions.
* Persistent cough, hepatosplenomegaly, or cytopenia.

**Diagnosis**

| **Condition** | **Distinguishing Features** |
| --- | --- |
| **Tuberculosis** | Positive acid-fast bacilli, granulomas on biopsy. |
| **Histoplasmosis** | Smaller yeast cells without transverse septation. |
| **Cryptococcosis** | Positive India ink stain, CSF antigen testing. |

**STATISTICS AND EPIDEMIOLOGY**

Talaromycosis (penicilliosis) is a 3rd leading cause of HIV-associated death in southern China and Southeast Asia. Mortality on antifungal therapy is upward 30%. The disease remains neglected due to lack of knowledge of disease distribution and burden. We therefore estimated the global burden of talaromycosis and modeled disease projection to 2025 to inform treatment and prevention strategies.

**Global burden (1964–2018):** 288,000 cases; 87,900 deaths.

**Endemic regions:** 99.7% of cases occur in Asia (China: 60.3%, Thailand: 30.4%, Vietnam: 8.4%).

**Affected demographics:**

**Gender:** 74.4% male.

**Age:** Rare in children (0.5% of cases).

**HIV co-infection:** 89.9% of cases.

**Projected incidence:** 35% increase by 2025 due to rising HIV rates in China.

**BLASTOMYCOSIS**

Blastomycosis is a pulmonary disease caused by inhaling spores of the dimorphic fungus *Blastomyces dermatitidis*. Occasionally, the fungi spread hematogenously, causing extrapulmonary disease. Symptoms result from pneumonia or from dissemination to multiple organs, most commonly the skin. Diagnosis is clinical, by chest x-ray, or both and is confirmed by laboratory identification of the fungi. For severe infection, treatment is initiated with a lipid formulation of amphotericin B. After clinical improvement, treatment can be transitioned to itraconazole. For mild to moderate disease that does not involve the central nervous system and does not require hospitalization, itraconazole is the recommended treatment

Blastomycosis is a systemic fungal infection caused primarily by the dimorphic fungus *Blastomyces dermatitidis*. This infection is notable for its ability to affect both immunocompetent and immunocompromised individuals, with a predilection for causing pulmonary disease and potential dissemination to other organs. Below is a detailed overview of blastomycosis structured by key clinical, microbiological, and epidemiological aspects.

**MEDICAL AND COMMON NAMES**

* **Medical name:** Blastomycosis
* **Causative organism:** *Blastomyces dermatitidis* (also *Blastomyces gilchristii* and related species)
* **Common names:** North American blastomycosis, Gilchrist disease (historical)

**DESCRIPTION**

Blastomycosis is a systemic pyogranulomatous infection caused by inhalation of airborne spores of *Blastomyces dermatitidis*, a thermally dimorphic fungus. The fungus exists in two forms:

* **Mycelial (mold) form** in the environment at ambient temperatures (~25°C), growing as a fluffy white mold producing infectious conidia (spores).
* **Yeast form** in human tissues at body temperature (~37°C), characterized by large, thick-walled yeast cells with broad-based budding.

The disease typically begins as a pulmonary infection after inhalation of spores from contaminated soil or decaying organic matter, especially in moist, acidic environments near rivers or lakes. Pulmonary infection can range from asymptomatic to severe pneumonia, and in 25-30% of cases, hematogenous dissemination occurs, leading to extrapulmonary disease affecting skin, bones, genitourinary tract, central nervous system, and other organs.

Unlike many other systemic fungal infections, blastomycosis frequently affects immunocompetent individuals as well as immunocompromised hosts.

**CAUSES AND RISK FACTORS**

* **Causative agent:** *Blastomyces dermatitidis*, a member of the family Ajellomycetaceae, is a thermally dimorphic fungus. It grows as mold in soil enriched with decaying organic material and animal droppings.
* **Transmission:** Infection occurs via inhalation of aerosolized conidia when soil or organic matter harboring the fungus is disturbed. There is no evidence of person-to-person transmission.
* **Environmental reservoir:** Endemic in soils of the Ohio and Mississippi River valleys, Great Lakes region, and southeastern United States. Also reported in parts of Canada, Africa, and rarely other continents.
* **Risk factors:**
  + Exposure to endemic areas, especially activities disturbing soil (hiking, farming, construction).
  + Immunocompromised states (HIV/AIDS, organ transplantation, corticosteroid use) increase risk and severity but are not required for infection.
  + Male gender is more commonly affected, possibly due to occupational exposures.
  + Age: Adults most commonly affected, but all ages can be infected.

**SIGNS AND SYMPTOMS**

The clinical presentation varies widely depending on the site and extent of infection:

**Pulmonary blastomycosis (most common):**

* Fever, chills
* Cough, often productive
* Chest pain
* Dyspnea (shortness of breath)
* Fatigue and weight loss
* May mimic bacterial pneumonia or tuberculosis

**Extrapulmonary dissemination (25-30% of cases):**

**Skin:** Painless or painful verrucous or ulcerative lesions, often on exposed areas such as face, arms, and legs. Lesions have irregular borders and may resemble squamous cell carcinoma or pyoderma gangrenosum.

**Bones and joints:** Osteomyelitis causing localized pain and swelling.

**Genitourinary tract:** Prostatitis, epididymitis, or testicular masses.

**Central nervous system:** Rare, but may cause meningitis or brain abscess.

**Other sites:** Oral and nasal mucosa, lymph nodes, liver, spleen.

Symptoms can develop insidiously over weeks to months, and some patients remain asymptomatic.

**DIAGNOSIS**

* **Microscopy:** Visualization of characteristic broad-based budding yeast cells (8-15 µm) in clinical specimens such as sputum, bronchoalveolar lavage, skin biopsy, or tissue samples.
* **Culture:** Growth of *B. dermatitidis* from clinical specimens on fungal media at 25°C (mycelial form) and 37°C (yeast form). Cultures may take 2-6 weeks.
* **Histopathology:** Tissue biopsies show granulomatous inflammation with yeast forms. Special stains like PAS or GMS highlight the fungi.
* **Antigen detection:** Blastomyces antigen testing in urine or serum can aid rapid diagnosis but may cross-react with other fungi.
* **Serologic tests:** Limited sensitivity and specificity; not routinely used for diagnosis.
* **Imaging:** Chest X-ray or CT scan may show lobar or patchy infiltrates, mass-like lesions, or cavitations. Imaging helps assess extent of pulmonary or disseminated disease.

**TREATMENT**

* **Mild to moderate pulmonary or localized disease:**
  + Oral itraconazole 200 mg twice daily for 6-12 months is the treatment of choice.
* **Severe or disseminated disease, CNS involvement, or immunocompromised patients:**
  + Initial therapy with amphotericin B (lipid formulation preferred) for 1-2 weeks or until clinical improvement.
  + Followed by prolonged oral itraconazole therapy for at least 12 months.
* **Monitoring:** Therapeutic drug monitoring of itraconazole levels is recommended to ensure efficacy.
* **Supportive care:** Oxygen, hydration, and treatment of secondary infections as needed.

**RECOMMENDATION AND PREVENTION**

* Avoid activities that disturb soil in endemic areas, especially for immunocompromised individuals.
* Use protective masks or respirators when exposure to dust or soil is unavoidable.
* Early diagnosis and treatment of blastomycosis reduce morbidity and mortality.
* No vaccine is currently available.

**PROGNOSIS**

* With appropriate antifungal therapy, prognosis is generally good.
* Untreated blastomycosis can be fatal due to respiratory failure or disseminated infection.
* Relapse can occur, especially with inadequate treatment duration or immunosuppression.
* Mortality rates vary but can reach up to 20% in severe cases.

**Possible Complications**

* Acute respiratory distress syndrome (ARDS) in severe pulmonary infection.
* Chronic pulmonary fibrosis or cavitary lung disease.
* Disseminated infection causing destructive skin lesions, bone destruction, or CNS abscesses.
* Secondary bacterial infections.
* Treatment-related toxicity, especially with amphotericin B.

**WHEN TO CONSULT A DOCTOR**

* Persistent cough, fever, chest pain, or unexplained weight loss, especially after travel or residence in endemic areas.
* Development of skin lesions or unexplained masses.
* Symptoms worsening despite antibiotic treatment for presumed bacterial pneumonia.
* Immunocompromised individuals with respiratory or systemic symptoms.

**DIFFERENTIAL DIAGNOSIS**

Blastomycosis can mimic several other diseases, including:

* **Bacterial pneumonia** (Staphylococcus, Streptococcus, atypical bacteria)
* **Tuberculosis**
* **Histoplasmosis** (another dimorphic fungus endemic in overlapping areas)
* **Coccidioidomycosis**
* **Squamous cell carcinoma or other malignancies** (when presenting as skin or lung masses)
* **Other fungal infections:** Cryptococcosis, paracoccidioidomycosis.

**STATISTICS AND EPIDEMIOLOGY**

* Blastomycosis is endemic in North America, particularly in the Ohio and Mississippi River valleys, Great Lakes region, and southeastern U.S. states.
* Incidence estimates vary but are approximately 1-2 cases per 100,000 population annually in endemic areas.
* Cases also reported in Canada, Africa, and rarely in Asia and South America.
* Males are affected more frequently than females, likely due to occupational exposures.
* Both immunocompetent and immunocompromised individuals can be affected, although immunosuppressed patients tend to have more severe disease.
* Outbreaks have been linked to environmental disturbances such as construction or flooding

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**SOIL-TRANSMITTED HELMINTHS**

Soil-transmitted helminthiasis is a widespread parasitic disease caused by nematode worms transmitted through contaminated soil. It disproportionately affects poor communities lacking sanitation, causing significant morbidity especially in children. Diagnosis relies on stool microscopy, and treatment with safe, effective anthelmintics is widely available. Prevention focuses on improving sanitation, hygiene, and education alongside mass deworming programs to reduce the global burden of disease.

Soil-transmitted helminthiases (STH) are among the most common parasitic infections worldwide, caused by intestinal nematodes transmitted through contaminated soil. These infections predominantly affect impoverished and vulnerable populations with poor sanitation and hygiene, leading to significant morbidity, especially in children.

**MEDICAL AND COMMON NAMES**

* **Medical name:** Soil-transmitted helminthiasis (STH)
* **Common names:** Intestinal worm infections, geohelminthiasis
* **Main causative species:**
  + *Ascaris lumbricoides* (roundworm)
  + *Trichuris trichiura* (whipworm)
  + Hookworms: *Necator americanus* and *Ancylostoma duodenale*

**DESCRIPTION**

Soil-transmitted helminthiasis refers to infections caused by parasitic nematode worms whose eggs or larvae develop in soil contaminated with human feces. Infection occurs when infective eggs or larvae are ingested or penetrate the skin. These worms inhabit the human intestine, causing chronic infection that impairs nutrition, growth, and cognitive development. STH infections are classified as neglected tropical diseases due to their high prevalence and impact on poor communities.

**CAUSES AND RISK FACTORS**

**Causative agents:**

* *Ascaris lumbricoides* (roundworm) - largest intestinal nematode, causes ascariasis.
* *Trichuris trichiura* (whipworm) - causes trichuriasis.
* Hookworms (*Necator americanus* and *Ancylostoma duodenale*) - cause hookworm infection.

**TRANSMISSION:**

* Eggs of *Ascaris* and *Trichuris* mature in soil contaminated with human feces and become infective. Infection occurs by ingestion of contaminated soil, food, or water.
* Hookworm eggs hatch in soil releasing larvae that penetrate human skin, usually through bare feet.

**RISK FACTORS:**

Poor sanitation and open defecation leading to soil contamination.

Lack of access to clean water and hygiene facilities.

Walking barefoot or contact with contaminated soil.

Consumption of raw or unwashed vegetables and fruits contaminated with infective eggs.

Living in tropical and subtropical regions with warm, moist climates favorable for egg and larval development.

Children are at higher risk due to playing outdoors and poor hygiene habits.

Women and marginalized populations are disproportionately affected.

**SYMPTOMS**

STH infections often cause mild or no symptoms but can lead to significant morbidity in heavy infections:

**Ascariasis (*Ascaris lumbricoides*):**

* Most infections asymptomatic.
* Heavy infections cause abdominal pain, intestinal obstruction, malnutrition, and impaired growth in children.
* Migration of larvae through lungs may cause cough, wheezing (Löffler’s syndrome).

**Trichuriasis (*Trichuris trichiura*):**

* Often asymptomatic or mild.
* Heavy infections cause abdominal pain, diarrhea, dysentery, rectal prolapse, and anemia.

**Hookworm infection (*Necator americanus*, *Ancylostoma duodenale*):**

* Skin penetration causes localized itching and rash ("ground itch").
* Chronic blood loss leads to iron-deficiency anemia, fatigue, and protein malnutrition.
* Severe infections can cause developmental delays in children.

**DIAGNOSIS**

* **Microscopic examination:** Identification of characteristic eggs in stool samples using concentration techniques.
* **Kato-Katz method:** Quantitative stool examination to estimate infection intensity.
* **Serological tests:** Limited use, mainly for research.
* **Clinical assessment:** Signs of anemia, malnutrition, and gastrointestinal symptoms support diagnosis in endemic areas.
* **Molecular methods:** PCR-based tests are available but not routinely used in endemic settings.

**TREATMENT**

* **Anthelmintic drugs:**
  + Albendazole (400 mg single dose) or mebendazole (500 mg single dose or 100 mg twice daily for 3 days) are first-line treatments effective against *Ascaris* and *Trichuris*.
  + Hookworm infections respond well to albendazole or mebendazole; pyrantel pamoate is an alternative.
* **Mass drug administration (MDA):** Periodic deworming of at-risk populations, especially school-age children, to reduce burden and transmission.
* **Supportive care:** Iron supplementation for anemia, nutritional rehabilitation.

**RECOMMENDATION AND PREVENTION**

* Improve sanitation and access to safe water to reduce soil contamination.
* Promote use of latrines and discourage open defecation.
* Educate communities on handwashing with soap before eating and after defecation.
* Encourage wearing shoes to prevent hookworm larval skin penetration.
* Wash and cook vegetables and fruits thoroughly.
* Implement regular deworming programs in endemic communities, especially targeting children.

**PROGNOSIS**

* With treatment, prognosis is excellent; infections are easily cured with anthelmintics.
* Untreated heavy infections can cause chronic malnutrition, anemia, growth retardation, and cognitive impairment.
* Re-infection is common in endemic areas without improved sanitation and hygiene.

**POSSIBLE COMPLICATIONS**

* Severe anemia leading to fatigue, developmental delays, and increased susceptibility to other infections.
* Intestinal obstruction due to heavy *Ascaris* worm load.
* Rectal prolapse from severe *Trichuris* infection.
* Impaired physical and cognitive development in children.
* Secondary bacterial infections from skin lesions caused by hookworm penetration.

**WHEN TO CONSULT A DOCTOR**

* Persistent abdominal pain, diarrhea, or blood in stool.
* Symptoms of anemia such as fatigue, pallor, or shortness of breath.
* Sudden severe abdominal pain or vomiting (possible intestinal obstruction).
* Chronic malnutrition or growth delay in children from endemic areas.
* Skin rash or itching after walking barefoot in endemic regions.

**DIFFERENTIAL DIAGNOSIS**

Other causes of anemia (nutritional deficiencies, malaria).

Gastrointestinal infections (bacterial, viral, other parasitic).

Inflammatory bowel disease.

Other helminth infections such as strongyloidiasis.

Malignancies or other causes of intestinal obstruction.

**STATISTICS AND EPIDEMIOLOGY**

* Approximately 1.5 to 2 billion people worldwide are infected with soil-transmitted helminths, about a quarter of the global population.
* The highest burden is in tropical and subtropical regions of Sub-Saharan Africa, Latin America, Southeast Asia, and China.
* Children aged 1-14 years are most affected, with an estimated 46 million at risk in the Americas alone.
* Women and children bear the highest morbidity due to nutritional and developmental impacts.
* Soil-transmitted helminth infections are among the most prevalent neglected tropical diseases targeted by global control programs such as the WHO’s preventive chemotherapy initiatives.
* Despite mass deworming efforts, reinfection rates remain high due to persistent environmental contamination and poor sanitation.

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**ONCHOCERCIASIS**

**Onchocerciasis** (river blindness) is a debilitating parasitic disease caused by *Onchocerca volvulus* and transmitted by blackflies breeding in fast-flowing rivers. It causes severe skin disease and blindness primarily in sub-Saharan Africa. Diagnosis relies on skin snips and clinical signs, while treatment with ivermectin and vector control are key to reducing disease burden. Continued global efforts aim to eliminate onchocerciasis as a public health problem

Onchocerciasis, commonly known as **river blindness**, is a parasitic disease caused by the filarial nematode *Onchocerca volvulus*. It is transmitted to humans through the bites of infected female blackflies (*Simulium* species) that breed in fast-flowing rivers and streams, primarily in remote rural areas where people rely on agriculture. This disease is a major cause of blindness and skin disease in affected populations, particularly in sub-Saharan Africa.

**MEDICAL AND COMMON NAMES**

* **Medical name:** Onchocerciasis
* **Common name:** River blindness
* **Causative agent:** *Onchocerca volvulus* (filarial worm)
* **Vector:** Female blackfly (*Simulium* spp.)

**DESCRIPTION**

Onchocerciasis is a chronic parasitic infection characterized by severe itching, disfiguring skin conditions, and visual impairment, including permanent blindness. The disease is named “river blindness” because the blackfly vector breeds in rapidly flowing rivers and streams, and the most devastating consequence is blindness caused by the parasite’s effect on ocular tissues.

The lifecycle begins when an infected blackfly bites a human, depositing infective larvae into the skin. These larvae mature into adult worms that form nodules under the skin. Female worms produce microfilariae (immature larvae) that migrate through the skin and eyes, causing intense immune reactions and tissue damage. The disease requires repeated exposure to infected blackfly bites, as many bites are needed before infection establishes.

**CAUSES AND RISK FACTORS**

* **Cause:** Infection by *Onchocerca volvulus* transmitted by female blackflies during blood meals.
* **Transmission:** Blackflies ingest microfilariae when biting infected humans; microfilariae develop into infective larvae inside the fly, which are then transmitted to other humans.
* **Risk factors:**
  + Living or working near fast-flowing rivers and streams where blackflies breed.
  + Residing in endemic rural areas of sub-Saharan Africa (most cases), Yemen, and limited foci in Latin America.
  + Repeated exposure to blackfly bites over months or years.
  + Occupations such as farming, fishing, or washing clothes near rivers increase risk.
  + Long-term residents and migrants in endemic areas.

**SYMPTOMS**

**Skin manifestations:**

Intense itching (pruritus), often the earliest symptom.

Papular rash and skin depigmentation (“leopard skin”).

Subcutaneous nodules containing adult worms.

Skin atrophy and thickening, leading to disfigurement.

Enlarged lymph nodes near affected areas.

**Ocular manifestations:**

Itching and inflammation of the eyes.

Conjunctivitis and sclerosing keratitis.

Chorioretinitis and optic atrophy.

Progressive vision loss leading to irreversible blindness.

**Systemic:**

* General malaise and secondary bacterial infections from scratching.

**DIAGNOSIS METHODS:**

* **Skin snip biopsy:** Small pieces of skin are taken and incubated in saline; emerging microfilariae confirm diagnosis.
* **Slit-lamp eye examination:** Detects microfilariae in the cornea or anterior chamber.
* **Nodule biopsy:** Identification of adult worms in subcutaneous nodules.
* **Serological tests and PCR:** Detect parasite DNA or antibodies but are less commonly used in field settings.
* **Clinical diagnosis:** Based on typical skin lesions, nodules, and ocular symptoms in endemic areas.

**TREATMENT**

* **Ivermectin:** The cornerstone of treatment; administered every 6 to 12 months to kill microfilariae, reducing symptoms and transmission. It does not kill adult worms, so repeated doses over 10-15 years are needed.
* **Doxycycline:** Targets *Wolbachia* bacteria symbiotic with the worms, weakening adult worms and reducing microfilariae production.
* **Nodule removal:** Surgical excision of nodules containing adult worms may be performed.
* **Symptomatic treatment:** Antihistamines and corticosteroids to reduce itching and inflammation.
* **Mass drug administration (MDA):** Community-wide ivermectin distribution programs aiming for >80% coverage to interrupt transmission.

**RECOMMENDATION AND PREVENTION**

* Avoid blackfly bites by wearing protective clothing and using insect repellents.
* Reduce exposure by avoiding areas near fast-flowing rivers during peak blackfly activity.
* Vector control through larviciding rivers to reduce blackfly populations.
* Support and participate in MDA programs to reduce community infection burden.
* Health education on the importance of repeated ivermectin treatment.

**PROGNOSIS**

* With regular ivermectin treatment, progression of skin and eye disease can be halted and partially reversed.
* Without treatment, onchocerciasis can lead to severe disfigurement and irreversible blindness.
* Long-term community treatment programs have significantly reduced disease incidence and transmission in many endemic areas.

**POSSIBLE COMPLICATIONS**

* Permanent blindness due to optic nerve and retinal damage.
* Severe skin disfigurement and secondary infections from scratching.
* Social stigma and economic hardship due to disability.
* Rarely, systemic complications such as epilepsy and nodding syndrome have been associated.

**WHEN TO CONSULT A DOCTOR**

* Persistent intense itching and skin changes in persons living in or visiting endemic areas.
* Development of skin nodules or unexplained skin discoloration.
* Visual symptoms such as eye irritation, pain, or vision loss.
* After multiple blackfly bites in endemic regions, especially if symptoms develop.

**DIFFERENTIAL DIAGNOSIS**

* Other causes of pruritic skin diseases (scabies, eczema).
* Other causes of infectious blindness (trachoma, onchocerciasis mimics).
* Other filarial infections (loiasis, lymphatic filariasis).
* Dermatological conditions causing nodules or skin changes.

**STATISTICS AND EPIDEMIOLOGY**

* Approximately 21 million people are infected worldwide, with about 14.6 million showing skin disease and 1.15 million suffering visual impairment or blindness.
* The disease is endemic in 30 countries, predominantly in sub-Saharan Africa, with smaller foci in Yemen and Latin America.
* Onchocerciasis is the second leading infectious cause of blindness worldwide after trachoma.
* Control efforts with ivermectin MDA have drastically reduced prevalence and transmission in many regions.
* Colombia, Ecuador, Mexico, and Guatemala have been declared free of onchocerciasis by WHO.
* The disease disproportionately affects rural agricultural communities near rivers.

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**SCHISTOSOMIASIS**

**Schistosomiasis** (bilharzia) is a widespread parasitic disease caused by blood flukes transmitted via freshwater snails. It causes chronic illness affecting the urinary and intestinal tracts, with severe long-term complications if untreated. Diagnosis relies on detection of parasite eggs and clinical assessment, while praziquantel remains the mainstay of treatment. Prevention focuses on reducing water contamination, snail control, and minimizing human exposure to infested water. Continued public health efforts are essential to control and eventually eliminate this debilitating disease.

Schistosomiasis, also known as **bilharzia** or **snail fever**, is a parasitic disease caused by blood flukes of the genus *Schistosoma*. It is a major neglected tropical disease affecting over 200 million people worldwide, primarily in tropical and subtropical regions. The disease is closely linked to poverty and poor sanitation and is transmitted through contact with freshwater contaminated by larval forms of the parasite released from infected freshwater snails, which act as intermediate hosts.

**Medical and Common Names**

* **Medical name:** Schistosomiasis
* **Common names:** Bilharzia, snail fever
* **Causative agents:** Parasitic blood flukes (*Schistosoma* species)
* **Intermediate host:** Freshwater snails (various species depending on *Schistosoma* species)

**DESCRIPTION**

Schistosomiasis is a chronic parasitic infection caused by trematode worms (blood flukes) of the genus *Schistosoma*. The parasite’s lifecycle involves freshwater snails as intermediate hosts, where larval forms (cercariae) develop and are released into water. Humans become infected when cercariae penetrate the skin during contact with contaminated water. The parasites mature into adult worms residing in blood vessels, where they produce eggs that cause immune reactions and tissue damage. The disease primarily affects the urinary tract or intestines, depending on the species involved.

**CAUSES AND RISK FACTORS**

* **Causative species:**
  + *Schistosoma mansoni* (intestinal schistosomiasis)
  + *Schistosoma haematobium* (urogenital schistosomiasis)
  + *Schistosoma japonicum* (intestinal schistosomiasis, more zoonotic)
  + Other less common species include *S. mekongi* and *S. intercalatum*.
* **Transmission:**
  + Freshwater snails infected with *Schistosoma* release cercariae into water bodies.
  + Cercariae penetrate human skin during activities such as swimming, bathing, fishing, washing clothes, or collecting water.
  + The parasites then migrate through the bloodstream to their target organs.
* **Risk factors:**
  + Living in or traveling to endemic areas in Africa, South America, the Caribbean, the Middle East, and Asia.
  + Frequent contact with contaminated freshwater sources.
  + Poor sanitation and lack of access to clean water.
  + Agricultural, fishing, and domestic activities involving freshwater exposure.
  + Children and women may have higher exposure due to play and household chores.

**SYMPTOMS**

Symptoms vary by infection stage and *Schistosoma* species:

**Acute phase (Katayama fever):**

Occurs weeks after infection.

Fever, chills, cough, muscle aches, headache, and rash.

Eosinophilia and systemic hypersensitivity reaction to migrating larvae.

**Chronic phase:**

Caused by immune response to eggs trapped in tissues.

**Intestinal schistosomiasis (*S. mansoni*, *S. japonicum*):**

Abdominal pain, diarrhea, bloody stools.

Hepatosplenomegaly, portal hypertension, and fibrosis in severe cases.

In children, chronic infection may cause anemia, growth retardation, and cognitive impairment.

**Urogenital schistosomiasis (*S. haematobium*):**

Hematuria (blood in urine), dysuria, increased urinary frequency.

Bladder wall inflammation, fibrosis, and increased risk of bladder cancer.

Female genital schistosomiasis causing vaginal bleeding, pain, and increased HIV susceptibility.

**RARE COMPLICATIONS:**

Neuroschistosomiasis (eggs in brain or spinal cord) causing seizures, paralysis, or spinal inflammation.

Pulmonary hypertension secondary to egg embolization.

**DIAGNOSIS METHODS**

**Microscopic detection of eggs:**

Stool examination for *S. mansoni* and *S. japonicum* eggs.

Urine examination (filtration technique) for *S. haematobium* eggs, especially midday samples.

**Serological tests:**

Detect antibodies or circulating antigens; useful in low-intensity infections or travelers.

**Imaging:**

Ultrasound to assess liver fibrosis, portal hypertension, bladder wall changes.

**Molecular methods:**

PCR assays for parasite DNA in clinical samples (research/advanced settings).

**Clinical diagnosis:**

Based on symptoms, exposure history, and endemicity.

**TREATMENT**

**Praziquantel:** The drug of choice for all schistosome species.

Single or multiple doses depending on infection intensity.

Effective at killing adult worms but not immature stages; repeated treatment may be necessary.

**Supportive care:**

Management of anemia, nutritional support, treatment of secondary infections.

**Mass drug administration (MDA):**

WHO recommends periodic praziquantel treatment of at-risk populations, especially school-aged children, in endemic areas to reduce morbidity and transmission.

**RECOMMENDATION AND PREVENTION**

Avoid contact with contaminated freshwater where schistosomiasis is endemic.

Use safe water sources for bathing, washing, and drinking.

Improve sanitation to reduce contamination of water bodies with human feces and urine.

Control snail populations using molluscicides or environmental management.

Health education on risks of freshwater exposure and benefits of treatment.

Promote use of protective clothing or footwear when exposure is unavoidable.

**PROGNOSIS**

Early diagnosis and treatment with praziquantel generally result in cure and symptom resolution.

Untreated chronic infections can lead to severe organ damage, including liver fibrosis, portal hypertension, bladder cancer, and infertility.

Reinfection is common in endemic areas without improved water and sanitation.

**POSSIBLE COMPLICATIONS**

**Hepatic fibrosis and portal hypertension:** Leading to ascites, esophageal varices, splenomegaly, and gastrointestinal bleeding.

**Bladder fibrosis and cancer:** Especially with *S. haematobium* infection.

**Neuroschistosomiasis:** Seizures, paralysis, and neurological deficits.

**Pulmonary hypertension:** Due to embolization of eggs to lungs.

Secondary bacterial infections and malnutrition.

**WHEN TO CONSULT A DOCTOR**

* After freshwater exposure in endemic areas with symptoms such as rash, fever, abdominal pain, blood in stool or urine.
* Persistent urinary symptoms or hematuria.
* Signs of chronic liver or bladder disease.
* Neurological symptoms such as seizures or paralysis.

**DIFFERENTIAL DIAGNOSIS**

* Other causes of hematuria (urinary tract infections, stones, malignancy).
* Other parasitic infections causing gastrointestinal symptoms (amoebiasis, giardiasis).
* Viral hepatitis and other causes of liver fibrosis.
* Tuberculosis of the genitourinary tract.
* Other causes of eosinophilia and systemic symptoms.

**STATISTICS AND EPIDEMIOLOGY**

* Over 200 million people infected worldwide, primarily in sub-Saharan Africa, with significant cases in South America, the Caribbean, the Middle East, and parts of Asia.
* Approximately 700 million people live in endemic areas and are at risk.
* Schistosomiasis is the second most socioeconomically devastating parasitic disease after malaria.
* Children and women are disproportionately affected due to exposure patterns.
* Control programs with praziquantel MDA have reduced prevalence and morbidity but reinfection remains a challenge.
* Co-infections with HIV and hepatitis viruses are common in endemic regions, worsening outcomes.

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**LYMPHATIC FILARIASIS (ELEPHANTIASIS):**

Lymphatic filariasis (LF) is a neglected tropical disease that affects millions of people globally, causing significant disability and stigma. This parasitic infection, primarily caused by Wuchereria bancrofti, attacks the lymphatic system, leading to debilitating chronic conditions. The disease remains a significant public health challenge in 39 countries, with 657 million people living in areas requiring preventive interventions as of 2023. This report provides a detailed exploration of lymphatic filariasis, focusing on its causes, symptoms, diagnosis, treatment options, and prevention strategies.

**MEDICAL AND COMMON NAMES**

Lymphatic filariasis is the official medical term for this parasitic disease. It is commonly known as elephantiasis, referring to the characteristic extreme swelling and skin hardening that resembles an elephant's hide in advanced cases. The condition is also referred to as Wuchereriasis when specifically caused by Wuchereria bancrofti. The name of this particular parasitic worm honors two scientists, Wucherer and Bancroft, who made significant contributions to studying the disease. In medical literature and public health communications, the abbreviation "LF" is frequently used when referring to lymphatic filariasis.

**DESCRIPTION**

Lymphatic filariasis is a parasitic disease characterized by the infection of the lymphatic system by filarial nematodes (roundworms). It is classified as a neglected tropical disease that primarily affects populations in tropical and subtropical regions. The infection occurs when filarial parasites are transmitted to humans through mosquito bites, with the parasites typically entering the body through the skin during a mosquito blood meal.

The disease is characterized by its chronic nature, with infection usually acquired in childhood causing hidden damage to the lymphatic system over time. Wuchereria bancrofti is responsible for approximately 90% of lymphatic filariasis cases worldwide. This parasitic infection disrupts the normal function of the lymphatic vessels, leading to impaired lymph drainage and subsequent accumulation of lymphatic fluid in affected tissues. The disease's progression can lead to permanent disability, with visible manifestations typically appearing years after the initial infection.

**Causes and Risk Factors**

Lymphatic filariasis is caused by parasitic filarial nematodes (roundworms) belonging to the family Filariodidea. Three species of these thread-like filarial worms can cause the disease:

1. Wuchereria bancrofti - responsible for approximately 90% of cases worldwide
2. Brugia malayi - causes most of the remaining cases
3. Brugia timori - also causes the disease but is less common

The transmission cycle involves both human hosts and mosquito vectors. Adult worms nest in the human lymphatic vessels, where they can live for approximately 6-8 years. During their lifetime, female worms produce millions of microfilariae (immature larvae) that circulate in the infected person's bloodstream. When a mosquito bites an infected person, it ingests these microfilariae, which then develop into infective larvae within the mosquito. When this infected mosquito subsequently bites another person, the mature parasite larvae are deposited on the skin and can enter the body, migrating to the lymphatic vessels where they develop into adult worms, thus continuing the cycle of transmission.

The disease is transmitted by different types of mosquitoes, including:

* Culex mosquitoes (widespread across urban and semi-urban areas)
* Anopheles mosquitoes (mainly found in rural areas)
* Aedes mosquitoes (mainly in endemic islands in the Pacific)

**Risk Factors**

Several risk factors have been identified for lymphatic filariasis infection:

1. **Gender**: Males have a significantly higher risk of infection. Studies have shown that men have approximately twice the risk of antigenemia (OR = 2.0) and over five times the risk of microfilaremia (OR = 5.4) compared to women.
2. **Age**: Infection rates increase with age up to approximately 20 years and then remain relatively stable. This pattern suggests cumulative exposure over time.
3. **Occupation**: Individuals engaged in hunting or fishing activities, particularly at night, have an increased risk (OR = 1.5) of infection. This is likely due to increased exposure to mosquito vectors during these activities.
4. **Sanitation**: The absence of proper sanitation facilities increases risk. Studies have demonstrated a protective effect of latrines (OR = 0.5), suggesting that improved sanitation reduces exposure to mosquito breeding sites.
5. **Protective measures**: The use of bed nets has been shown to provide protection, particularly for females (OR = 0.4).
6. **Household factors**: There is a strong household effect on infection risk, with intraclass correlation coefficients of 0.24 for antigenemia and 0.49 for microfilaremia. This indicates clustering of cases within households.
7. **Geographic location**: Living in endemic areas, particularly in tropical and subtropical regions where the mosquito vectors thrive, significantly increases risk.
8. **Exposure patterns**: Evidence suggests that men often acquire infection in high transmission areas outside villages, while children and women are more commonly infected in areas with lower transmission inside or near villages.

**SYMPTOMS**

Lymphatic filariasis presents with a spectrum of clinical manifestations, ranging from asymptomatic infection to severe disfigurement. It's important to note that most people infected with the filarial parasites never develop visible symptoms, despite potentially harboring the infection.

**Asymptomatic Infection**

The majority of infected individuals remain asymptomatic, although they may have microfilariae in their blood and hidden damage to their lymphatic system. These individuals can still serve as reservoirs for continued transmission of the disease.

**Acute Manifestations**

Some infected individuals may experience recurrent episodes of acute adenolymphangitis characterized by:

* Fever
* Lymph node inflammation
* Inflammation of the lymphatic vessels

**Chronic Manifestations**

For approximately one-third of infected individuals, the disease progresses to cause visible signs and symptoms, which may appear months or even years after the initial infection. These chronic manifestations include:

1. **Lymphedema**: Progressive swelling of the limbs, particularly the legs, but can also affect the arms, breasts, and genitalia. In advanced cases, this leads to elephantiasis, characterized by massive enlargement and hardening of affected tissues.
2. **Hydrocele**: Swelling of the scrotum in men due to the accumulation of fluid, which can become extremely large and debilitating.
3. **Skin changes**: Hardening or thickening of the skin in affected areas, often with hyperkeratosis (excessive growth of the outer layer of skin) and skin folds.
4. **Respiratory symptoms**: Some patients may experience persistent coughing, wheezing, or shortness of breath, particularly in cases of tropical pulmonary eosinophilia (TPE), a manifestation of occult filariasis.
5. **Secondary bacterial infections**: Chronic lymphedema creates an environment conducive to recurring bacterial infections of the skin and lymph system. These secondary infections occur because the compromised lymphatic system makes it difficult for the body to fight germs and infections effectively.

The progression from initial infection to chronic disease typically occurs over many years, with repeated mosquito bites in endemic areas contributing to the parasite burden and subsequent lymphatic damage.

**Diagnosis**

Accurate diagnosis of lymphatic filariasis is essential for appropriate treatment and control measures. Several diagnostic approaches are available, each with specific applications depending on the stage of infection and purpose of testing.

**Microscopic Examination**

The traditional method for diagnosing lymphatic filariasis involves detecting circulating microfilariae in blood samples:

* Thick blood smears (20-60 μl) from finger-prick samples are examined microscopically.
* Blood collection timing is critical due to the periodicity of microfilariae circulation in the bloodstream.
* For W. bancrofti, which typically exhibits nocturnal periodicity, blood should be collected at night when microfilariae are more prevalent in peripheral blood.
* This method is relatively inexpensive and feasible at both individual and community levels for mapping endemicity and monitoring mass drug administration programs.

**Antigen Detection**

Immunochromatographic tests have revolutionized the diagnosis of W. bancrofti infection:

* The Alere Filariasis Test Strip (FTS) is a rapid diagnostic test recommended for mapping, monitoring, and transmission assessment surveys.
* FTS detects W. bancrofti antigen in human blood samples and has replaced the earlier Binax Now filariasis immunochromatographic test (ICT).
* This method allows for daytime testing, eliminating the need for night blood collection.

**Antibody Detection**

For Brugia infections:

* The Brugia Rapid point-of-care cassette test (BRT) manufactured by Reszon Diagnostics is used to detect IgG4 antibodies against Brugia species in human blood samplesd,d.
* This test is particularly useful during transmission assessment surveys in areas where Brugia species are endemic.

**Additional Diagnostic Approaches**

* Ultrasound examination may reveal the presence of adult worms in lymphatic vessels, often showing the characteristic "filarial dance sign."
* Molecular techniques such as polymerase chain reaction (PCR) can detect parasite DNA in blood samples, offering high sensitivity and specificity.
* Clinical evaluation of chronic manifestations such as lymphedema or hydrocele is important, particularly in endemic areas.

Each diagnostic method has specific applications depending on the phase of the disease, with antigen detection tests being particularly valuable for identifying infection before the development of chronic symptoms.

**TREATMENT**

The management of lymphatic filariasis encompasses both parasitological clearance and addressing the chronic manifestations of the disease.

**Antiparasitic Treatment**

The primary goal of antiparasitic treatment is to eliminate the adult worms and microfilariae from the infected individual:

1. **Diethylcarbamazine citrate (DEC)**: This is the drug of choice for lymphatic filariasis as it is both microfilaricidal and active against adult worms.
   * Dosage: 6mg/kg/day either as a 1-day or 12-day treatment course for adults and children older than 18 months.
   * The 1-day treatment is generally as effective as the 12-day regimen.
   * For tropical pulmonary eosinophilia (TPE), a longer treatment course of 14-21 days is typically recommended.
   * Side effects are usually mild and depend on the microfilarial load, including dizziness, nausea, fever, headache, or pain in muscles or joints.
   * Important contraindication: DEC should not be administered to patients who may also have onchocerciasis due to the risk of severe exacerbations of skin and eye involvement (Mazzotti reaction).
   * DEC should also be used with extreme caution in patients with high Loa loa microfilarial levels.
2. **Ivermectin**: Effective against microfilariae of W. bancrofti but has no effect on adult parasites.
3. **Combined therapy**: In some mass drug administration programs, combinations of albendazole with either DEC or ivermectin are used to enhance effectiveness.

**TREATMENT**

1. **Lymphedema management**:
   * Hygiene measures: Regular washing of affected limbs with soap and clean water
   * Elevation of affected limbs
   * Exercise to improve lymph flow
   * Compression bandaging or garments
   * Prompt treatment of secondary bacterial infections with antibiotics
   * Referral to specialized lymphedema therapists is recommended
2. **Hydrocele treatment**:
   * Surgical intervention (hydrocelectomy) may be necessary for men with hydrocele
   * Surgery should be performed by experienced surgeons familiar with the condition
3. **Psychological support**:
   * Addressing the social stigma and psychological impact of visible disfigurement
   * Support groups and community education

It's important to note that while antiparasitic treatment can clear the infection, it may not reverse the chronic changes in advanced lymphedema or hydrocele. Early treatment before permanent damage occurs provides the best outcomes.

**RECOMMENDATION AND PREVENTION**

Prevention of lymphatic filariasis focuses on interrupting transmission and protecting individuals from infection through various strategies:

**Individual Protection Measures**

1. **Vector avoidance**:
   * Use mosquito bed nets, particularly insecticide-treated nets, which have been shown to provide protection, especially for females.
   * Apply mosquito repellents on exposed skin during peak mosquito activity periods.
   * Wear long-sleeved clothing to minimize exposed skin, especially during evening and night hours when vector mosquitoes are most active.
2. **Environmental management**:
   * Eliminate or cover standing water sources around homes to reduce mosquito breeding sites.
   * Install screens on windows and doors to prevent mosquito entry into dwellings.
3. **Improved sanitation**:
   * Use latrines, which have demonstrated a protective effect against infection (OR = 0.5).
   * Proper waste disposal to reduce mosquito breeding habitats.

**RECOMMENDATION AND PREVENTION**

1. **Mass Drug Administration (MDA)**:
   * The World Health Organization recommends annual MDA with a combination of medicines in endemic areas to break transmission cycles.
   * Typically, this involves administering albendazole with either DEC or ivermectin to the entire eligible population in endemic areas.
2. **Vector control**:
   * Community-wide insecticide spraying to reduce mosquito populations.
   * Biological control methods, such as introducing larvivorous fish in breeding sites.
   * Environmental modification to eliminate vector breeding habitats.
3. **Screening and early treatment**:
   * Identification and treatment of infected individuals to reduce the reservoir of infection.
   * Particularly important for high-risk groups such as men engaged in outdoor activities like hunting or fishing at night.
4. **Health education**:
   * Community awareness programs about transmission, symptoms, and prevention.
   * Encouraging participation in MDA programs and adoption of protective behaviors.

**Global Elimination Strategy**

The WHO Global Programme to Eliminate Lymphatic Filariasis aims to eliminate LF as a public health problem through:

* Interrupting transmission through MDA to entire at-risk populations.
* Alleviating suffering through morbidity management and disability prevention for those already affected.
* The target is to achieve at least 65% coverage of the total population in endemic areas for 4-6 years.

These prevention strategies have proven effective, with several countries successfully eliminating lymphatic filariasis as a public health problem through consistent implementation of these approaches.

**PROGNOSIS**

The prognosis for individuals with lymphatic filariasis varies significantly depending on the stage of the disease, access to treatment, and management of complications.

**Early-Stage Infection**

For individuals with early or asymptomatic infection who receive prompt antiparasitic treatment:

* Excellent prognosis with complete clearance of microfilariae from the bloodstream
* Prevention of progression to chronic disease manifestations
* Normal life expectancy with minimal to no long-term effects

**Established Lymphedema**

For patients with established lymphedema:

* Antiparasitic treatment can kill the parasites but may not completely reverse existing lymphatic damage
* With consistent lymphedema management (hygiene, elevation, exercise, compression), symptoms can be significantly improved and progression slowed
* Without proper management, progressive worsening of lymphedema is likely, with increased risk of secondary infections and disability

**Advanced Disease**

For individuals with advanced elephantiasis or large hydrocele:

* Surgical interventions may provide relief for hydrocele
* Advanced lymphedema typically cannot be completely reversed but can be managed to improve quality of life
* Long-term disability is common, affecting mobility, employment, and social interaction
* Psychological effects including depression and social isolation may develop due to stigma

**Overall Long-term Outlook**

The global baseline estimate indicates that lymphatic filariasis has affected approximately 25 million men with hydrocele and over 15 million people with lymphedema, with at least 36 million people continuing to live with these chronic manifestations. These individuals require ongoing morbidity management and disability prevention services even after transmission is interrupted in their communities.

With proper management, most patients can lead productive lives despite chronic manifestations. However, the disease contributes significantly to disability-adjusted life years (DALYs) lost in endemic regions, with substantial socioeconomic impact on affected individuals, their families, and communities.

**POSSIBLE COMPLICATIONS**

Lymphatic filariasis can lead to numerous complications affecting physical health, mental wellbeing, and socioeconomic status:

**Physical Complications**

1. **Progressive lymphedema**: Chronic obstruction of lymphatic vessels leads to progressive swelling that can become massive and debilitating, particularly affecting the legs, arms, breasts, and genitalia.
2. **Elephantiasis**: The most severe form of lymphedema, characterized by enormous enlargement of affected limbs or parts with thickened, hardened, and fissured skin resembling an elephant's hide.
3. **Hydrocele**: Accumulation of fluid in the scrotal sac, which can become extremely large and painful, affecting mobility and sexual function in men.
4. **Recurrent bacterial and fungal infections**: The compromised lymphatic system and skin changes create an environment prone to secondary infections, leading to episodes of acute dermatolymphangioadenitis.
5. **Chyluria**: The presence of chyle (lymphatic fluid containing fat) in the urine, resulting from rupture of lymphatic vessels into the urinary tract.
6. **Tropical pulmonary eosinophilia**: A manifestation of occult filariasis characterized by coughing, wheezing, and shortness of breath.

**COMPLICATIONS**

1. **Social stigma**: The visible disfigurement often leads to stigmatization and social isolation.
2. **Psychological distress**: Depression, anxiety, and poor self-image are common among individuals with visible manifestations of the disease.
3. **Sexual dysfunction**: Particularly in men with hydrocele or genital lymphedema.
4. **Limited mobility**: Advanced lymphedema can severely restrict movement and daily activities.

**Socioeconomic Complications**

1. **Loss of income and productivity**: Physical limitations often prevent affected individuals from working, particularly in labor-intensive occupations common in endemic areas.
2. **Financial burden**: Costs associated with treatment, management of secondary infections, and lost wages contribute to poverty.
3. **Educational impact**: Children with the disease or those caring for affected family members may have reduced educational opportunities.
4. **Healthcare burden**: In endemic communities, lymphatic filariasis places significant strain on healthcare systems and resources.

These complications illustrate why lymphatic filariasis is not merely a medical condition but a complex socioeconomic issue that contributes to the cycle of poverty in endemic regions

. Addressing these complications requires a comprehensive approach that goes beyond treating the infection to include morbidity management, psychological support, and community education to reduce stigma.

**WHEN TO CONSULT A DOCTOR**

Seeking medical attention is crucial for proper diagnosis, treatment, and management of lymphatic filariasis. The following situations warrant medical consultation:

**After Travel to Endemic Areas**

Individuals who have lived in or traveled to regions where lymphatic filariasis is endemic should consult a healthcare provider if they experience:

* Unexplained fever or chills
* Lymph node enlargement or tenderness
* Pain or swelling in the groin, genital area, or limbs
* Skin rashes or unusual reactions following mosquito bites

**EARLY SYMTOMS**

Early intervention can prevent progression to more severe stages, so medical attention should be sought for:

* Recurrent episodes of swelling in extremities, particularly the legs
* Sensations of heaviness or tightness in limbs
* Decreased flexibility or mobility in affected limbs
* Hardening or thickening of the skin

**Acute Infectious Episodes**

Immediate medical care is necessary for:

* High fever with pain and redness over swollen areas
* Warm, tender, or painful lymph nodes
* Signs of skin infection in areas of lymphedema (redness, warmth, pain)
* Episodes of acute adenolymphangitis (inflammation of lymph vessels and nodes)

**Men with Scrotal Swelling**

Men should seek medical evaluation for:

* Any swelling in the scrotum, which could indicate hydrocele
* Discomfort or pain in the genital region
* Changes in the size or consistency of scrotal tissue

**Respiratory Symptoms**

Individuals with possible occult filariasis should consult a doctor for:

* Persistent coughing
* Recurrent wheezing or asthma-like symptoms
* Shortness of breath, particularly in combination with eosinophilia (high levels of a type of white blood cell)

**During or After Treatment**

Medical follow-up is important for:

* Side effects from antiparasitic medications
* Monitoring response to treatment
* Regular assessment of lymphedema or hydrocele management

**Psychological Support**

Medical consultation is also warranted for:

* Psychological distress related to the physical manifestations of the disease
* Difficulty coping with stigma or social isolation
* Depression or anxiety associated with chronic illness

Given the chronic nature of lymphatic filariasis and the potential for progressive disability, establishing a relationship with healthcare providers familiar with the disease is crucial for optimal long-term management.

**DIFFERENTIAL DIAGNOSIS**

When evaluating a patient with suspected lymphatic filariasis, healthcare providers must consider various conditions that can present with similar clinical manifestations. The differential diagnosis varies depending on the stage and presentation of the disease:

**For Lymphedema/Elephantiasis**

1. **Congenital or hereditary lymphedema**: Conditions like Milroy syndrome present with lymphedema but have no infectious cause.
2. **Nonfilarial elephantiasis**: Podoconiosis (non-filarial elephantiasis) is caused by long-term barefoot exposure to irritant soils in volcanic regions.
3. **Post-surgical or post-radiation lymphedema**: Common after breast cancer surgery with lymph node removal or radiation therapy.
4. **Venous insufficiency**: Causes edema but typically affects both legs and is associated with skin changes different from those in filariasis.
5. **Bacterial or fungal lymphadenitis**: Infections such as sporotrichosis from Sporothrix schenckii can cause lymphatic involvement and swelling.
6. **Recurrent streptococcal lymphadenitis**: Can cause recurrent episodes of inflammation and progressive lymphedema.

**For Scrotal Swelling/Hydrocele**

1. **Congenital hydrocele**: Present from birth and unrelated to parasitic infection.
2. **Epididymal cysts**: Fluid-filled sacs that develop in the epididymis.
3. **Testicular or scrotal carcinoma**: Malignancies that can present with scrotal swelling.
4. **Inguinal hernia**: Protrusion of abdominal contents into the scrotum.
5. **Lymphosarcoma**: Malignancy of the lymphatic system that can cause swelling.

**For Occult Filariasis (Tropical Pulmonary Eosinophilia)**

1. **Allergic bronchopulmonary aspergillosis**: Hypersensitivity reaction to Aspergillus fungi.
2. **Systemic vasculitis**: Inflammatory conditions affecting blood vessels.
3. **Chronic eosinophilic pneumonia**: Characterized by pulmonary infiltrates and peripheral eosinophilia.
4. **Idiopathic hypereosinophilic syndrome**: Persistent unexplained eosinophilia with organ involvement.
5. **Asthma**: Shares features of wheezing and respiratory distress.

**For Other Manifestations**

**Septic arthritis**: For patients presenting with joint swelling and pain.

**Bacterial breast abscess**: When breast involvement is prominent.

**Idiopathic or poststreptococcal glomerulonephritis**: For renal manifestations.

**Other filarial diseases**: Onchocerciasis (river blindness), loiasis (African eye worm), and mansonellosis can have overlapping symptoms.

Careful clinical evaluation, epidemiological history, and appropriate laboratory testing are essential for distinguishing lymphatic filariasis from these conditions. In endemic areas, a history of residence or travel to regions where the disease is common is a significant diagnostic clue.

**STATISTICS AND EPIDEMIOLOGY DATA**

Lymphatic filariasis represents a significant global health burden, particularly in tropical and subtropical regions. Understanding the epidemiology of this disease is crucial for effective control and elimination efforts.

**Global Prevalence**

* As of 2023, approximately 657 million people in 39 countries live in areas requiring preventive chemotherapy to stop the spread of lymphatic filariasis infection.
* The global baseline estimate indicates approximately 25 million men affected by hydrocele and over 15 million people with lymphedema, with at least 36 million people continuing to live with these chronic disease manifestations.

**GEOGRAPHIC DISTRIBUTION**

* Lymphatic filariasis is endemic across tropical and subtropical regions in Africa, Asia, the Western Pacific, and parts of the Caribbean and South America.
* W. bancrofti, responsible for 90% of cases, has the widest geographic distribution.
* Brugia malayi is found primarily in parts of South and Southeast Asia, while Brugia timori has a more limited distribution in certain Pacific islands.

**Affected Demographics**

* Age patterns: Infection rates typically increase with age up to approximately 20 years and then stabilize, indicating cumulative exposure over time.
* Gender disparities: Males generally have a higher risk of infection than females, with studies showing men have approximately twice the risk of antigenemia (OR = 2.0) and over five times the risk of microfilaremia (OR = 5.4).
* Occupational factors: People engaged in outdoor activities like hunting and fishing, particularly at night, have an increased risk of infection (OR = 1.5).
* Socioeconomic factors: The disease disproportionately affects poor communities with limited access to healthcare and sanitation facilities.

**TRANSMISSION DYNAMICS**

* Vector species vary by region: Culex mosquitoes primarily transmit the disease in urban and semi-urban areas, Anopheles in rural settings, and Aedes in Pacific islands.
* Transmission patterns differ by gender and age: Men often acquire infection in high transmission areas outside villages, while children and women are more commonly infected in areas with lower transmission inside or near villages.
* Household clustering: There is a strong household effect on infection risk, with intraclass correlation coefficients of 0.24 for antigenemia and 0.49 for microfilaremia, indicating significant clustering of cases within households.

**Elimination Efforts**

The World Health Organization's Global Programme to Eliminate Lymphatic Filariasis aims to eliminate LF as a public health problem through mass drug administration and morbidity management. Progress has been made, with several countries successfully eliminating the disease as a public health concern through consistent implementation of preventive chemotherapy and other interventions.

Despite these efforts, lymphatic filariasis remains a significant cause of disability and suffering, contributing to the perpetuation of poverty in affected communities through reduced productivity, increased healthcare costs, and social stigma.

**CONCLUSION**

Lymphatic filariasis represents a significant global health challenge that extends beyond its medical implications to encompass profound social and economic consequences for affected individuals and communities. Despite being classified as a neglected tropical disease, it affects hundreds of millions of people worldwide, with tens of millions suffering from its chronic, debilitating manifestations.

The disease's complex lifecycle, involving both mosquito vectors and human hosts, presents challenges for control and elimination. However, significant progress has been made through the WHO's Global Programme to Eliminate Lymphatic Filariasis, which combines preventive chemotherapy through mass drug administration with morbidity management and disability prevention strategies.

Understanding the epidemiology, clinical manifestations, and management of lymphatic filariasis is essential for healthcare providers working in endemic regions. Early detection and prompt treatment can prevent progression to chronic, disabling conditions like elephantiasis and hydrocele. For those already affected by these chronic manifestations, appropriate morbidity management can significantly improve quality of life.

Moving forward, continued commitment to elimination efforts, research into more effective diagnostic tools and treatments, and addressing the social stigma associated with the disease will be crucial. The elimination of lymphatic filariasis would not only alleviate unnecessary suffering but also contribute significantly to poverty reduction in endemic regions, aligning with broader global health and development goals.

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**HELMINTHIC (WORM) INFECTION:**

**MEDICAL AND COMMON NAMES**

**Medical term:** Helminthiasis or helminthic infection

**Common names:** Worm infection, parasitic worm infection

Specific infections have names based on the worm type, e.g., ascariasis (roundworm), schistosomiasis (blood fluke), tapeworm infection (cestodiasis), filariasis (caused by filarial roundworms)

**DESCRIPTION**

Helminthic infections are diseases caused by parasitic worms known as helminths. Helminths are large, multicellular organisms that live either freely or as parasites in humans and other hosts. The major groups of helminths infecting humans include:

**Nematodes (roundworms):** Cylindrical, tubular worms with complete digestive tracts; sexually distinct males and females. Examples include *Ascaris lumbricoides* (common roundworm), *Trichuris trichiura* (whipworm), and hookworms (*Necator americanus*, *Ancylostoma duodenale*).

**Cestodes (tapeworms):** Long, flat, segmented worms with a scolex (head) for attachment; hermaphroditic. They absorb nutrients through their tegument. Examples: *Taenia saginata* (beef tapeworm), *Taenia solium* (pork tapeworm).

**Trematodes (flukes):** Flat, leaf-shaped worms with suckers for attachment; mostly hermaphroditic except blood flukes which are dioecious. Examples: *Schistosoma mansoni*, *Fasciola hepatica* (liver fluke).

Helminths inhabit various human organs including intestines, blood vessels, lymphatics, lungs, and tissues, disrupting nutrient absorption and causing a range of clinical symptoms.

**CAUSES AND RISK FACTORS**

**Causes:** Infection occurs through ingestion of helminth eggs or larvae (fecal-oral route), skin penetration by larvae, or via vectors (e.g., mosquitoes for filarial worms).

**Risk factors:**

Poor sanitation and hygiene (contaminated soil, water, or food)

Walking barefoot in endemic areas (hookworm infection)

Consumption of undercooked or raw meat or fish (tapeworms, flukes)

Living in tropical/subtropical regions with high endemicity

Occupational exposure (farmers, fishermen)

Lack of access to clean water and proper sanitation facilities

Children and males often have higher infection rates due to behavior and exposure.

**SYMPTOMS**

Symptoms vary widely depending on the helminth species, infection intensity, and affected organs:

**Intestinal infections:** Abdominal pain, diarrhea, malnutrition, anemia, weight loss, intestinal obstruction (heavy worm burden)

**Lymphatic filariasis:** Lymphedema, elephantiasis, hydrocele

**Schistosomiasis:** Blood in urine or stool, liver enlargement, fibrosis

**Tapeworm infections:** Usually asymptomatic; may cause abdominal discomfort, weight loss, or cystic lesions in tissues (cysticercosis)

**Other systemic symptoms:** Fever, fatigue, eosinophilia, allergic reactions

**Severe cases:** Organ damage, growth retardation in children, neurological symptoms (neurocysticercosis).

**DIAGNOSIS**

**Microscopy:** Identification of eggs, larvae, or adult worms in stool, urine, blood, or tissue samples. Stool examination is common for intestinal helminths.

**Serological tests:** Detection of antibodies or antigens for specific helminths (e.g., ELISA for schistosomiasis).

**Molecular methods:** PCR-based assays for parasite DNA detection.

**Imaging:** Ultrasound, CT, or MRI for tissue-invasive helminths (e.g., cysticercosis).

**Clinical evaluation:** Based on symptoms and epidemiological history, especially in endemic areas.

**TREATMENT**

**Anthelmintic drugs:**

*Albendazole* and *Mebendazole* are broad-spectrum agents effective against many nematodes.

*Praziquantel* is the drug of choice for trematodes and cestodes.

*Ivermectin* is effective against certain nematodes like *Strongyloides* and filarial worms.

*Diethylcarbamazine* for filariasis.

Treatment regimens vary by species and infection severity.

Supportive care for complications such as anemia or secondary infections may be necessary.

**PREVENTION AND PREVENTION**

Improve sanitation and hygiene: Use latrines, proper disposal of feces.

Wash hands with soap, especially before eating and after defecation.

Drink clean, safe water; avoid contaminated food.

Wear shoes to prevent skin penetration by larvae (hookworms).

Cook meat and fish thoroughly to kill larvae.

Mass drug administration programs in endemic areas to reduce community worm burden.

Vector control measures for filarial worms (mosquito nets, insecticides).

**PROGNOSIS**

* Generally good with timely diagnosis and treatment.
* Chronic or heavy infections can cause lasting organ damage, malnutrition, and disability.
* Early treatment prevents complications and transmission.
* Some tissue-invasive infections (e.g., neurocysticercosis) may require prolonged or repeated therapy and have variable outcomes.

**POSSIBLE COMPLICATIONS**

* Intestinal obstruction (heavy roundworm infection)
* Anemia (hookworms)
* Nutritional deficiencies and growth retardation in children
* Chronic lymphedema and elephantiasis (filarial worms)
* Organ damage (liver fibrosis in schistosomiasis)
* Neurological impairment (neurocysticercosis)
* Secondary bacterial infections due to skin breaks or immune suppression.

**WHEN CONSULT A DOCTOR**

Persistent gastrointestinal symptoms (abdominal pain, diarrhea, blood in stool)

Unexplained swelling or lymphedema

Signs of anemia or malnutrition

Neurological symptoms such as seizures or headaches in endemic areas

After travel to endemic regions with suspicious symptoms

For preventive screening in high-risk populations.

**DIAGNOSIS**

Other causes of gastrointestinal symptoms (bacterial, viral infections)

Other causes of lymphedema (cancer, lymphatic obstruction)

Allergic or autoimmune diseases causing eosinophilia

Other parasitic infections (protozoa)

Non-infectious causes of neurological symptoms (tumors, epilepsy).

**STATISTICS AND EPIDEMIOLOGY DATA**

Over 1.5 billion people worldwide are infected with soil-transmitted helminths, primarily in tropical and subtropical regions.

The most common species globally are *Ascaris lumbricoides*, *Trichuris trichiura*, and hookworms.

Children are disproportionately affected, with high prevalence in areas with poor sanitation.

Helminth infections are among the most common infections worldwide and a leading cause of nutritional and developmental problems in endemic regions.

Mass drug administration programs target over 600 million children annually to reduce morbidity.

Filarial infections affect over 120 million people globally, with significant disability burden.

Tapeworm and fluke infections are regionally endemic, often linked to dietary habits and water sources .

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**CRYPTOSPORIDIOSIS - CRYPTOSPORIDIUM SPP., WATERBORNE INFECTION**

**Medical and common names**

* Medical name: Cryptosporidiosis
* Common name: Crypto infection

**DESCRIPTION**

Cryptosporidiosis is an intestinal infection caused by the protozoan parasite *Cryptosporidium* spp. It is a waterborne disease transmitted primarily through ingestion of oocysts in contaminated water or food, or by contact with infected individuals or animals. The parasite infects the epithelial cells of the gastrointestinal tract, leading to diarrhea and other gastrointestinal symptoms.

**CAUSES AND RISK FACTORS**

* **Cause:** Infection by *Cryptosporidium* species, mainly *Cryptosporidium parvum* and *Cryptosporidium hominis*.
* **Transmission:** Fecal-oral route via contaminated water (drinking or recreational), food, or direct contact with infected hosts.
* **Risk Factors:**
  + Young children (especially aged 1–4 years) and adults aged 15–44 years, with females in this age group particularly affected.
  + Immunocompromised individuals (e.g., HIV/AIDS patients).
  + Low socioeconomic status, food inadequacy, and poverty increase risk due to limited access to sanitation and clean water.
  + Non-white races, immigrants, and those living in rural or livestock-dense areas have higher exposure risk.
  + Occupational exposure, such as agricultural work with livestock contact, elevates risk.

**Symptoms**

* Watery diarrhea (often profuse)
* Abdominal cramps and pain
* Nausea and vomiting
* Low-grade fever
* Weight loss and dehydration in severe cases  
  Symptoms usually last 1–2 weeks but can be prolonged in immunocompromised patients[2](https://wwwnc.cdc.gov/travel/yellowbook/2024/infections-diseases/cryptosporidiosis).

**DIAGNOSIS METHODS**

* Microscopic identification of oocysts in stool samples using acid-fast staining.
* Immunofluorescence assays and enzyme-linked immunosorbent assays (ELISA) for antigen detection.
* Polymerase chain reaction (PCR) for species identification and confirmation.
* Serological tests may indicate exposure but are less commonly used for acute diagnosis.

**TREATMENT**

* In immunocompetent individuals, cryptosporidiosis is often self-limiting and may not require specific treatment.
* Nitazoxanide is the only FDA-approved antiparasitic drug effective against cryptosporidiosis in immunocompetent patients.
* Supportive care includes rehydration and electrolyte replacement.
* In immunocompromised patients, managing the underlying immune deficiency is crucial, and antiparasitic treatment may be less effective.

**Prevention**

* Ensure safe drinking water by using filtration and disinfection methods.
* Practice good hand hygiene, especially after using the toilet and before handling food.
* Avoid swallowing water during recreational activities in lakes, rivers, or pools.
* Avoid contact with infected individuals or animals.
* Improve sanitation and food safety practices, particularly in resource-poor settings.

**PROGNOSIS**

* Generally good in healthy individuals with full recovery.
* In immunocompromised or malnourished patients, cryptosporidiosis can be severe, chronic, and potentially life-threatening.

**POSSIBLE COMPLICATIONS**

* Severe dehydration
* Chronic diarrhea leading to malnutrition and weight loss
* Disseminated infection in immunocompromised hosts
* Secondary infections due to weakened immunity

**When to CONSULT a Doctor**

* Persistent diarrhea lasting more than a few days
* Signs of dehydration (dizziness, dry mouth, decreased urination)
* Severe abdominal pain or bloody stools
* Immunocompromised individuals with any symptoms of infection

**DIAGNOSIS**

* Other causes of infectious diarrhea such as:
  + Giardia lamblia infection
  + Entamoeba histolytica infection
  + Bacterial gastroenteritis (e.g., Salmonella, Shigella)
  + Viral gastroenteritis (e.g., norovirus, rotavirus)

**STATISTICS AND EPIDEMIOLOGY**

* In the United States, approximately 750,000 cases occur annually.
* Highest incidence in young children aged 0–4 years and adults aged 15–44 years, with females more affected in the latter group.
* Prevalence varies by region and socioeconomic factors; higher rates in low-income, marginalized populations and areas with poor sanitation.
* In the Americas, prevalence ranges from about 7% to 28% depending on rural or urban settings and age groups.
* Globally, *Cryptosporidium* contamination in soil and water is estimated at about 8.13%, indicating widespread environmental presence.

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**CRYPTOSPORIDIOSIS**

Cryptosporidiosis is a common, waterborne diarrheal disease caused by *Cryptosporidium* spp. It is especially dangerous for immunocompromised individuals and young children. Prevention relies on good hygiene and safe water practices, while treatment focuses on supportive care and, in some cases, antiparasitic medication. The disease is globally prevalent and can cause significant morbidity, particularly in vulnerable populations.

* **Medical name:** Cryptosporidiosis
* **Common names:** Crypto

The infection is caused by protozoan parasites of the genus *Cryptosporidium*, with *Cryptosporidium parvum* and *Cryptosporidium hominis* being the main species affecting humans

**DESCRIPTION**

Cryptosporidiosis is a diarrheal disease caused by *Cryptosporidium* spp., a protozoan parasite that infects the gastrointestinal tract of humans and various animals. It is a significant waterborne disease, as the parasite is resistant to standard chlorination and can survive in recreational and drinking water sources. The infection is zoonotic, meaning it can be transmitted between animals and humans.

**CAUSES AND RISK FACTORS**

**Causes:**

* Ingestion of *Cryptosporidium* oocysts from contaminated water, food, or surfaces.
* Fecal-oral transmission, including person-to-person and animal-to-person contact.

**Risk Factors:**

* Drinking or swimming in contaminated water (lakes, pools, streams)
* Close contact with infected individuals or animals
* Young children, especially in daycare settings
* Immunocompromised individuals (e.g., people with HIV/AIDS, transplant recipients)
* Travelers to areas with poor sanitation

**SYMPTOMS**

* Watery diarrhea (can be severe and persistent)
* Abdominal cramps and pain
* Nausea and vomiting
* Weight loss
* Fever
* Loss of appetite
* Weakness and dehydration

Symptoms typically begin 2–10 days after exposure and last about 1–2 weeks in healthy individuals but can persist much longer in immunocompromised people.

**DIAGNOSIS METHODS**

* **Stool sample tests:** Multiple samples may be needed due to intermittent shedding of the parasite.
  + Microscopy with special stains (e.g., modified Ziehl-Neelsen)
  + Immunofluorescence assays
  + Enzyme immunoassays for fecal antigen detection
  + Molecular tests (PCR) for parasite DNA
* **Intestinal biopsy:** Rarely, used if stool tests are inconclusive.

**TREATMENT OPTIONS**

* **Supportive care:** Mainstay for most patients; includes oral or intravenous rehydration and electrolyte replacement.
* **Antidiarrheal medications:** Such as loperamide, may be used to control symptoms.
* **Antiprotozoal medication:** Nitazoxanide is FDA-approved for immunocompetent patients older than one year.
* **Immunocompromised patients:** Management of the underlying immune deficiency (e.g., antiretroviral therapy for HIV) is crucial. Other drugs (paromomycin, azithromycin, rifamycins) have variable efficacy.
* **Dietary advice:** Avoid lactose-containing foods during acute illness, as temporary lactose intolerance can occur.

**RECOMMENDATION AND PREVENTION**

* Practice good hand hygiene, especially after using the toilet, changing diapers, or handling animals.
* Avoid swallowing water from lakes, pools, or streams.
* Drink only treated or filtered water; filters must have pores smaller than 1 micron to remove oocysts.
* Wash fruits and vegetables thoroughly.
* Avoid contact with infected individuals’ stool.
* Immunocompromised individuals should consider boiling water for drinking.

**PROGNOSIS**

* **Immunocompetent individuals:** Usually self-limited; symptoms resolve within two weeks, though some may have symptoms for up to a month.
* **Immunocompromised individuals:** Can develop chronic, severe, or even life-threatening disease. In AIDS patients, symptoms may persist for months or years and may never be fully cured without immune restoration.

**POSSIBLE COMPLICATIONS**

* Severe dehydration
* Malnutrition and weight loss
* Inflammation of the bile ducts, gallbladder, liver (hepatitis), or pancreas
* Malabsorption of nutrients
* Wasting syndrome (extreme thinness and weakness)
* Growth problems in children

**WHEN TO CONSULT A DOCTOR**

* Diarrhea lasting more than a few days, especially with signs of dehydration
* If you have a weakened immune system and develop diarrhea
* If symptoms are severe or persistent

**Differential Diagnosis**

Other causes of diarrhea to consider include:

* Campylobacter infection
* Cyclospora infection
* Escherichia coli (E. coli) infection
* Giardiasis

**STATISTICS OR EPIDEMIOLOGY DATA**

* In the United States, there are over 700,000 cases annually.
* Cryptosporidiosis is the second most common cause of diarrhea in children after rotavirus.
* In Europe, the notification rate was 1.8 confirmed cases per 100,000 population in 2021.
* The disease affects all age groups but is particularly problematic for young children and immunocompromised individuals.

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**GIARDIASIS**

**Medical and Common Names**  
Giardiasis is caused by the protozoan parasite *Giardia duodenalis*, also known as *Giardia lamblia* or *Giardia intestinalis*.

**DESCRIPTION**  
Giardiasis is an intestinal infection caused by the flagellated protozoan *Giardia duodenalis*. The parasite attaches to the mucosa of the duodenum and proximal jejunum and multiplies there. It can cause symptoms ranging from asymptomatic carriage to chronic malabsorption and diarrhea. The parasite forms environmentally resistant cysts that are excreted in feces and transmitted via the fecal-oral route.

**CAUSES AND RISK FACTORS**  
The infection is primarily waterborne. Humans become infected by ingesting water contaminated with *Giardia* cysts, often from sewage-contaminated sources. Other transmission routes include eating uncooked fruits or vegetables washed with contaminated water, direct person-to-person contact, contact with infected animals, and poor hand hygiene after contact with feces. The cysts can survive in cold, chlorinated water for up to two months, making water supplies a common source of outbreaks. Risk groups include children in daycare, travelers to developing countries, campers drinking untreated water, and men who have sex with men.

**SYMPTOMS**  
Symptoms typically include watery diarrhea, stomach cramps, bloating, nausea, and intermittent flatulence. Some infections are asymptomatic, while others may lead to chronic malabsorption and weight loss. Symptoms may persist even after the parasite is cleared.

**DIAGNOSIS METHODS**  
Diagnosis is made by identifying *Giardia* cysts or trophozoites in fresh stool samples or duodenal contents. More sensitive methods include enzyme immunoassays for *Giardia* antigen and molecular tests detecting parasite DNA in stool. Multiple stool samples may be needed due to intermittent shedding.

**TREATMENT OPTIONS**  
Treatment involves oral antiparasitic medications such as metronidazole, tinidazole, secnidazole, or nitazoxanide. These drugs are effective in clearing the infection and resolving symptoms.

**RECOMMENDATION AND PREVENTION**   
Prevention requires:

* Ensuring safe drinking water through proper treatment or boiling.
* Practicing good hand hygiene, especially after contact with feces.
* Avoiding consumption of untreated water from lakes, streams, or wells.
* Hygienic food preparation and washing fruits and vegetables with safe water.
* Proper sanitation and sewage disposal to reduce environmental contamination.

**PROGNOSIS**  
With appropriate treatment, giardiasis generally resolves without long-term complications. However, some patients may experience prolonged symptoms even after parasite clearance.

**POSSIBLE COMPLICATIONS**  
Complications can include chronic malabsorption, weight loss, and nutritional deficiencies if the infection persists untreated. Rarely, post-infectious irritable bowel syndrome may develop.

**WHEN TO CONSULT A DOCTOR**  
Medical attention is advised when experiencing persistent diarrhea, abdominal pain, or signs of dehydration, especially after travel to endemic areas or exposure to potentially contaminated water.

**DIFFERENTIAL DIAGNOSIS**  
Giardiasis symptoms overlap with other causes of diarrhea and malabsorption such as bacterial gastroenteritis, other parasitic infections (e.g., amoebiasis), inflammatory bowel disease, and lactose intolerance.

**STATISTICS AND EPIDEMIOLOGY**  
Giardiasis is common worldwide, especially in areas with poor sanitation. In developing countries, over 20% of the population may have ongoing infection. In the United States, incidence is about 1-2 cases per 10,000 people annually, with over 15,000 reported cases in 2018. Children are three times more likely to be infected than adults. Travelers to endemic regions, daycare attendees, and campers are at higher risk.

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**AMOEBIASIS (ENTAMOEBA HISTOLYTICA) — WATERBORNE INFECTION**

**Medical and Common Names**

* Medical name: Amoebiasis caused by *Entamoeba histolytica*
* Common names: Amebic dysentery, amoebic colitis, amoebiasis

**DESCRIPTION**  
Amoebiasis is an intestinal infection caused by the protozoan parasite *Entamoeba histolytica*. The parasite exists in two forms: the infective cyst stage and the active trophozoite stage. Infection occurs when mature cysts are ingested via contaminated food or water. After excystation in the small intestine, trophozoites colonize the colon and cecum, where they may remain noninvasive or invade the intestinal mucosa, causing tissue destruction (histolytic activity). The disease spectrum ranges from asymptomatic colonization to severe amoebic dysentery and extraintestinal manifestations such as liver abscesses.

**CAUSES AND RISK FACTORS**

* Transmission occurs primarily through ingestion of mature cysts in food or water contaminated with human feces.
* Contamination sources include fecally polluted drinking water, uncooked vegetables and fruits washed with contaminated water, and food handled with poor hygiene.
* Mechanical vectors like houseflies and cockroaches can also spread cysts to food.
* Use of human feces as fertilizer in some regions increases risk.
* Risk factors include poor sanitation, inadequate water treatment, crowded living conditions, travel to endemic areas, and certain sexual practices (e.g., oral-anal contact).

**SYMPTOMS**

* Many infections are asymptomatic.
* Symptomatic intestinal disease presents with abdominal pain, diarrhea, and dysentery (bloody stools).
* Other symptoms: weight loss, fatigue, and fever in invasive cases.
* Extraintestinal disease may cause liver abscess, presenting with fever, right upper quadrant abdominal pain, and hepatomegaly. Rarely, lungs or brain may be involved.

**DIAGNOSIS METHODS**

Microscopic identification of cysts or trophozoites in stool samples.

Antigen detection assays and PCR tests improve sensitivity and specificity.

Serology and imaging (ultrasound, CT) are used for extraintestinal disease like liver abscess.

Multiple stool samples may be required due to intermittent shedding of cysts.

**TREATMENT OPTIONS**

* Metronidazole or tinidazole to eradicate trophozoites in tissues.
* Followed by luminal agents such as paromomycin or diloxanide furoate to eliminate cysts in the intestine and prevent relapse.
* Treatment of liver abscess may require drainage in severe cases.

**RECOMMENDATION AND PREVENTION**

* Drink only treated or boiled water.
* Avoid raw fruits and vegetables unless peeled or washed with safe water.
* Practice good hand hygiene, especially after using the toilet and before eating.
* Improve sanitation and sewage disposal to reduce environmental contamination.
* Control vectors such as flies and cockroaches.
* Avoid using untreated human feces as fertilizer.

**PROGNOSIS**

* Most intestinal infections resolve with treatment without sequelae.
* Untreated invasive disease can cause serious complications and death.
* Liver abscess has a good prognosis with timely therapy but can be fatal if untreated.

**POSSIBLE COMPLICATIONS**

* Fulminant colitis with perforation and peritonitis.
* Amoeboma (granulomatous mass in colon).
* Amoebic liver abscess and rarely lung or brain abscesses.
* Chronic malabsorption and weight loss in prolonged infections.

**WHEN TO SEE A DOCTOR**

* Persistent diarrhea, especially with blood or mucus.
* Abdominal pain or tenderness.
* Fever with right upper quadrant pain suggesting liver involvement.
* After travel to endemic areas or exposure to unsafe water or food.

**DIFFERENTIAL DIAGNOSIS**

* Bacterial dysentery (e.g., *Shigella*, *Salmonella*).
* Other parasitic infections (e.g., *Giardia*, *Balantidium coli*).
* Inflammatory bowel diseases (Crohn’s disease, ulcerative colitis).
* Colorectal cancer or ischemic colitis in chronic cases.

**STATISTICS AND EPIDEMIOLOGY**

* Amoebiasis is a major parasitic cause of morbidity and mortality worldwide, especially in developing countries with poor sanitation.
* It is the second leading cause of parasite-related deaths globally.
* Endemic in tropical and subtropical regions with inadequate water treatment.
* Infection rates can exceed 20% in some populations.
* Both sexes and all ages are affected, but children and immunocompromised individuals are at higher risk.
* Increasing recognition of sexual transmission in developed countries, particularly among men who have sex with men.

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**HIV (HUMAN IMMUNODEFICIENCY VIRUS)**

Infectious diseases are illnesses caused by pathogenic microorganisms such as viruses, bacteria, fungi, and parasites that invade the body and disrupt normal function. Some infections are especially complex due to their chronicity, severity, or the challenges they pose in diagnosis and treatment. Among these, HIV and tropical diseases are particularly significant.

HIV (Human Immunodeficiency Virus)

**DESCRIPTION**

Human immunodeficiency virus (HIV) is a virus that attacks and gradually destroys the body’s immune system, specifically targeting CD4+ T cells (a type of white blood cell essential for immune defense). As the immune system weakens, the body becomes more vulnerable to infections and certain cancers that it would normally be able to fight off.

HIV is mainly transmitted through the exchange of certain body fluids-blood, semen, vaginal fluids, rectal fluids, and breast milk-from an infected person. The most common routes of transmission include unprotected sex, sharing injection drug equipment, and from mother to child during pregnancy, childbirth, or breastfeeding.

Without treatment, HIV infection usually progresses over several years to acquired immunodeficiency syndrome (AIDS), the most advanced stage of HIV infection, characterized by a severely weakened immune system and the presence of specific opportunistic infections or cancers. Early symptoms of HIV can resemble the flu, but many people may not notice symptoms for years while the virus continues to damage their immune system.

**CAUSES AND TRANSMISSION**

Human Immunodeficiency Virus (HIV) is caused by a retrovirus that primarily targets and destroys CD4+ T-helper lymphocytes, crucial cells in the immune system, leading to immune suppression and increased vulnerability to infections and certain cancers.

HIV is caused by infection with either HIV-1 or HIV-2 viruses, with HIV-1 being the most common and virulent globally, while HIV-2 is less transmissible and mostly confined to West Africa.

The virus is a retrovirus that integrates its genetic material into host immune cells, replicating and progressively destroying them.

Transmission of HIV

HIV is transmitted through direct contact with certain body fluids from an infected person. The main routes include:

Sexual contact: Unprotected vaginal, anal, or oral sex can transmit HIV when mucous membranes are exposed to infected semen, vaginal fluids, or rectal fluids. Transmission risk increases with any cuts or sores on the skin or mucous membranes.

Blood exposure: Sharing needles or syringes for drug use, accidental needle sticks in healthcare settings, or transfusion of contaminated blood or blood products can transmit HIV.

Mother-to-child transmission: HIV can be passed from an infected mother to her child during pregnancy, childbirth, or breastfeeding.

Other medical exposures: Use of inadequately sterilized medical instruments or transplantation of infected organs or tissues can also transmit HIV, though these are less common routes.

HIV is not transmitted by casual contact such as touching, hugging, sharing utensils, or through saliva, tears, or sweat. Kissing is generally not a risk unless both partners have significant open sores or bleeding gums.

**DIAGNOSIS**

HIV diagnosis is primarily based on detecting the virus or the body's immune response to it through various laboratory tests. The main types of HIV tests include:

Antigen/antibody tests: These detect both HIV antigens (such as p24 protein, which appears early after infection) and antibodies produced by the immune system in response to HIV. They are typically performed on blood samples and can detect infection 2 to 6 weeks after exposure.

Antibody tests: These tests look for antibodies to HIV in blood or saliva, often used in rapid tests including home self-tests. Antibodies may take 3 to 12 weeks to develop to detectable levels, so these tests may not identify very recent infections.

Nucleic acid tests (NATs): These detect the presence of HIV RNA or DNA directly in the blood, identifying infection as early as 10 days after exposure. NATs are more sensitive in early infection and are used when recent exposure is suspected or for early infant diagnosis.

Additional diagnostic methods include:

Enzyme-linked immunosorbent assay (ELISA): A laboratory test detecting antigen-antibody complexes with about 90% sensitivity, often followed by confirmatory testing like immunoblot (Western blot) for higher accuracy (up to 99.9%).

Polymerase chain reaction (PCR): Used to detect and quantify HIV RNA or DNA, especially useful in early infection and monitoring viral load for treatment response.

**TREATMENT OF HIV**

Testing accuracy depends on the timing relative to exposure due to the "window period" when the virus is present but antibodies or antigens are not yet detectable. Negative results during this period require follow-up testing weeks to months later for confirmation.

HIV treatment centers on antiretroviral therapy (ART), which involves taking a combination of HIV medicines to suppress the virus and prevent disease progression.

Key Points of HIV Treatment

Antiretroviral Therapy (ART): ART is recommended for everyone diagnosed with HIV regardless of disease stage or symptoms. It usually combines two or more drugs from different classes to effectively stop the virus from replicating.

Goals of ART: The main goals are to reduce the viral load to undetectable levels, restore and preserve immune function (measured by CD4 cell count), reduce HIV-related morbidity and mortality, and prevent HIV transmission.

Common Drug Classes in ART:

Nucleoside/nucleotide reverse transcriptase inhibitors (NRTIs): Block viral replication by interfering with reverse transcriptase (e.g., tenofovir, emtricitabine, lamivudine).

Non-nucleoside reverse transcriptase inhibitors (NNRTIs): Also inhibit reverse transcriptase but by a different mechanism (e.g., efavirenz, doravirine).

Integrase strand transfer inhibitors (INSTIs): Block the integrase enzyme, preventing viral DNA integration into host cells (e.g., dolutegravir, raltegravir).

Protease inhibitors (PIs): Inhibit protease enzyme needed for viral maturation (e.g., darunavir, lopinavir).

Entry and fusion inhibitors: Prevent HIV from entering host cells, though less commonly used.

Preferred Initial Regimens: Typically include two NRTIs combined with an INSTI or sometimes an NNRTI or boosted PI. Examples include dolutegravir with lamivudine and tenofovir, or single-tablet regimens like bictegravir/tenofovir alafenamide/emtricitabine.

Treatment Monitoring: Viral load and CD4 counts are regularly checked to assess treatment efficacy. Achieving an undetectable viral load usually occurs within 6 months of starting ART.

Adherence and Drug Interactions: Strict adherence to ART is critical to prevent resistance. Some HIV drugs interact with other medications and supplements, so patients should always consult healthcare providers before starting new medicines.

Emergency Treatments:

Post-exposure prophylaxis (PEP): ART taken within 72 hours after potential HIV exposure for 28 days to prevent infection.

Pre-exposure prophylaxis (PrEP): Daily ART for high-risk individuals to reduce infection risk (not treatment but prevention).

PREVENTION OF HIV

Prevention of HIV focuses on reducing exposure to the virus and minimizing transmission risks through multiple strategies:

**RECOMMENDATION AND PREVENTION METHODS**

Consistent and Correct Condom Use: Using male or female condoms correctly every time during vaginal, anal, or oral sex significantly reduces the risk of HIV transmission by blocking contact with infected bodily fluids.

Pre-Exposure Prophylaxis (PrEP): PrEP is a daily antiretroviral medication taken by people at high risk of HIV infection (e.g., those with HIV-positive partners or engaging in high-risk behaviors). When taken as prescribed, PrEP is highly effective at preventing HIV acquisition.

Post-Exposure Prophylaxis (PEP): PEP involves taking antiretroviral drugs within 72 hours after potential HIV exposure (such as unprotected sex or needle sharing) for 28 days to prevent infection.

Antiretroviral Therapy (ART) for People with HIV: Effective ART reduces the viral load in an HIV-positive person to undetectable levels, making sexual transmission virtually impossible (known as undetectable = untransmittable, U=U).

Avoid Sharing Needles and Injection Equipment: Using sterile needles and syringes and participating in needle exchange programs reduce HIV transmission among people who inject drugs.

Voluntary Medical Male Circumcision: Circumcision reduces the risk of heterosexual men acquiring HIV by lowering the risk of viral entry through the foreskin.

Preventing Mother-to-Child Transmission: Testing pregnant women for HIV and providing ART during pregnancy, labor, and breastfeeding, along with safe delivery practices and infant prophylaxis, dramatically reduce perinatal HIV transmission.

Safe Sexual Practices: Choosing sexual activities with low or no risk of HIV transmission, such as oral sex (with caution), and avoiding contact with blood or genital sores help reduce risk.

Use of Lubricants: Water-based lubricants reduce condom breakage and mucosal tears during sex, lowering transmission risk.

Regular Testing and Early Diagnosis: Knowing one’s HIV status and that of partners enables timely treatment and prevention measures.

In summary, HIV prevention is multifaceted, combining barrier methods like condoms, biomedical interventions such as PrEP and ART, harm reduction for drug users, and strategies to prevent mother-to-child transmission. These approaches, when used consistently and correctly, effectively reduce new HIV infections.

StatPearls article on HIV and AIDS provides comprehensive clinical guidance on HIV pathophysiology, diagnosis, and management, emphasizing the importance of local clinical guidelines and baseline laboratory evaluations including CD4 count and viral load testing.

World Health Organization (WHO) Consolidated Guidelines on HIV Prevention, Testing, Treatment, Service Delivery and Monitoring offer updated global recommendations on HIV care continuum, including prevention strategies like PrEP and ART regimens for treatment.

Cleveland Clinic and Mayo Clinic resources detail HIV causes, transmission routes, diagnostic testing methods (antigen/antibody tests, nucleic acid tests), and treatment options including antiretroviral therapy (ART).

The International Antiviral Society-USA Panel provides recent recommendations on HIV treatment initiation, preferred ART regimens, and prevention strategies such as PrEP and PEP, highlighting rapid treatment initiation for better outcomes.

Medscape’s overview of HIV infection and AIDS discusses ART benefits, timing of therapy initiation, and monitoring protocols to achieve viral suppression and improve survival.

WHO African regional HIV guidelines further support prevention, diagnosis, and treatment approaches tailored to resource-limited settings.

**Malaria—Plasmodium Spp transmitted by Anopheles Mosquitoes**

**DESCRIPTION**

Malaria is a life-threatening infectious disease caused by single-celled parasites of the genus Plasmodium. The disease is transmitted to humans primarily through the bites of infected female Anopheles mosquitoes. Malaria is prevalent in tropical and subtropical regions, especially in sub-Saharan Africa, where the majority of global cases and deaths occur.

Causes and Transmission

Malaria is caused by a protozoan parasite from the genus Plasmodium.

Malaria is caused by five main species of Plasmodium parasites: P. falciparum, P. vivax, P. malariae, P. ovale, and P. knowlesi.

P. falciparum is the most deadly and common in Africa, while P. vivax is more prevalent outside sub-Saharan Africa

The disease spreads when an infected Anopheles mosquito bites a person, injecting the parasite into the bloodstream.                                                             The parasites first travel to the liver, mature, and then infect red blood cells, leading to cycles of illness[5](https://www.healthline.com/health/malaria)[6](https://en.wikipedia.org/wiki/Malaria)[8](https://www.medparkhospital.com/en-US/disease-and-treatment/malaria).

In rare cases, malaria can also be transmitted through blood transfusions or contaminated needles.

**SYMPTOMS**

Symptoms typically begin 10–15 days after infection and can range from mild to severe. Common symptoms include:

High fever and chills

Headache

Muscle aches and fatigue

Nausea, vomiting, and diarrhea

Severe malaria can cause:

Confusion or seizures

Difficulty breathing

Kidney failure

Coma

Anemia and jaundice (yellowing of the skin)

Enlargement of the spleen

Children under 5, pregnant women, travelers, and people with weakened immune systems are at higher risk for severe diseases like malaria.

 Diagnosis of Malaria

Malaria diagnosis involves identifying the Plasmodium parasite or its genetic material in a person's blood. Accurate diagnosis is essential for effective treatment and prevention of complications.

Types of Malaria Diagnosis

1. Clinical Diagnosis (Preliminary & Not Definitive)

Based on signs and symptoms such as:

High fever (often periodic)

Chills and sweating

Headache, nausea, vomiting

Muscle pain

Fatigue

Limitations: Symptoms are non-specific and similar to those of other illnesses like flu, typhoid, or dengue. Laboratory confirmation is needed.

2. Microscopic Diagnosis (Gold Standard)

Blood Smear Examination

A drop of blood is placed on a slide, stained (usually with Giemsa stain), and examined under a microscope.

 Two Types of Smears:

| Smear Type | Purpose |
| --- | --- |
| Thick smear | Detects presence of parasites (more sensitive) |
| Thin smear | Identifies the specific Plasmodium species |

Advantages:

High sensitivity and specificity

Can determine parasite count and species

 Limitations:

Requires trained personnel

Time-consuming

May miss low-level infections if not carefully done

3. Rapid Diagnostic Tests (RDTs)

Detect specific antigens (proteins) produced by malaria parasites in a finger-prick blood sample.

Provide results in 15–20 minutes.

Advantages:

Quick and easy to use

Useful in remote or resource-limited settings

 Limitations:

Less sensitive than microscopy, especially with low parasite levels

Some RDTs only detect P. falciparum

False positives may occur after treatment due to lingering antigens

4. Molecular Tests (PCR – Polymerase Chain Reaction)

Detect parasite DNA/RNA with very high sensitivity and specificity.

Advantages:

Can detect very low levels of parasites

Identifies all Plasmodium species accurately

 Limitations:

Expensive

Requires sophisticated lab facilities

Not used routinely in endemic areas

5. Serological Tests

Detect antibodies against Plasmodium.

Used mainly in research or epidemiological studies, not for acute diagnosis.

Additional Tests (After Diagnosis)

To assess severity or complications:

Complete blood count (CBC) – may show anemia or low platelets

Liver and kidney function tests

Blood glucose – to monitor for hypoglycemia, especially in P. falciparum infection

Summary Table

| Test Type | Purpose | Usefulness |
| --- | --- | --- |
| Clinical Diagnosis | Symptom-based | Not reliable alone |
| Microscopy | Parasite detection and identification | Most accurate in skilled hands |
| Rapid Diagnostic Test | Antigen detection | Good for quick diagnosis |
| PCR | DNA-based detection | Research and confirmatory |
| Serology | Antibody detection | Historical exposure (not current) |

**Treatment of Malaria**

The treatment of malaria depends on:

The Plasmodium species causing the infection

The severity of the disease (uncomplicated or severe)

The patient’s age, weight, and pregnancy status

Drug resistance patterns in the region (especially for Plasmodium falciparum)

 Uncomplicated Malaria Treatment

 First-Line Treatment (WHO Recommended):

Artemisinin-based Combination Therapies (ACTs)

ACTs combine artemisinin (fast-acting) with another antimalarial (long-acting) to kill all parasite forms and reduce resistance risk.

| ACT Example | Components |
| --- | --- |
| Artemether + Lumefantrine (Coartem) | Fast and effective; widely used |
| Artesunate + Amodiaquine | Used in many African countries |
| Artesunate + Mefloquine | Common in Asia |
| Dihydroartemisinin + Piperaquine | Long half-life; used in parts of Asia & Africa |

Note:

Duration is typically 3 days

Must complete the full course even if symptoms improve

 For P. vivax and P. ovale

These species can lie dormant in the liver (hypnozoites) and cause relapses.

Primaquine (after ACT treatment)

Kills dormant liver stages (radical cure)

G6PD testing is required before use to avoid hemolytic anemia

2. Severe or Complicated Malaria Treatment

Severe malaria is a medical emergency—often caused by P. falciparum—and requires hospitalization.

First-line drug:

Intravenous Artesunate

Given every 12 hours for 24 hours, then daily

Once stable, switch to a full course of oral ACT

Alternative (if IV Artesunate is unavailable):

Quinine dihydrochloride IV infusion (with monitoring)

Followed by oral treatment with ACT when patient improves

Special Treatment Cases

 Pregnant Women:

1st trimester: Quinine + clindamycin

2nd/3rd trimester: ACTs are generally safe (e.g., artemether-lumefantrine)

Children & Infants:

ACTs are effective and safe (dose adjusted by weight)

Severe cases need IV artesunate

Drug Resistance & Challenges

Drug resistance, especially to chloroquine and sometimes to artemisinin, is a growing concern.

Regions with known resistance (e.g., Southeast Asia) may require different ACTs or second-line drugs.

Monitoring and surveillance of resistance are critical.

 Supportive Care for Severe Malaria

Fluids and electrolytes

Antipyretics (e.g., paracetamol for fever)

Blood transfusions (if severe anemia)

Treatment of complications like kidney failure or hypoglycemia

 Summary Table

| Condition | Recommended Treatment |
| --- | --- |
| Uncomplicated malaria | ACT (e.g., artemether-lumefantrine) |
| P. vivax/P. ovale | ACT + primaquine (for liver stages) |
| Severe malaria | IV artesunate, then ACT |
| Pregnancy (1st trimester) | Quinine + clindamycin |
| Pregnancy (2nd/3rd trimester) | ACT (e.g., artemether-lumefantrine) |
| G6PD-deficient patients | Avoid primaquine |

Prevention

Malaria can be prevented by avoiding mosquito bites and by taking medicines. Talk to a doctor about taking medicines such as chemoprophylaxis before travelling to areas where malaria is common.

Lower the risk of getting malaria by avoiding mosquito bites:

Use mosquito nets when sleeping in places where malaria is present.

Use mosquito repellents (containing DEET, IR3535 or Icaridin) after dusk.

Use coils and vaporizers.

Wear protective clothing.

Use window screens.

Vector control

Vector control is a vital component of malaria control and elimination strategies as it is highly effective in preventing infection and reducing disease transmission. The 2 core interventions are insecticide-treated nets (ITNs) and indoor residual spraying (IRS).

Progress in global malaria control is threatened by emerging resistance to insecticides among Anopheles mosquitoes. However, new generation nets, which provide better protection against malaria than pyrethroid-only nets, are becoming more widely available and represent an important tool in global efforts to combat malaria.

Anopheles stephensi presents an added challenge for malaria control in Africa. Originally native to parts of south Asia and the Arabian Peninsula, the invasive mosquito species has been expanding its range over the last decade, with detections reported to date in eight African countries. An. stephensi thrives in urban settings, endures high temperatures and is resistant to many of the insecticides used in public health.

Chemoprophylaxis

Travellers to malaria endemic areas should consult their doctor several weeks before departure. The medical professional will determine which chemoprophylaxis drugs are appropriate for the country of destination. In some cases, chemoprophylaxis drugs must be started 2–3 weeks before departure. All prophylactic drugs should be taken on schedule for the duration of the stay in the malaria risk area and should be continued for 4 weeks after the last possible exposure to infection since parasites may still emerge from the liver during this period.

Preventive chemotherapies

Preventive chemotherapy is the use of medicines, either alone or in combination, to prevent malaria infections and their consequences. It requires giving a full treatment course of an antimalarial medicine to vulnerable populations at designated time points during the period of greatest malarial risk, regardless of whether the recipients are infected with malaria.

Preventive chemotherapy includes perennial malaria chemoprevention (PMC), seasonal malaria chemoprevention (SMC), intermittent preventive treatment of malaria in pregnancy (IPTp) and school-aged children (IPTsc), post-discharge malaria chemoprevention (PDMC) and mass drug administration (MDA). These safe and cost-effective strategies are intended to complement ongoing malaria control activities, including vector control measures, prompt diagnosis of suspected malaria, and treatment of confirmed cases with antimalarial medicines.

Vaccine

Since October 2021, WHO has recommended broad use of the RTS,S/AS01 malaria vaccine among children living in regions with moderate to high P. falciparum malaria transmission. The vaccine has been shown to significantly reduce malaria, and deadly severe malaria, among young children. In October 2023, WHO recommended a second safe and effective malaria vaccine, R21/Matrix-M. Vaccines are now being rolled out in routine childhood immunization programmes across Africa.  Malaria vaccines in Africa are expected to save tens of thousands of young lives every year. The highest impact will be achieved, however, when the vaccines are introduced alongside a mix of other WHO-recommended malaria interventions such as bed nets and chemoprophylaxis.

Early diagnosis and treatment of malaria reduces disease, prevents deaths and contributes to reducing transmission. WHO recommends that all suspected cases of malaria be confirmed using parasite-based diagnostic testing (through either microscopy or a rapid diagnostic test).

Malaria is a serious infection and always requires treatment with medicine.

Multiple medicines are used to prevent and treat malaria. Doctors will choose one or more based on:

the type of malaria

whether a malaria parasite is resistant to a medicine

the weight or age of the person infected with malaria

whether the person is pregnant.

These are the most common medicines for malaria:

Artemisinin-based combination therapy medicines are the most effective treatment for P. falciparum malaria.

Chloroquine is recommended for treatment of infection with the P. vivax parasite only in places where it is still sensitive to this medicine.

Primaquine should be added to the main treatment to prevent relapses of infection with the P. vivax and P. ovale parasites.

Most medicines used are in pill form. Some people may need to go to a health centre or hospital for injectable medicines.

Antimalarial drug resistance

Subsequent to the emergence of partial artemisinin resistance in the Greater Mekong subregion, WHO is very concerned about confirmed partial artemisinin resistance in Eritrea, Rwanda, Uganda and the United Republic of Tanzania. Based on available evidence, such resistance is also suspected in Ethiopia, Namibia, Sudan and Zambia.

In 2022, WHO developed a strategy to curb antimalarial drug resistance in Africa. Regular monitoring of antimalarial drug efficacy is needed to inform treatment policies in malaria-endemic countries, and to ensure early detection of, and response to, drug resistance.

Global Impact

In 2023, there were an estimated 263 million malaria cases and 597,000 deaths globally, with 94% of cases and 95% of deaths occurring in the WHO African Region

Children under 5 years old account for about 76% of malaria deaths in this region

Summary Table

| Aspect | Details |
| --- | --- |
| Cause | Plasmodium parasites via Anopheles mosquito bites |
| Main Symptoms | Fever, chills, headache, muscle aches, fatigue, nausea |
| Severe Complications | Seizures, confusion, organ failure, coma, death |
| Diagnosis | Blood smear microscopy, rapid diagnostic tests |
| Treatment | Antimalarial drugs (ACTs, quinine, etc.) |
| Prevention | Mosquito nets, repellents, prophylactic drugs, vaccines |
| High-Risk Groups | Children under 5, pregnant women, travelers, immunocompromised |
| Global Burden | ~263 million cases, ~597,000 deaths (2023), mostly in Africa |

Malaria remains a major public health challenge but is preventable and curable with prompt and effective interventions.

Leishmaniasis spp., transmitted by sandflies

DESCRIPTION

Leishmaniasis is a parasitic disease caused by protozoa of the genus Leishmania. It is transmitted to humans through the bite of infected female phlebotomine sandflies.

There are three main forms:

Cutaneous leishmaniasis – causes skin sores or ulcers.

Mucocutaneous leishmaniasis – affects the mucous membranes of the nose, mouth, and throat.

Visceral leishmaniasis (kala-azar) – the most severe form, affecting internal organs such as the spleen, liver, and bone marrow, and can be fatal if untreated.

Leishmaniasis is found in parts of the tropics, subtropics, and southern Europe. Risk factors include poor housing conditions, malnutrition, and weakened immune systems.

Treatment depends on the type and severity of the disease and may involve antiparasitic drugs, such as amphotericin B or miltefosine. There is no effective vaccine for humans as of now.

Transmission and Risk Factors

Transmitted by the bite of infected sandflies (genus Phlebotomus in the Old World, Lutzomyia in the New World).

Risk factors include poverty, malnutrition, deforestation, urbanization, and a weak immune system.

The disease can also affect animals, especially dogs and rodents, which can serve as reservoirs.

Symptoms of Leishmaniasis

Leishmaniasis presents in several clinical forms, each with distinct symptoms. The main types are cutaneous, mucocutaneous (mucosal), and visceral leishmaniasis.

Cutaneous Leishmaniasis (CL)

Most common form.

Symptoms usually appear within weeks to months after a sand fly bite.

Characterized by one or more skin sores at the site of the bite. These sores:

Often start as papules (bumps) or nodules (lumps).

May enlarge and develop into ulcers with raised edges and a central crater, sometimes covered by a scab or crust.

Are usually painless, though secondary bacterial infection can cause pain and redness.

May heal on their own over months to a year, often leaving permanent scars.

Swollen lymph nodes near the sores can occur.

Mucocutaneous (Mucosal) Leishmaniasis (ML)

Typically develops as a complication of cutaneous leishmaniasis, often months to years after the initial skin lesion heals.

Affects mucous membranes, primarily in the nose, but also the mouth and throat.

Symptoms include:

Sores and tissue destruction in the nose, mouth, or throat.

Stuffy nose, nasal discharge, or nosebleeds.

Severe cases can lead to facial disfigurement if untreated.

Rare and mostly seen in certain regions, such as Central and South America

Diagnosis of Leishmaniasis

Leishmaniasis diagnosis relies on a combination of clinical evaluation and laboratory methods to detect the Leishmania parasite or its DNA in patient samples. The choice of diagnostic tests depends on the clinical form (cutaneous, mucosal, or visceral) and available resources.

Key Diagnostic Methods

Parasitological Detection

Microscopic examination: Direct visualization of Leishmania amastigotes in tissue samples (e.g., skin lesions for cutaneous leishmaniasis [CL], bone marrow or spleen aspirates for visceral leishmaniasis [VL]) using Giemsa-stained smears. Sensitivity varies by region: 50–70% for CL caused by Old World species (Africa, Asia, Europe) and 15–30% for New World species (Americas).

Bone marrow aspiration: Preferred over riskier splenic aspiration for VL due to lower hemorrhage risk, with sensitivity of 60–85%.

Culture: In vitro culturing of aspirates or biopsies to isolate parasites, though this is time-consuming and limited to specialized labs.

Molecular Testing

PCR: Detects Leishmania DNA with high sensitivity (>80% for CL when combined with microscopy) and enables species identification, critical for guiding treatment.

Serological Tests

Rapid diagnostic tests (RDTs): Used for VL to detect antibodies (e.g., rK39 antigen tests). Sensitivity drops in HIV-coinfected patients.

Aldehyde test: A historical method for VL detecting elevated serum globulins, though nonspecific.

Histopathology

Skin or mucosal biopsies for CL and mucocutaneous leishmaniasis (ML), identifying granulomatous inflammation and parasites.

Clinical and Epidemiological Considerations

VL diagnosis: Combines clinical signs (prolonged fever, weight loss, splenomegaly) with parasitological or serological confirmation

CL diagnosis: Relies on clinical presentation (ulcerative/nodular lesions) and parasitological confirmation; serology has limited utility.

Species identification: Critical due to varying treatment responses, achieved via PCR or isoenzyme analysis.

Challenges and Recommendations

Low sensitivity in CL: New World species often yield fewer parasites, necessitating combined methods (e.g., PCR with microscopy).

Invasive procedures: Splenic aspiration, while highly sensitive for VL, carries hemorrhage risks and is avoided in favor of bone marrow sampling.

Reference labs: CDC and specialized centers provide advanced testing (e.g., species-specific PCR) but require pre-approval for sample submission.

Special Cases

HIV coinfection: Atypical parasite localization (e.g., gastrointestinal tract) and reduced serological sensitivity necessitate molecular testing.

Post-kala-azar dermal leishmaniasis (PKDL): Diagnosed via skin biopsy or PCR, as macular lesions often lack detectable parasites.

Early diagnosis improves outcomes, particularly for VL, which is fatal if untreated. Access to molecular methods remains limited in endemic regions, underscoring the need for affordable, point-of-care tests.

**TREATMENT OF LEISHMANIASIS**

The treatment of leishmaniasis depends on the clinical form (cutaneous, mucocutaneous, or visceral), the infecting Leishmania species, geographic region, and host factors such as immune status.

Treatment of Visceral Leishmaniasis (VL)

First-line therapy:

Liposomal amphotericin B (L-AmB) is the preferred treatment for immunocompetent patients in North America and many endemic areas. The FDA-approved regimen is 3 mg/kg IV on days 1–5, 14, and 21 (total 21 mg/kg).

In South Asia, single or short-course L-AmB regimens (e.g., 10 mg/kg single dose or 3–5 mg/kg daily for 3–5 days) are effective.

Alternative therapies:

Miltefosine, an oral agent, is effective for VL caused by L. donovani in patients ≥12 years old and ≥30 kg, given at 2.5 mg/kg/day for 28 days. It is not recommended for L. infantum infections or in pregnancy.

Pentavalent antimonials (e.g., meglumine antimoniate or sodium stibogluconate) at 20 mg SbV/kg/day IV or IM for 28 days remain effective in regions with low antimony resistance (<10%), but are less favored in South Asia due to resistance.

Paromomycin IM is used in East Africa, often combined with antimonials.

HIV coinfection:

Liposomal amphotericin B is preferred, often at higher cumulative doses (20–60 mg/kg). Pentavalent antimonials are generally avoided due to toxicity and lower efficacy.

Miltefosine can be used adjunctively but with caution.

Supportive care: Hydration, nutritional support, and treatment of concurrent infections (e.g., malaria, tuberculosis) are essential.

Treatment of Cutaneous Leishmaniasis (CL)

Many CL lesions heal spontaneously within 3–6 months; treatment is indicated for persistent, disfiguring, ulcerative, or disseminated lesions.

Local therapy: Intralesional injections of pentavalent antimonials (1–2 ml infiltrated into lesion edges) every 3–7 days for 2–4 weeks.

Systemic therapy: For multiple or severe lesions, IM pentavalent antimonials (20 mg/kg daily for 10–20 days) or oral miltefosine for 28 days are effective options.

Antibiotics may be needed for secondary bacterial infections of ulcers.

Treatment of Mucocutaneous Leishmaniasis (ML)

Strong recommendation for systemic pentavalent antimonials, often combined with oral pentoxifylline to reduce inflammation.

Treatment duration is generally 28 days or longer depending on clinical response.

Additional Notes

Azole antifungals such as ketoconazole and fluconazole have limited and variable efficacy; ketoconazole has been removed from some treatment guidelines due to lack of benefit and toxicity concerns.

Treatment choice should consider Leishmania species, geographic region, patient immune status, and potential drug toxicities.

Close clinical monitoring is necessary to adjust therapy and manage adverse effects.

In summary, liposomal amphotericin B is the cornerstone for VL treatment, with miltefosine and pentavalent antimonials as alternatives depending on region and patient factors. CL and ML treatment relies on pentavalent antimonials and miltefosine, with local therapy for limited CL lesions.

Prevention of Leishmaniasis

Prevention of leishmaniasis primarily focuses on reducing exposure to the sand fly vector, as there are currently no vaccines or drugs available to prevent infection.

Key Prevention Strategies

Avoid Sand Fly Bites

Sand flies are much smaller than mosquitoes and can enter through smaller openings, so protecting living and sleeping areas is crucial.

Use well-screened or air-conditioned rooms.

Employ insecticide sprays indoors to kill sand flies.

Use insecticide-treated bed nets, especially when sleeping in unscreened areas. Untreated nets also provide some protection but are less effective.

Wear protective clothing outdoors-long-sleeved shirts, long pants, socks, and tuck shirts into pants to minimize exposed skin.

Apply EPA-registered insect repellents containing DEET on exposed skin and around clothing edges.

Environmental and Vector Control

Reduce sand fly populations by insecticide spraying of houses and surrounding areas.

Remove or modify sand fly breeding sites, such as cracks in walls, by plastering with mud or lime.

Use insecticide-treated nets and curtains to reduce indoor sand fly contact.

Vector control is essential to interrupt transmission and reduce disease burden.

Reservoir Control

In zoonotic leishmaniasis, controlling animal reservoirs (e.g., dogs, rodents) through measures such as culling or treatment can reduce transmission.

Surveillance and management of reservoir hosts are part of integrated control strategies.

Behavioral Measures

Avoid outdoor activities from dusk to dawn when sand flies are most active.

Sleep on higher floors if possible, as sand flies are weak fliers.

Use fans indoors to reduce sand fly activity.

Community and Socioeconomic Interventions

Improve housing conditions to reduce vector-human contact.

Conduct health education campaigns to raise awareness of leishmaniasis and preventive measures.

Strengthen surveillance systems to detect and respond to outbreaks promptly.

Summary

No vaccines or prophylactic drugs exist for leishmaniasis, so personal protection against sand fly bites and vector control remain the cornerstone of prevention. Combining insecticide-treated nets, protective clothing, indoor insecticide spraying, environmental management, and reservoir control offers the best approach to reduce transmission and disease incidence.

African Trypanosomiasis (sleeping sickness) - trypanosoma brucei, tsetse fly vector

DESCRIPTION

Trypanosomiasis, commonly known as African sleeping sickness, is a parasitic disease caused by Trypanosoma brucei species transmitted to humans by the bite of infected tsetse flies. There are two main forms: Trypanosoma brucei gambiense (chronic form, responsible for over 90% of cases in West and Central Africa) and Trypanosoma brucei rhodesiense (acute form, found in East and Southern Africa).

CAUSES AND TRANSMISSION

African trypanosomiasis (sleeping sickness) is caused by protozoan parasites of the Trypanosoma brucei species complex, primarily T. brucei gambiense (chronic form in West and Central Africa) and T. brucei rhodesiense (acute form in East and Southern Africa)

Causes

The disease is caused by Trypanosoma brucei subspecies, which are extracellular parasites that multiply in the blood, lymph, and central nervous system of humans.

Transmission

The primary mode of transmission is cyclical transmission via the bite of an infected tsetse fly (Glossina species), which serves as both vector and host for the parasite.

Tsetse flies acquire the infection by feeding on infected humans or animals. After a developmental cycle of about 15–21 days inside the fly’s midgut and salivary glands, the parasites become infective metacyclic trypomastigotes that are transmitted to a new host during the fly’s blood meal.

Transmission occurs early in the blood-feeding process when the fly injects saliva containing infective parasites into the skin.

The parasite modifies the tsetse fly’s salivary composition and feeding behavior, prolonging feeding time and increasing the likelihood of biting multiple hosts, which enhances transmission.

Mechanical transmission by other biting flies (e.g., tabanids, stomoxes) can occur for some animal trypanosomes like T. vivax but is not a significant route for human-infective species.

Other rare transmission routes include mother-to-child (vertical) transmission across the placenta.

In summary, African trypanosomiasis is caused by Trypanosoma brucei parasites transmitted mainly through the bite of infected tsetse flies, which become infective after a developmental cycle in the fly and transmit parasites during blood feeding. The parasite’s manipulation of fly feeding behavior increases transmission efficiency among mammalian hosts.

Diagnosis of Trypanosomiasis

Diagnosis of African trypanosomiasis (sleeping sickness) is primarily confirmed by detecting the parasite Trypanosoma brucei in body fluids through microscopy.

Diagnostic Methods

Microscopic identification of trypanosomes is the definitive diagnostic method. Samples examined include:

Blood smears (thin or thick) or buffy coat preparations, especially for T. b. rhodesiense, which typically has higher parasitemia.

Lymph node aspirates, particularly for T. b. gambiense, where parasites may be found in enlarged cervical lymph nodes.

Chancre fluid obtained from the bite site in T. b. rhodesiense infections.

Bone marrow aspirates can be used but are less common.

Cerebrospinal fluid (CSF) examination is essential for staging the disease and detecting CNS involvement in the late (second) stage.

Wet preparations of fresh samples are examined for motile trypanosomes, as motility aids detection, especially in blood and lymph node aspirates. Smears are also stained with Giemsa or Field stain for parasite visualization.

Concentration techniques improve sensitivity due to often low parasite levels, especially in T. b. gambiense infections. These include:

Centrifugation followed by buffy coat examination.

Mini anion-exchange centrifugation technique (mAECT).

Quantitative buffy coat (QBC) technique.

Microhematocrit centrifugation.

Serological tests such as the Card Agglutination Test for Trypanosomiasis (CATT) are used mainly for screening in endemic areas for T. b. gambiense. CATT has high sensitivity (~91%) but limited specificity and cannot confirm diagnosis alone. It is not used for T. b. rhodesiense diagnosis.

Lumbar puncture and CSF analysis are mandatory for staging:

Presence of trypanosomes or >5 white blood cells/μL in CSF indicates second-stage (neurological) disease.

Elevated CSF protein and nonspecific IgM may also support CNS involvement.

Other laboratory findings may include anemia, monocytosis, and elevated serum polyclonal IgM, but these are nonspecific.

Summary

Diagnosis relies on direct microscopic detection of trypanosomes in blood, lymph node aspirates, chancre fluid, or CSF, supported by concentration methods to increase sensitivity. Serological screening (CATT) is useful for T. b. gambiense in mass screening but not definitive. CSF examination is critical for staging disease and guiding treatment decisions

Treatment of human African trypanosomiasis (HAT)

Treatment of human African trypanosomiasis (HAT) varies by causative species (T. b. gambiense or T. b. rhodesiense), disease stage (first/hemolymphatic vs. second/neurological), patient age, and weight.

First Stage (Hemolymphatic Stage) Treatment

For T. b. gambiense:

Fexinidazole, an oral antitrypanosomal drug, is now the first-line treatment for both first and second stages in patients ≥6 years old and weighing ≥20 kg. It is taken once daily for 10 days under medical supervision with food to ensure efficacy.

Pentamidine (intramuscular or intravenous) remains an alternative for patients who cannot take fexinidazole.

For T. b. rhodesiense:

Fexinidazole is also recommended as first-line treatment for patients ≥6 years old and ≥20 kg.

For children <6 years or weighing <20 kg, suramin is preferred if CSF shows ≤5 white blood cells/μL and no trypanosomes.

Pentamidine may be used as interim therapy while awaiting definitive treatment.

Second Stage (CNS Involvement) Treatment

For T. b. gambiense:

Fexinidazole is recommended for nonsevere second-stage disease.

NECT (Nifurtimox-Eflornithine Combination Therapy) is the standard treatment for more severe cases: nifurtimox orally (15 mg/kg/day in three doses for 10 days) combined with eflornithine IV (400 mg/kg/day in two infusions for 7 days).

Eflornithine monotherapy can be used if NECT is unavailable.

Melarsoprol is reserved for relapses or when other treatments are contraindicated due to its high toxicity.

For T. b. rhodesiense:

Fexinidazole is first-line for patients ≥6 years and ≥20 kg with CNS involvement.

Melarsoprol remains the treatment of choice for children <6 years or <20 kg, or severe CNS disease. It is given intravenously at 2.2 mg/kg daily for 10 days but carries significant risk of severe adverse effects including encephalopathy.

Prednisolone is often co-administered to reduce inflammatory complications during melarsoprol treatment.

Additional Considerations

Treatment must be administered under close medical supervision, often requiring hospitalization for intravenous therapies.

Monitoring for adverse effects and follow-up lumbar punctures for treatment response and relapse detection are essential.

WHO provides free access to NECT and fexinidazole in endemic countries, improving treatment accessibility and reducing reliance on toxic drugs like melarsoprol.

Fexinidazole’s oral administration reduces the need for lumbar puncture in eligible patients and improves treatment feasibility in resource-limited settings.

Summary Table

| Disease Stage | Species | First-line Treatment | Notes |
| --- | --- | --- | --- |
| First stage | T. b. gambiense | Fexinidazole (oral) or Pentamidine | Fexinidazole preferred ≥6 years, ≥20 kg |
| First stage | T. b. rhodesiense | Fexinidazole (oral) or Suramin (children <6 yrs) | Suramin requires test dose; pentamidine interim |
| Second stage (nonsevere) | T. b. gambiense | Fexinidazole (oral) | Suitable for nonsevere CNS disease |
| Second stage (severe) | T. b. gambiense | NECT or Eflornithine monotherapy | Hospitalization required |
| Second stage | T. b. rhodesiense | Fexinidazole (≥6 yrs, ≥20 kg) or Melarsoprol (children <6 yrs or severe) | Melarsoprol highly toxic; prednisolone adjunct |

These updated WHO guidelines emphasize fexinidazole as a revolutionary oral treatment simplifying management of both gambiense and rhodesiense HAT, reducing the need for toxic drugs and invasive procedures

Prevention of Human African Trypanosomiasis (HAT)

Prevention of human African trypanosomiasis (sleeping sickness) centers on avoiding bites from the tsetse fly, the disease’s sole vector, as there are no vaccines or prophylactic drugs available.

Key Prevention Measures

Avoid contact with tsetse flies:

Stay away from tsetse-infested areas, especially dense vegetation and bushes where flies rest.

Avoid traveling to endemic regions when possible.

Protective clothing:

Wear medium-weight, neutral-colored clothing (avoid bright or dark colors that attract tsetse flies).

Use long-sleeved shirts and long pants to reduce exposed skin, as tsetse flies can bite through thin fabrics.

Insect repellents and treated clothing:

Use insect repellents, although their effectiveness against tsetse flies is limited.

Permethrin-treated clothing may help prevent other insect bites but is less effective against tsetse flies.

Environmental and behavioral strategies:

Inspect vehicles for tsetse flies before entering, as they are attracted to moving vehicles.

Avoid bushes and shaded areas during peak tsetse activity times.

Use insecticide spraying and traps to reduce local tsetse populations in endemic areas.

Community and vector control:

Early diagnosis and treatment of cases reduce human reservoirs and transmission.

Vector control programs, including insecticide-treated targets and aerial spraying, help suppress tsetse populations.

Surveillance systems identify high-risk areas and guide interventions.

Avoidance of high-risk activities:

Limit outdoor activities during dawn and dusk when tsetse flies are most active.

Sleep in screened or air-conditioned rooms when in endemic areas.

Additional Notes

Pentamidine was once used for prophylaxis against T. b. gambiense but is no longer recommended due to serious side effects.

Ongoing efforts by WHO and partners focus on integrated control combining vector management, case detection, and treatment access.

Socioeconomic improvements, education, and housing enhancements also contribute to reducing disease risk.

In summary, preventing African trypanosomiasis relies on minimizing exposure to tsetse flies through protective clothing, behavioral modifications, vector control, and prompt treatment of infected individuals to interrupt transmission

World Health Organization (WHO) Guidelines for the Treatment of Human African Trypanosomiasis, 2024  
Provides updated evidence-based recommendations on therapeutic choices including the use of fexinidazole and NECT.  
WHO Guidelines 2024

Centers for Disease Control and Prevention (CDC) Clinical Guidance for Human African Trypanosomiasis  
Details diagnostic approaches, staging, and treatment protocols with practical clinical guidance.  
CDC Clinical Guidance 2024

BMJ Best Practice: African Trypanosomiasis  
Summary of clinical features, diagnosis, and treatment including the importance of microscopy and staging.  
BMJ Best Practice 2024

MSD Manual Professional Version: African Trypanosomiasis  
Covers clinical presentation, diagnostic methods including concentration techniques, and prevention strategies.  
MSD Manual 2022

WHO Report on Control of Human African Trypanosomiasis, 2004  
Discusses control strategies including active case detection, treatment, and vector control efforts.  
WHO Control Report 2004

World Health Organization (WHO) Guidelines for Malaria, 30 November 2024  
The most recent comprehensive guidelines covering prevention, diagnosis, and treatment of malaria, including updated recommendations on artemisinin-based combination therapies (ACTs), primaquine use, and malaria vaccines.  
Available at WHO IRIS: [WHO Malaria Guidelines 2024]

WHO Global Malaria Programme – New and Updated Malaria Guidance  
Provides ongoing updates and consolidated recommendations on malaria control and treatment strategies.  
WHO website: [Global Malaria Programme Guidance]

WHO Malaria Guidelines: Treatment of Malaria (2023 update)  
Detailed treatment protocols for uncomplicated and severe malaria, including special populations such as pregnant women and HIV co-infected patients.  
WHO Publication: [Treatment of Malaria PDF]

PAHO – WHO Guidelines for the Treatment of Malaria  
Regional adaptation of WHO malaria treatment guidelines with practical clinical recommendations.  
PAHO Document: [PAHO Malaria Treatment Guidelines]

Medscape Malaria Guidelines (2024)  
Clinical overview and treatment guidelines for malaria, updated with recent evidence.  
Medscape Reference: [Malaria Guidelines]

Leishmaniasis References

While the search results do not directly provide recent leishmaniasis guidelines, the following sources are authoritative for diagnosis, treatment, and prevention of leishmaniasis:

World Health Organization (WHO) Leishmaniasis Fact Sheets and Guidelines  
WHO provides detailed information on leishmaniasis epidemiology, diagnosis, treatment options, and control measures.  
WHO Leishmaniasis Portal: WHO Leishmaniasis Information

Centers for Disease Control and Prevention (CDC) Leishmaniasis Resources  
Comprehensive clinical guidance on diagnosis, treatment, and prevention of leishmaniasis.  
CDC Leishmaniasis: CDC Leishmaniasis

WHO Technical Report Series on Leishmaniasis Control  
Provides in-depth guidance on integrated control strategies including vector management and reservoir control.

Here are authoritative references for Malaria, Trypanosomiasis (Chagas disease), and HIV:

**Malaria**

* While the search results did not provide direct references for malaria, standard authoritative sources include:
  + World Health Organization (WHO) Malaria fact sheets and reports
  + CDC Malaria website
  + Peer-reviewed articles in journals such as *The Lancet Infectious Diseases* and *Clinical Microbiology Reviews*

**Trypanosomiasis (Chagas Disease caused by *Trypanosoma cruzi*)**

* Medscape overview of Chagas disease, including epidemiology, transmission, and clinical features
* ScienceDirect articles on Chagas disease, including treatment, congenital transmission, and global control strategies
* NCBI StatPearls chapter on Chagas disease
* BMJ Best Practice references on Chagas disease clinical management
* WHO fact sheet on Chagas disease detailing transmission, prevention, and treatment
* CDC DPDx page on American Trypanosomiasis with diagnostic and epidemiologic information
* Wikipedia summary on Chagas disease with research updates on diagnostics and treatments
* ScienceDirect book chapter on American Trypanosomiasis (Chagas disease)

**HIV**

* No specific search results were provided for HIV references here. Authoritative references include:
  + WHO HIV/AIDS fact sheets and guidelines
  + CDC HIV/AIDS website
  + UNAIDS global reports
  + Peer-reviewed journals such as *The Lancet HIV* and *AIDS*

**REFERENCES**

1. <https://emedicine.medscape.com/article/214581-overview>
2. <https://www.sciencedirect.com/science/article/abs/pii/S0140673623017877>
3. <https://www.ncbi.nlm.nih.gov/books/NBK459272/>
4. <https://bestpractice.bmj.com/topics/en-gb/1160/references>
5. <https://www.who.int/news-room/fact-sheets/detail/chagas-disease-(american-trypanosomiasis)>
6. <https://www.cdc.gov/dpdx/trypanosomiasisamerican/index.html>
7. <https://en.wikipedia.org/wiki/Chagas_disease>
8. <https://www.sciencedirect.com/book/9780128010297/american-trypanosomiasis-chagas-disease>

**BACTERIAL AND VIRAL DISEASES**

**Yaws-Definition**

Yaws is a chronic, contagious, non-venereal infectious disease caused by the bacterium *Treponema pallidum* subspecies *pertenue*. It primarily affects the skin, bones, and cartilage, leading to disfiguring and debilitating lesions mostly in children living in warm, humid tropical regions

**Causes and Transmission**

* **Cause**: Infection with *Treponema pallidum pertenue*, a spirochete bacterium closely related to the syphilis-causing organism.
* **Transmission**: Direct skin-to-skin contact with fluid from active lesions, usually in poor, rural communities with low hygiene standards. The bacterium enters through minor skin abrasions.
* **Epidemiology**: Over 80,000 cases reported annually, mostly in children under 15 years (75–80%), with peak incidence at ages 6–10. Endemic in parts of Africa, Asia, Latin America, and Pacific islands. Ghana, Papua New Guinea, and Solomon Islands report >10,000 cases/year.

**Signs and Symptoms**

* **Primary stage**: Painless papillomas or “Mother Yaw” lesions, 2–5 cm, often on lower limbs. Lesions are highly contagious and may ulcerate.
* **Secondary stage**: Disseminated skin lesions (papillomatous, ulcerative), bone involvement causing pain, swelling, and deformities (osteoperiostitis, dactylitis).
* **Tertiary stage (rare)**: Disfiguring gummas, nasal cartilage destruction, bone deformities, and palmar/plantar hyperkeratosis.

## Diagnosis Methods

* **Clinical diagnosis** based on characteristic lesions in endemic areas.
* **Darkfield microscopy** of lesion exudate to visualize spirochetes (gold standard).
* **Serologic tests** (RPR, VDRL) are used but not definitive due to cross-reactivity with syphilis[1](https://www.who.int/news-room/fact-sheets/detail/yaws)[2](https://rarediseases.org/rare-diseases/yaws/).
* **Imaging** (X-rays) may show bone changes in secondary stage.

## Treatment Options

* **Preferred**: Single oral dose of azithromycin 30 mg/kg (max 2 g).
* **Alternative**: Single intramuscular benzathine penicillin (0.6 million units for children under 10, 1.2 million units for older patients).
* **Follow-up**: Reexamination 4 weeks post-treatment; >95% cure rate.
* **Resistance testing** recommended if treatment failure suspected.

## Prevention

* No vaccine available.
* Hygiene improvement and health education to reduce transmission.
* Mass drug administration (MDA) campaigns targeting entire endemic communities (Total Community Treatment - TCT).
* Treatment of close contacts to prevent spread.

**Prognosis**

* Excellent with timely antibiotic treatment; most patients fully recover without sequelae.
* Untreated cases can progress to disabling tertiary lesions causing permanent disfigurement.

**Possible Complications**

* Chronic disfigurement: nasal destruction, bone deformities, joint damage.
* Secondary bacterial infections of skin lesions.
* Disability due to bone and joint involvement.

**When to See a Doctor / Red Flags**

* Persistent painless skin lesions or ulcers, especially in children from endemic regions.
* Bone pain, swelling, or deformity.
* Non-healing or worsening lesions despite treatment.
* Systemic symptoms such as fever or lymphadenopathy.

**Differential Diagnosis**

* Syphilis (venereal treponematosis).
* Other tropical skin infections: leprosy, cutaneous leishmaniasis, fungal infections.
* Non-infectious dermatologic conditions causing ulcers or papillomas.

**Recent Guidelines or Updates**

* WHO recommends azithromycin as first-line treatment and supports MDA for eradication.
* WHO’s Morges Strategy involves TCT followed by targeted treatment and surveillance aiming for eradication by 2020 (ongoing efforts).
* Azithromycin included in WHO Essential Medicines List for yaws treatment.

**Epidemiology and Statistics**

* Historically endemic in >90 countries; now limited to pockets in ~14 countries with active surveillance.
* Estimated 80,000+ cases annually worldwide, predominantly children under 15 years.
* Successful eradication campaigns in India and Ecuador have interrupted transmission.

**References**

[1](https://www.who.int/news-room/fact-sheets/detail/yaws) World Health Organization. Yaws Fact Sheet. 2023.  
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[3](https://my.clevelandclinic.org/health/diseases/25011-yaws) Cleveland Clinic. Yaws: Causes, Symptoms, Diagnosis & Treatment.  
[4](https://wwwnc.cdc.gov/eid/article/17/6/10-1575_article) CDC. Outcome Predictors in Treatment of Yaws. 2011.  
[5](https://iris.who.int/bitstream/handle/10665/259902/9789241512695-eng.pdf) WHO. Eradication of Yaws. 2016.  
[6](https://www.sciencedirect.com/science/article/abs/pii/S0140673612621308) ScienceDirect. Yaws overview.  
[7](https://dermnetnz.org/topics/yaws) DermNet NZ. Yaws.

**Melioidosis (Whitmore Disease)**

**Definition**

Melioidosis is an infectious disease caused by the gram-negative bacterium *Burkholderia pseudomallei*. It can cause a wide spectrum of illness ranging from localized skin infections to severe pneumonia, septicemia, and multi-organ abscesses. Known as the “great mimicker,” it often resembles tuberculosis or other bacterial infections.

**Causes and Transmission**

* **Cause**: Infection with *Burkholderia pseudomallei*, a soil- and water-dwelling bacterium.
* **Transmission**:
  + Entry via skin abrasions, wounds, inhalation of contaminated dust or water droplets, or ingestion of contaminated water or food.
  + Person-to-person transmission is extremely rare.
* **Epidemiology**:
  + Endemic in tropical regions, especially Southeast Asia (northeast Thailand) and northern Australia.
  + Also reported in South Asia, China, Middle East, Africa, and recently detected in the Gulf Coast of Mississippi, USA.
  + High-risk groups include diabetics, immunocompromised, farmers, and those with chronic diseases.

**Signs and Symptoms**

* **Incubation**: Average 9 days (range 1–21 days).
* **Acute melioidosis**: Fever, chills, cough, chest pain, pneumonia, abscesses in liver, spleen, prostate, or skin, septic shock.
* **Chronic melioidosis**: Symptoms lasting >2 months, mimicking tuberculosis with weight loss, productive cough, lymphadenopathy, and abscesses.
* **Latent infection**: Possible dormancy for years with reactivation risk, especially in immunocompromised individuals.
* **Severe manifestations**: Encephalomyelitis, septic arthritis, osteomyelitis, prostatic abscesses, parotid abscesses in children, and multiorgan failure.

**Diagnosis Methods**

* **Microbiological culture**: Isolation of *B. pseudomallei* from blood, sputum, pus, urine, or other fluids is definitive.
* **Microscopy**: Gram-negative bacilli with bipolar staining (“safety pin” appearance).
* **Imaging**: Chest X-ray or CT scans to detect pneumonia or abscesses.
* **Molecular tests**: PCR assays in specialized labs.
* **Serology**: Limited utility due to endemic exposure and cross-reactivity.

**Treatment Options**

* **Intensive phase**: Intravenous ceftazidime (2 g every 6–8 hours) or meropenem/imipenem for 10–14 days or longer depending on severity.
* **Eradication phase**: Oral trimethoprim-sulfamethoxazole (TMP-SMX) for 3–6 months to prevent relapse.
* **Alternatives**: Amoxicillin-clavulanic acid for those intolerant to TMP-SMX.
* **Supportive care**: Surgical drainage of abscesses if needed.
* Mortality ranges from 10% in developed healthcare settings to up to 40% in resource-limited areas.

**Prevention**

* Avoid contact with contaminated soil and water, especially with open wounds.
* Protective clothing and footwear for at-risk occupations.
* No vaccine currently available.
* Early diagnosis and treatment critical to reduce morbidity and mortality.

**Prognosis**

* Variable; good with early diagnosis and appropriate therapy.
* High mortality without treatment or in severe cases.
* Risk of relapse if eradication phase not completed.

**Possible Complications**

* Septic shock and multiorgan failure.
* Lung abscesses, cavitary lesions.
* Disseminated abscesses in liver, spleen, prostate, kidneys.
* Osteomyelitis, septic arthritis.
* Neurological involvement (encephalomyelitis).

**When to See a Doctor / Red Flags**

* Persistent fever with respiratory symptoms after travel or residence in endemic areas.
* Painful skin lesions or abscesses.
* Signs of systemic infection: confusion, hypotension, severe abdominal pain.
* Recurrence of symptoms after initial treatment.

**Differential Diagnosis**

* Tuberculosis (similar chronic pulmonary and systemic symptoms).
* Other bacterial pneumonias and abscess-forming infections.
* Fungal infections (histoplasmosis).
* Other causes of septicemia and soft tissue infections.

**Recent Guidelines or Updates**

* CDC Yellow Book 2024 recommends prolonged antimicrobial therapy with initial IV ceftazidime or carbapenems followed by oral TMP-SMX.
* WHO and tropical medicine guidelines emphasize early diagnosis and prolonged treatment to prevent relapse.
* Recognition of melioidosis as an emerging infectious disease in new geographic areas due to climate change and travel.

**Epidemiology and Statistics**

* Estimated 165,000 cases globally per year with approximately 89,000 deaths.
* Diabetes mellitus is a major risk factor, present in up to 50% of cases.
* Increasing recognition in non-endemic countries due to travel and environmental changes.

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**Relapsing Fever**

**Definition**

Relapsing fever is an acute infectious disease characterized by recurring episodes of fever, caused by infection with *Borrelia* species spirochetes.

**Causes and Transmission**

* **Cause**: Infection with *Borrelia* spp., primarily *Borrelia recurrentis* (louse-borne) and various *Borrelia* species transmitted by soft ticks (tick-borne).
* **Transmission**:
  + Louse-borne relapsing fever (epidemic) transmitted by the human body louse (*Pediculus humanus corporis*).
  + Tick-borne relapsing fever transmitted by Ornithodoros soft ticks, often in rodent habitats.
* **Epidemiology**:
  + Louse-borne form occurs in crowded, poor hygiene conditions, mainly in East Africa.
  + Tick-borne form occurs in rural areas of Africa, North America, and parts of Asia.

**Signs and Symptoms**

* Sudden onset of high fever, chills, headache, muscle and joint pain, nausea, vomiting.
* Fever lasts about 3–7 days, followed by afebrile period, then relapse of fever (hence "relapsing").
* Other symptoms: rash, hepatosplenomegaly, jaundice, neurological symptoms in severe cases.
* Jarisch-Herxheimer reaction may occur after antibiotic treatment.

**Diagnosis Methods**

* Blood smear microscopy during febrile episodes to visualize spirochetes.
* PCR and serologic tests available in specialized labs.
* Clinical diagnosis supported by epidemiological context.

**Treatment Options**

* Antibiotics: doxycycline or erythromycin are first-line treatments.
* For pregnant women and children, erythromycin preferred.
* Supportive care for symptoms and Jarisch-Herxheimer reaction management.

**Prevention**

* Improve hygiene and living conditions to control lice.
* Avoid exposure to tick habitats; use insect repellents and protective clothing.
* Control of rodent populations in endemic areas.

**Prognosis**

* Generally good with prompt antibiotic treatment.
* Mortality higher in untreated louse-borne relapsing fever (up to 40%).
* Jarisch-Herxheimer reaction can be severe but manageable.

**Possible Complications**

* Severe anemia, neurological complications, myocarditis, hepatitis.
* Death from complications in untreated cases.

**When to See a Doctor / Red Flags**

* Repeated episodes of fever with chills and headache.
* Severe weakness, confusion, or jaundice.
* Symptoms following exposure to lice or tick-infested areas.

**Differential Diagnosis**

* Malaria, typhoid fever, leptospirosis, dengue fever, and other febrile illnesses.

**Recent Guidelines or Updates**

* WHO and CDC recommend doxycycline as first-line therapy; emphasize control of lice and ticks.
* Increased surveillance in endemic regions.

**Epidemiology and Statistics**

* Louse-borne relapsing fever causes hundreds of thousands of cases during epidemics in Africa.
* Tick-borne relapsing fever is endemic in parts of the western US, Africa, and Asia.

**References**

* CDC. Relapsing Fever. 2024.
* WHO. Louse-borne relapsing fever factsheet. 2023.
* Mayo Clinic. Relapsing Fever Overview. 2024.
* CDC DPDx. Relapsing Fever.

**Trachoma**

**Definition**

Trachoma is a chronic infectious eye disease caused by *Chlamydia trachomatis* leading to conjunctival inflammation, scarring, and potentially blindness.

**Causes and Transmission**

* **Cause**: *Chlamydia trachomatis* serovars A, B, Ba, and C.
* **Transmission**: Direct contact with eye and nose secretions, fomites (towels), and eye-seeking flies (*Musca sorbens*).
* **Epidemiology**:
  + Endemic in poor, rural areas of Africa, Asia, Middle East, and parts of Latin America.
  + Leading infectious cause of blindness worldwide.

**Signs and Symptoms**

* Early: follicular conjunctivitis, eye irritation, discharge.
* Recurrent infections cause conjunctival scarring.
* Advanced: trichiasis (inward turning eyelashes), corneal opacity, and blindness.

**Diagnosis Methods**

* Clinical examination with magnification to detect follicles and scarring.
* Laboratory tests: conjunctival swabs for PCR or direct immunofluorescence.
* WHO simplified grading system used for field diagnosis.

**Treatment Options**

* Antibiotics: single-dose oral azithromycin or topical tetracycline ointment.
* Surgery for trichiasis to prevent corneal damage.
* Repeated treatment in endemic communities.

**Prevention**

* WHO SAFE strategy: Surgery, Antibiotics, Facial cleanliness, and Environmental improvement.
* Improved sanitation and access to clean water.
* Health education to reduce transmission.

**Prognosis**

* Good with early treatment and surgery.
* Untreated cases progress to blindness.

**Possible Complications**

* Corneal scarring and blindness.
* Secondary bacterial infections.

**When to See a Doctor / Red Flags**

* Persistent eye redness, discharge, or pain.
* Inward turning eyelashes or vision changes.

**Differential Diagnosis**

* Other causes of conjunctivitis and corneal scarring (viral, bacterial, allergic).

**Recent Guidelines or Updates**

* WHO recommends mass antibiotic treatment in endemic areas combined with hygiene and environmental interventions.
* Azithromycin is preferred due to ease of administration.

**Epidemiology and Statistics**

* Approximately 1.9 million people visually impaired or blind due to trachoma globally.
* Over 80 million people live in endemic areas requiring intervention.
* Most cases in sub-Saharan Africa.

**References**

* WHO. Trachoma Fact Sheet. 2023.
* CDC. Trachoma. 2024.
* Mayo Clinic. Trachoma Overview. 2023.
* The Carter Center. Trachoma Program.
* WHO Alliance for the Global Elimination of Trachoma by 2030 (GET2020).

**Leishmaniasis**

**Definition**

Leishmaniasis is a parasitic disease caused by protozoa of the genus *Leishmania*, transmitted by the bite of infected female phlebotomine sandflies. It manifests mainly in three clinical forms: cutaneous, mucocutaneous, and visceral leishmaniasis.

**Causes and Transmission**

* **Cause**: Infection with various *Leishmania* species (e.g., *L. donovani*, *L. infantum*, *L. major*, *L. braziliensis*).
* **Transmission**: Bite of infected female sandflies.
* **Epidemiology**:
  + Endemic in tropical and subtropical regions including parts of Asia, Africa, the Americas, and the Mediterranean.
  + Approximately 700,000 to 1 million new cases annually worldwide.
  + Visceral leishmaniasis (kala-azar) is fatal if untreated.

**Signs and Symptoms**

* **Cutaneous leishmaniasis**: Skin ulcers at the bite site, painless, with raised edges; may heal spontaneously but leave scars.
* **Mucocutaneous leishmaniasis**: Destructive lesions of mucous membranes of nose, mouth, and throat.
* **Visceral leishmaniasis**: Fever, weight loss, hepatosplenomegaly, anemia, pancytopenia, hypergammaglobulinemia.

**Diagnosis Methods**

* **Microscopic examination**: Identification of amastigotes in tissue samples (skin biopsy, bone marrow, spleen aspirates).
* **Culture**: Growth of promastigotes in specialized media.
* **Serology**: Antibody detection (e.g., rK39 antigen test) especially for visceral leishmaniasis.
* **Molecular tests**: PCR assays for species identification.

**Treatment Options**

* **Cutaneous leishmaniasis**: Local therapies (cryotherapy, thermotherapy), systemic treatments with pentavalent antimonials, amphotericin B, miltefosine.
* **Visceral leishmaniasis**: Liposomal amphotericin B is first-line; alternatives include miltefosine, pentavalent antimonials.
* **Mucocutaneous leishmaniasis**: Prolonged systemic therapy with amphotericin B or antimonials.
* Treatment choice depends on species, geographic location, and patient factors.

**Prevention**

* Avoid sandfly bites using insect repellents, bed nets, protective clothing.
* Vector control programs targeting sandfly populations.
* No effective vaccine currently available.
* Early diagnosis and treatment to reduce transmission.

**Prognosis**

* Cutaneous leishmaniasis usually self-limited but may cause disfiguring scars.
* Visceral leishmaniasis is fatal if untreated but curable with appropriate therapy.
* Mucocutaneous form can cause severe morbidity.

**Possible Complications**

* Secondary bacterial infections of skin ulcers.
* Mucosal destruction leading to airway obstruction.
* Post-kala-azar dermal leishmaniasis (PKDL) after visceral leishmaniasis treatment.
* Relapse or treatment failure, especially in immunocompromised patients.

**When to See a Doctor / Red Flags**

* Persistent skin ulcers not healing.
* Fever, weight loss, and abdominal swelling.
* Nasal or oral mucosal lesions.
* History of travel or residence in endemic areas.

**Differential Diagnosis**

* Cutaneous ulcers from bacterial or fungal infections.
* Tuberculosis, leprosy, or other granulomatous diseases.
* Malignancies in chronic lesions.

**Recent Guidelines or Updates**

* WHO recommends liposomal amphotericin B as first-line for visceral leishmaniasis.
* Miltefosine approved for oral treatment in some regions.
* Integrated vector management and surveillance emphasized.
* Research ongoing for vaccine development.

**Epidemiology and Statistics**

* Estimated 700,000 to 1 million new cases annually globally.
* Visceral leishmaniasis causes approximately 20,000 to 40,000 deaths per year.
* Most affected regions include India, Bangladesh, Sudan, Brazil, and Ethiopia.

**References**

* **INFECTIOUS DISEASE (COMPLEX INFECTION: HIV, TROPICAL DISEASES)**

**Rickettsial Diseases (e.g., Scrub Typhus, Murine Typhus)**

**Definition**

Rickettsial diseases are a group of infections caused by obligate intracellular bacteria of the genus *Rickettsia* and related genera, transmitted by arthropod vectors such as ticks, mites, fleas, and lice. They typically cause febrile illnesses with rash and systemic symptoms.

**Causes and Transmission**

* **Cause**: Various *Rickettsia* species, including:
  + *Orientia tsutsugamushi* (Scrub typhus)
  + *Rickettsia typhi* (Murine typhus)
  + *Rickettsia rickettsii* (Rocky Mountain spotted fever)
* **Transmission**:
  + Scrub typhus: bite of infected chigger mites (*Leptotrombidium*).
  + Murine typhus: fleas from rodents.
  + Other rickettsioses: ticks, lice.
* **Epidemiology**:
  + Scrub typhus is endemic in the “tsutsugamushi triangle” (Asia-Pacific region).
  + Murine typhus occurs worldwide, especially in urban areas with rodent infestations.

**Signs and Symptoms**

* Sudden onset of high fever, headache, myalgia, malaise.
* Maculopapular or petechial rash developing 2–5 days after fever onset (may be absent in some cases).
* Eschar (a necrotic black scab at the bite site) is characteristic of scrub typhus.
* Lymphadenopathy, conjunctival injection, hepatosplenomegaly may occur.
* Severe cases can involve pneumonitis, meningoencephalitis, myocarditis, multi-organ failure.

**Diagnosis Methods**

* **Clinical diagnosis** supported by epidemiological context and presence of eschar.
* **Serologic tests**: Indirect immunofluorescence assay (IFA) is gold standard.
* **PCR**: Detection of rickettsial DNA in blood or tissue samples.
* **Weil-Felix test**: Older, less specific test rarely used now.

**Treatment Options**

* **First-line**: Doxycycline (100 mg twice daily for 7–14 days).
* **Alternatives**: Azithromycin, chloramphenicol (for pregnant women or doxycycline intolerance).
* Early treatment reduces complications and mortality.

**Prevention**

* Avoidance of vector habitats (wooded, grassy areas).
* Use of insect repellents (DEET), protective clothing.
* Rodent control to reduce flea populations.
* No vaccines currently available.

**Prognosis**

* Generally good with prompt antibiotic treatment.
* Mortality varies by species and treatment delay; untreated scrub typhus mortality up to 30%.
* Murine typhus usually mild but can be severe in elderly or immunocompromised.

**Possible Complications**

* Pneumonitis, acute respiratory distress syndrome (ARDS).
* Meningoencephalitis, seizures.
* Myocarditis, renal failure.
* Secondary bacterial infections.

**When to See a Doctor / Red Flags**

* Fever with rash and eschar after exposure to endemic areas.
* Severe headache, confusion, difficulty breathing.
* Persistent high fever unresponsive to common antibiotics.

**Differential Diagnosis**

* Other febrile illnesses with rash: dengue, malaria, typhoid, leptospirosis.
* Viral exanthems.
* Other tick-borne diseases.

**Recent Guidelines or Updates**

* CDC and WHO emphasize early empirical doxycycline treatment in suspected cases.
* Increasing recognition of scrub typhus outside traditional endemic zones due to travel and climate change.
* Research ongoing for vaccine development.

**Epidemiology and Statistics**

* Scrub typhus causes an estimated 1 million cases annually worldwide.
* Murine typhus is endemic in urban areas globally with sporadic outbreaks.
* High incidence in Asia-Pacific, parts of Africa, and the Americas.

**References**

* CDC. Rickettsial Diseases. 2024.
* WHO. Scrub Typhus Fact Sheet. 2023.
* Mayo Clinic. Rickettsial Infections Overview. 2024.
* CDC DPDx. Rickettsial Diseases.
* Parola P, Raoult D. “Tick-borne bacterial diseases.” Clin Microbiol Rev. 2023.
* UpToDate. Overview of Rickettsial Infections. 2024.

**Dengue Fever**

**Definition**

Dengue fever is an acute viral illness caused by the dengue virus, a flavivirus transmitted primarily by *Aedes aegypti* mosquitoes. It ranges from a mild febrile illness to severe dengue hemorrhagic fever and dengue shock syndrome.

**Causes and Transmission**

* **Cause**: Infection with one of four dengue virus serotypes (DENV-1 to DENV-4).
* **Transmission**: Bite of infected *Aedes aegypti* and *Aedes albopictus* mosquitoes, active during daytime.
* **Epidemiology**:
  + Endemic in tropical and subtropical regions worldwide, including Asia, Latin America, Africa, and the Caribbean.
  + Estimated 100–400 million infections annually, with about 6.5 million reported cases in 2023.
  + Affects all age groups, with higher risk of severe disease in secondary infections with different serotypes.

**Signs and Symptoms**

* Sudden onset high fever (40°C/104°F), severe headache, retro-orbital pain, myalgia, arthralgia (“breakbone fever”).
* Rash (maculopapular or petechial), nausea, vomiting, and mild bleeding (e.g., gums, nose).
* Severe dengue: plasma leakage, hemorrhage, organ impairment, shock.
* Warning signs: abdominal pain, persistent vomiting, mucosal bleeding, lethargy, hepatomegaly.

**Diagnosis Methods**

* **Laboratory tests**:
  + NS1 antigen detection (early infection).
  + Dengue IgM and IgG antibody serology.
  + PCR for viral RNA detection.
* **Hematology**: Leukopenia, thrombocytopenia, hemoconcentration in severe cases.

**Treatment Options**

* No specific antiviral treatment.
* Supportive care: adequate hydration, fever control with acetaminophen (avoid NSAIDs).
* Close monitoring for warning signs and complications.
* Hospitalization for severe dengue for fluid management and supportive care.

**Prevention**

* Vector control: elimination of mosquito breeding sites, insecticide spraying.
* Personal protection: insect repellents, window screens, bed nets.
* Vaccine: Dengvaxia licensed in some countries for seropositive individuals; other vaccines under development.
* Community education and surveillance.

**Prognosis**

* Most cases are self-limited with full recovery.
* Severe dengue can be fatal if not promptly managed.
* Early recognition and supportive care reduce mortality to <1%.

**Possible Complications**

* Dengue hemorrhagic fever with bleeding and plasma leakage.
* Dengue shock syndrome causing circulatory failure.
* Organ failure (liver, heart, brain).
* Secondary bacterial infections.

**When to See a Doctor / Red Flags**

* High fever with severe headache and body pain.
* Bleeding from gums, nose, or under the skin.
* Persistent vomiting, abdominal pain, difficulty breathing.
* Signs of shock: cold clammy skin, rapid weak pulse, restlessness.

**Differential Diagnosis**

* Malaria, chikungunya, Zika virus infection.
* Typhoid fever, leptospirosis, influenza.
* Other viral hemorrhagic fevers.

**Recent Guidelines or Updates**

* WHO 2023 guidelines emphasize early diagnosis, risk stratification, and fluid management.
* CDC updates on vaccine recommendations and vector control strategies.
* Research ongoing on antiviral drugs and improved vaccines.

**Epidemiology and Statistics**

* Approximately 100–400 million dengue infections annually worldwide.
* 6.5 million cases reported in 2023.
* Increasing incidence linked to urbanization, climate change, and global travel.

**References**

1. World Health Organization. Dengue and severe dengue. Fact sheet. 2023.
2. CDC. Dengue. 2024.
3. Mayo Clinic. Dengue Fever. 2023.
4. Bhatt S, et al. The global distribution and burden of dengue. Nature. 2013.
5. WHO. Dengue Guidelines for Diagnosis, Treatment, Prevention and Control. 2023.
6. UpToDate. Dengue virus infection. 2024.

**Yellow Fever**

**Definition**

Yellow fever is an acute viral hemorrhagic disease caused by the yellow fever virus, a flavivirus transmitted by *Aedes aegypti* mosquitoes. It is characterized by fever, jaundice, bleeding, and can be fatal.

**Causes and Transmission**

* **Cause**: Infection with yellow fever virus.
* **Transmission**:
  + Urban cycle: *Aedes aegypti* mosquitoes transmitting between humans.
  + Sylvatic (jungle) cycle: transmission between non-human primates and forest mosquitoes (*Haemagogus* and *Sabethes* species), with incidental human infection.
* **Epidemiology**:
  + Endemic in tropical regions of Africa and South America.
  + Approximately 200,000 cases and 30,000 deaths annually worldwide (WHO estimates).
  + Outbreaks occur in urban and jungle settings.

**Signs and Symptoms**

* **Incubation**: 3–6 days.
* **Initial phase**: Sudden onset fever, chills, headache, backache, muscle aches, nausea, vomiting.
* **Toxic phase (in ~15% of cases)**: Return of high fever, jaundice (yellowing of skin and eyes), abdominal pain, bleeding (from mouth, nose, eyes, stomach), kidney failure, shock.
* **Recovery or death**: Toxic phase lasts 7–10 days; mortality in severe cases is 20–50%.

**Diagnosis Methods**

* **Laboratory tests**:
  + Detection of yellow fever virus RNA by PCR.
  + Serology: IgM antibodies and neutralizing antibodies.
  + Virus isolation in specialized labs.
* **Clinical diagnosis** based on symptoms and epidemiological context.

**Treatment Options**

* No specific antiviral treatment available.
* Supportive care: hydration, blood transfusions, management of shock and organ failure.
* Intensive care may be required for severe cases.

**Prevention**

* **Vaccination**: Single-dose live attenuated yellow fever vaccine provides lifelong immunity and is highly effective.
* **Vector control**: Reducing mosquito breeding sites, insecticide spraying.
* **Travel precautions**: Vaccination required for travel to endemic areas; proof of vaccination may be required.
* **Public health measures**: Surveillance and outbreak response.

**Prognosis**

* Most infected people recover fully.
* Severe cases have high mortality (20–50%).
* Survivors of toxic phase may have prolonged convalescence.

**Possible Complications**

* Hemorrhagic manifestations causing severe bleeding.
* Multi-organ failure (liver, kidneys, heart).
* Secondary infections.
* Death in severe cases.

**When to See a Doctor / Red Flags**

* Sudden high fever with jaundice and bleeding symptoms.
* Severe abdominal pain, vomiting blood or black stools.
* Signs of shock: rapid heartbeat, low blood pressure, confusion.

**Differential Diagnosis**

* Other viral hemorrhagic fevers (Ebola, Marburg).
* Severe malaria.
* Hepatitis viruses.
* Leptospirosis.

**Recent Guidelines or Updates**

* WHO recommends vaccination as the primary prevention tool.
* International Health Regulations require proof of vaccination for travelers to endemic areas.
* Recent outbreaks have underscored the need for improved vaccination coverage and vector control.
* Research ongoing for antiviral therapies and improved vaccines.

**Epidemiology and Statistics**

* Estimated 200,000 cases and 30,000 deaths annually worldwide.
* Endemic in 47 countries in Africa and Central/South America.
* Vaccination coverage gaps contribute to periodic outbreaks.

**References**

1. World Health Organization. Yellow Fever Fact Sheet. 2023.
2. CDC. Yellow Fever. 2024.
3. Mayo Clinic. Yellow Fever Overview. 2023.
4. Monath TP, Vasconcelos PF. Yellow fever. J Clin Virol. 2015.
5. UpToDate. Yellow fever virus infection. 2024.

**Zika Virus Infection**

**Definition**

Zika virus infection is a mosquito-borne viral illness caused by the Zika virus, a flavivirus primarily transmitted by *Aedes* mosquitoes. It is usually mild but can cause serious birth defects when contracted during pregnancy.

**Causes and Transmission**

* **Cause**: Infection with Zika virus.
* **Transmission**:
  + Primarily through the bite of infected *Aedes aegypti* and *Aedes albopictus* mosquitoes.
  + Sexual transmission, blood transfusion, and vertical transmission (mother to fetus) have been documented.
* **Epidemiology**:
  + First identified in Uganda in 1947; outbreaks in Africa, Asia, Pacific Islands, and the Americas.
  + Major outbreak in the Americas in 2015–2016.
  + Endemic in tropical and subtropical regions.

**Signs and Symptoms**

* Incubation period: 3–14 days.
* Usually mild or asymptomatic.
* Symptoms include: low-grade fever, rash, conjunctivitis, arthralgia, myalgia, headache.
* Symptoms typically last 2–7 days.
* Severe disease is rare.

**Diagnosis Methods**

* **Laboratory tests**:
  + RT-PCR for viral RNA detection in blood or urine (most reliable within first week of illness).
  + Serology (IgM antibodies), but cross-reactivity with other flaviviruses (e.g., dengue) limits specificity.
* **Clinical diagnosis** based on symptoms and exposure history.

**Treatment Options**

* No specific antiviral treatment.
* Supportive care: rest, hydration, acetaminophen for fever and pain.
* Avoid NSAIDs until dengue is ruled out due to bleeding risk.

**Prevention**

* Avoid mosquito bites using insect repellents, protective clothing, and bed nets.
* Control mosquito breeding sites.
* Safe sex practices to prevent sexual transmission.
* Pregnant women advised to avoid travel to endemic areas.
* No vaccine currently licensed, though several candidates are in development.

**Prognosis**

* Generally excellent; most recover fully without complications.
* Infection during pregnancy can cause congenital Zika syndrome, including microcephaly and other severe fetal brain defects.

**Possible Complications**

* Congenital Zika syndrome (microcephaly, brain abnormalities, eye defects).
* Guillain-Barré syndrome (rare neurological complication).
* Other neurological disorders reported.

**When to See a Doctor / Red Flags**

* Fever with rash and conjunctivitis after travel to or residence in endemic areas.
* Pregnant women with possible exposure should seek medical advice promptly.
* Neurological symptoms such as weakness or paralysis.

**Differential Diagnosis**

* Dengue fever, chikungunya, measles, rubella, parvovirus B19, other viral exanthems.

**Recent Guidelines or Updates**

* WHO and CDC recommend mosquito bite prevention and sexual transmission precautions.
* CDC provides guidelines for pregnant women and newborn screening.
* Research ongoing for vaccines and therapeutics.

**Epidemiology and Statistics**

* Estimated hundreds of thousands of cases during the 2015–2016 Americas outbreak.
* Endemic in parts of Africa, Asia, and the Americas.
* Congenital Zika syndrome cases documented mainly in Brazil and neighboring countries.

**References**

1. World Health Organization. Zika Virus Fact Sheet. 2023.
2. CDC. Zika Virus. 2024.
3. Mayo Clinic. Zika Virus Infection. 2023.
4. Musso D, Gubler DJ. Zika Virus. Clin Microbiol Rev. 2016.
5. UpToDate. Zika virus infection. 2024.

**Chikungunya Virus Infection**

**Definition**

Chikungunya is a mosquito-borne viral disease caused by the chikungunya virus (CHIKV), an alphavirus transmitted primarily by *Aedes* mosquitoes. It is characterized by fever and severe joint pain.

**Causes and Transmission**

* **Cause**: Infection with chikungunya virus.
* **Transmission**:
  + Bite of infected *Aedes aegypti* and *Aedes albopictus* mosquitoes.
  + No evidence of human-to-human transmission except vertical transmission (mother to child) and rare blood transfusion cases.
* **Epidemiology**:
  + Endemic in Africa, Asia, Indian subcontinent, and outbreaks reported in the Americas and Europe.
  + First identified in Tanzania in 1952; large outbreaks since 2004.
  + Transmitted in urban and sylvatic cycles.

**Signs and Symptoms**

* Incubation period: 2–12 days.
* Sudden onset of high fever, severe polyarthralgia/polyarthritis (often symmetrical and debilitating), headache, muscle pain, rash.
* Joint pain can persist for months to years in some patients (chronic arthritis).
* Other symptoms: fatigue, nausea, conjunctivitis.

**Diagnosis Methods**

* **Laboratory tests**:
  + RT-PCR for viral RNA detection during acute phase (first week).
  + Serology: detection of IgM and IgG antibodies.
* **Clinical diagnosis** supported by epidemiological context and symptoms.

**Treatment Options**

* No specific antiviral treatment.
* Supportive care: rest, hydration, analgesics, and antipyretics (acetaminophen preferred).
* NSAIDs used cautiously after ruling out dengue.
* Physical therapy may be needed for chronic joint symptoms.

**Prevention**

* Avoid mosquito bites using insect repellents, protective clothing, and bed nets.
* Vector control: elimination of mosquito breeding sites, insecticide spraying.
* No licensed vaccine currently available, though candidates are in development.

**Prognosis**

* Most patients recover fully within weeks; some develop chronic joint pain lasting months or years.
* Mortality is rare but may occur in elderly or those with comorbidities.

**Possible Complications**

* Chronic arthritis resembling rheumatoid arthritis.
* Neurological complications (rare): encephalitis, Guillain-Barré syndrome.
* Neonatal infections from vertical transmission.

**When to See a Doctor / Red Flags**

* Severe joint pain limiting daily activities.
* Persistent or worsening symptoms beyond acute phase.
* Neurological symptoms such as weakness or altered consciousness.

**Differential Diagnosis**

* Dengue fever, Zika virus infection, rheumatoid arthritis, other viral arthritides.

**Recent Guidelines or Updates**

* WHO recommends supportive care and vector control as primary measures.
* Research ongoing for vaccines and antiviral agents.
* CDC provides guidance on diagnosis, treatment, and prevention.

**Epidemiology and Statistics**

* Millions of cases reported worldwide since 2004 outbreaks.
* Significant outbreaks in the Caribbean and Americas since 2013.
* Endemic in Africa and Asia with periodic epidemics.

**References**

1. World Health Organization. Chikungunya Fact Sheet. 2023.
2. CDC. Chikungunya Virus. 2024.
3. Mayo Clinic. Chikungunya Virus Infection. 2023.
4. Weaver SC, Lecuit M. Chikungunya virus and the global spread of a mosquito-borne disease. N Engl J Med. 2015.
5. UpToDate. Chikungunya virus infection. 2024.

**Rift Valley Fever (RVF)**

**Definition**

Rift Valley Fever is a viral zoonotic disease caused by the Rift Valley fever virus (RVFV), a Phlebovirus transmitted primarily by mosquitoes and through contact with infected livestock.

**Causes and Transmission**

* **Cause**: Infection with Rift Valley fever virus, a member of the *Phlebovirus* genus.
* **Transmission**:
  + Bite of infected mosquitoes, mainly *Aedes* and *Culex* species.
  + Direct contact with blood, organs, or bodily fluids of infected animals (livestock such as sheep, cattle, goats).
  + Laboratory exposure and aerosol transmission possible.
* **Epidemiology**:
  + Endemic in sub-Saharan Africa and Arabian Peninsula.
  + Outbreaks often follow heavy rainfall and flooding which increase mosquito populations.
  + Affects both animals and humans; livestock outbreaks cause significant economic losses.

**Signs and Symptoms**

* **Incubation period**: 2–6 days.
* Most human infections are mild or asymptomatic.
* **Mild disease**: Fever, headache, muscle pain, joint pain, photophobia, nausea, vomiting.
* **Severe disease (in ~1-2%)**:
  + Hemorrhagic fever with bleeding, jaundice, liver failure.
  + Encephalitis with neurological symptoms.
  + Ocular disease causing vision loss.
* Symptoms usually last 4–7 days.

**Diagnosis Methods**

* **Laboratory tests**:
  + RT-PCR for viral RNA detection.
  + Serology: detection of IgM and IgG antibodies by ELISA.
  + Virus isolation in specialized labs.
* **Clinical diagnosis** supported by epidemiological exposure.

**Treatment Options**

* No specific antiviral treatment available.
* Supportive care: hydration, pain and fever management, treatment of complications.
* Hospitalization for severe cases with bleeding or neurological involvement.

**Prevention**

* Avoid mosquito bites using repellents, protective clothing, and bed nets.
* Control mosquito breeding sites.
* Use protective equipment when handling livestock or animal products.
* Vaccination of livestock in endemic areas to reduce transmission.
* No licensed human vaccine widely available; some vaccines used in research or veterinary settings.

**Prognosis**

* Most recover fully without complications.
* Severe cases can be fatal (case fatality rate up to 10–20%).
* Long-term sequelae possible in neurological and ocular cases.

**Possible Complications**

* Hemorrhagic fever with bleeding and shock.
* Encephalitis leading to neurological deficits.
* Vision loss due to retinitis or optic neuritis.
* Death in severe cases.

**When to See a Doctor / Red Flags**

* High fever with bleeding, jaundice, or neurological symptoms after exposure to endemic areas or livestock.
* Sudden vision changes.
* Severe headache or confusion.

**Differential Diagnosis**

* Other viral hemorrhagic fevers (Ebola, Marburg, Crimean-Congo).
* Malaria, leptospirosis, typhoid fever.
* Other encephalitis-causing viruses.

**Recent Guidelines or Updates**

* WHO emphasizes integrated surveillance of human and animal cases.
* CDC recommends protective measures for people in contact with livestock.
* Research ongoing for human vaccines and antiviral therapies.
* Outbreak response includes vector control and livestock vaccination.

**Epidemiology and Statistics**

* Outbreaks occur cyclically, often linked to heavy rainfall and flooding.
* Thousands of human cases reported during outbreaks, with hundreds of deaths.
* Economic impact significant due to livestock losses.

**References**

1. World Health Organization. Rift Valley Fever Fact Sheet. 2023.
2. CDC. Rift Valley Fever. 2024.
3. Mayo Clinic. Rift Valley Fever Overview. 2023.
4. Bird BH, et al. Rift Valley fever virus. J Am Vet Med Assoc. 2017.
5. UpToDate. Rift Valley fever virus infection. 2024.

**Crimean-Congo Hemorrhagic Fever (CCHF)**

**Definition**

Crimean-Congo Hemorrhagic Fever is a severe tick-borne viral hemorrhagic fever caused by the Crimean-Congo hemorrhagic fever virus (CCHFV), a Nairovirus of the family *Bunyaviridae*. It is characterized by sudden onset of fever, hemorrhagic manifestations, and high mortality.

**Causes and Transmission**

* **Cause**: Infection with Crimean-Congo hemorrhagic fever virus (CCHFV).
* **Transmission**:
  + Bite of infected *Hyalomma* ticks (primary vector).
  + Contact with blood or tissues of infected livestock or humans.
  + Nosocomial transmission through exposure to infected body fluids.
* **Epidemiology**:
  + Endemic in Africa, the Balkans, the Middle East, and parts of Asia.
  + Cases often occur in agricultural workers, slaughterhouse workers, healthcare workers.

**Signs and Symptoms**

* **Incubation period**: 1–13 days depending on mode of transmission.
* Sudden onset of high fever, headache, muscle aches, dizziness, neck pain, backache.
* Nausea, vomiting, diarrhea, abdominal pain.
* Hemorrhagic phase (2–4 days after onset): petechiae, ecchymoses, bleeding from gums, nose, gastrointestinal tract.
* Hepatomegaly, jaundice, hypotension, shock.
* Severe cases may progress to multi-organ failure and death.

**Diagnosis Methods**

* **Laboratory tests**:
  + RT-PCR for viral RNA detection (early diagnosis).
  + Serology: detection of IgM and IgG antibodies by ELISA.
  + Virus isolation in high-containment labs.
* **Clinical diagnosis** supported by exposure history and symptoms.

**Treatment Options**

* No specific antiviral approved; ribavirin used off-label with some evidence of benefit.
* Supportive care: fluid management, blood transfusions, management of bleeding and organ failure.
* Strict infection control to prevent nosocomial spread.

**Prevention**

* Avoid tick bites using protective clothing and repellents.
* Control of tick populations in livestock.
* Safe handling of livestock and animal products.
* Use of personal protective equipment (PPE) in healthcare settings.
* No licensed vaccine widely available; some vaccines used in limited settings.

**Prognosis**

* Case fatality rate ranges from 10% to 40%.
* Early supportive care improves outcomes.
* Survivors may have prolonged convalescence.

**Possible Complications**

* Severe hemorrhage and shock.
* Multi-organ failure including liver, kidney, and lung failure.
* Secondary infections.

**When to See a Doctor / Red Flags**

* Sudden high fever with bleeding symptoms after tick exposure or contact with livestock.
* Rapid deterioration with bleeding or shock.
* Healthcare workers with exposure to infected patients.

**Differential Diagnosis**

* Other viral hemorrhagic fevers (Ebola, Marburg, Lassa).
* Severe bacterial sepsis.
* Dengue hemorrhagic fever.
* Malaria with severe complications.

**Recent Guidelines or Updates**

* WHO and CDC recommend ribavirin treatment in severe cases and emphasize strict infection control.
* Surveillance and tick control programs are critical in endemic areas.
* Research ongoing for vaccines and antiviral therapies.

**Epidemiology and Statistics**

* Endemic in over 30 countries across Africa, Asia, and Europe.
* Hundreds to thousands of cases reported annually, often with outbreaks.
* High-risk occupations include farmers, abattoir workers, and healthcare personnel.

**References**

1. World Health Organization. Crimean-Congo Hemorrhagic Fever Fact Sheet. 2023.
2. CDC. Crimean-Congo Hemorrhagic Fever. 2024.
3. Mayo Clinic. Crimean-Congo Hemorrhagic Fever Overview. 2023.
4. Ergönül Ö. Crimean-Congo haemorrhagic fever. Lancet Infect Dis. 2006.
5. UpToDate. Crimean-Congo hemorrhagic fever virus infection. 2024.

**Ebola Virus Disease (EVD)**

**Definition**

Ebola virus disease is a severe, often fatal hemorrhagic fever caused by infection with Ebola virus, a member of the *Filoviridae* family. It is characterized by sudden onset of fever, hemorrhagic symptoms, and multi-organ failure.

**Causes and Transmission**

* **Cause**: Infection with Ebola virus (several species including Zaire ebolavirus, Sudan ebolavirus).
* **Transmission**:
  + Direct contact with blood, secretions, organs, or other bodily fluids of infected people or animals (e.g., fruit bats, primates).
  + Contaminated surfaces and materials (e.g., bedding, needles).
  + Human-to-human transmission via broken skin or mucous membranes.
* **Epidemiology**:
  + Endemic in Central and West Africa.
  + Major outbreaks include 2014–2016 West Africa epidemic and periodic flare-ups.

**Signs and Symptoms**

* **Incubation period**: 2–21 days (average 8–10 days).
* Sudden onset of fever, fatigue, muscle pain, headache, sore throat.
* Followed by vomiting, diarrhea, rash, impaired kidney and liver function.
* Internal and external bleeding (hemorrhagic manifestations) in severe cases.
* Multi-organ failure and shock may develop.

**Diagnosis Methods**

* **Laboratory tests**:
  + RT-PCR for viral RNA detection (gold standard).
  + Antigen detection tests.
  + Serology for antibodies (IgM, IgG).
* Diagnosis requires high-containment labs due to biosafety risks.

**Treatment Options**

* No specific antiviral cure; supportive care is critical.
* Supportive treatment includes fluid replacement, electrolyte balance, oxygen therapy, treatment of secondary infections.
* Recently approved treatments include monoclonal antibodies (e.g., Inmazeb, Ebanga) and antiviral remdesivir.
* Experimental vaccines (rVSV-ZEBOV) have been used in outbreak control.

**Prevention**

* Avoid contact with infected individuals and animals.
* Use of personal protective equipment (PPE) for healthcare workers.
* Safe burial practices.
* Vaccination of at-risk populations and contacts during outbreaks.
* Community education and surveillance.

**Prognosis**

* Case fatality rate varies from 25% to 90% depending on outbreak and care quality.
* Early supportive care improves survival.
* Survivors may have long-term complications including joint pain, vision problems, and psychological effects.

**Possible Complications**

* Severe hemorrhage and shock.
* Multi-organ failure.
* Secondary infections.
* Post-Ebola syndrome in survivors.

**When to See a Doctor / Red Flags**

* Sudden fever with vomiting, diarrhea, and bleeding after exposure to endemic areas or infected persons.
* Rapid deterioration with signs of shock or hemorrhage.
* Healthcare workers with exposure to Ebola patients.

**Differential Diagnosis**

* Other viral hemorrhagic fevers (Marburg, Lassa).
* Malaria, typhoid fever, meningococcemia.
* Severe bacterial sepsis.

**Recent Guidelines or Updates**

* WHO and CDC provide updated protocols for outbreak response, infection control, and vaccination.
* Approval of monoclonal antibody therapies has improved treatment options.
* Continued emphasis on early detection and supportive care.

**Epidemiology and Statistics**

* Since discovery in 1976, over 35 outbreaks reported.
* 2014–2016 West Africa outbreak caused over 28,000 cases and 11,000 deaths.
* Sporadic outbreaks continue in Central Africa.

**References**

1. World Health Organization. Ebola Virus Disease Fact Sheet. 2023.
2. CDC. Ebola (Ebola Virus Disease). 2024.
3. Mayo Clinic. Ebola Virus Disease. 2023.
4. Kuhn JH et al. Filoviruses: A Compendium of 40 Years of Epidemiological, Clinical, and Laboratory Studies. 2019.
5. UpToDate. Ebola virus disease. 2024.

**Marburg Virus Disease (MVD)**

**Definition**

Marburg virus disease is a severe hemorrhagic fever caused by Marburg virus, a member of the *Filoviridae* family closely related to Ebola virus. It causes acute febrile illness with high fatality rates.

**Causes and Transmission**

* **Cause**: Infection with Marburg virus.
* **Transmission**:
  + Contact with bodily fluids (blood, saliva, vomit, urine) of infected humans or animals.
  + Exposure to infected fruit bats (*Rousettus aegyptiacus*), the natural reservoir.
  + Human-to-human transmission through direct contact or contaminated fomites.
* **Epidemiology**:
  + Endemic in parts of Africa, with outbreaks in Uganda, Angola, Democratic Republic of Congo.
  + Sporadic outbreaks with high mortality.

**Signs and Symptoms**

* **Incubation period**: 2–21 days.
* Sudden onset of fever, chills, headache, myalgia.
* Progression to severe hemorrhagic manifestations: rash, bleeding from gums, nose, gastrointestinal tract.
* Multi-organ failure, shock, and death in severe cases.

**Diagnosis Methods**

* **Laboratory tests**:
  + RT-PCR for viral RNA detection.
  + Virus isolation in high-containment labs.
  + Serology for antibodies.
* Requires biosafety level 4 (BSL-4) facilities.

**Treatment Options**

* No specific antiviral treatment.
* Supportive care: fluid replacement, oxygen therapy, treatment of secondary infections.
* Experimental therapies under investigation.

**Prevention**

* Avoid contact with fruit bats and infected persons.
* Use of PPE in healthcare settings.
* Safe burial practices.
* No licensed vaccine currently available, though research is ongoing.

**Prognosis**

* Case fatality rate ranges from 24% to 88% depending on outbreak and care.
* Early supportive care improves survival chances.

**Possible Complications**

* Severe hemorrhage and shock.
* Multi-organ failure.
* Long convalescence with possible neurological sequelae.

**When to See a Doctor / Red Flags**

* Sudden fever with bleeding symptoms after exposure to endemic areas or infected individuals.
* Rapid clinical deterioration with hemorrhage or shock.

**Differential Diagnosis**

* Ebola virus disease.
* Other viral hemorrhagic fevers.
* Severe bacterial infections.

**Recent Guidelines or Updates**

* WHO and CDC emphasize infection control and outbreak containment.
* Research ongoing for vaccines and therapeutics.
* Experimental vaccines have shown promise in preclinical studies.

**Epidemiology and Statistics**

* Several outbreaks since first identification in 1967.
* Case fatality rates vary widely but often exceed 50%.
* Endemic in Central and East Africa.

**References**

1. World Health Organization. Marburg Virus Disease Fact Sheet. 2023.
2. CDC. Marburg Hemorrhagic Fever. 2024.
3. Mayo Clinic. Marburg Virus Disease. 2023.
4. Towner JS et al. Marburg virus infection. Lancet. 2012.
5. UpToDate. Marburg virus disease. 2024.

**Lassa Fever**

**Definition**

Lassa fever is an acute viral hemorrhagic illness caused by Lassa virus, an arenavirus endemic in West Africa. It ranges from mild febrile illness to severe multi-organ disease with hemorrhagic manifestations.

**Causes and Transmission**

* **Cause**: Infection with Lassa virus.
* **Transmission**:
  + Exposure to urine or feces of infected *Mastomys* rats (multimammate rat), the natural reservoir.
  + Human-to-human transmission via contact with blood, secretions, or bodily fluids of infected persons.
  + Nosocomial transmission is common in healthcare settings without proper precautions.
* **Epidemiology**:
  + Endemic in Nigeria, Sierra Leone, Liberia, Guinea, and other parts of West Africa.
  + Estimated 100,000 to 300,000 infections and ~5,000 deaths annually.

**Signs and Symptoms**

* **Incubation period**: 6–21 days.
* Gradual onset of fever, weakness, headache, sore throat, muscle pain.
* Gastrointestinal symptoms: nausea, vomiting, diarrhea, abdominal pain.
* Severe cases: facial swelling, bleeding, respiratory distress, neurological symptoms (hearing loss, seizures).
* Approximately 80% of infections are mild or asymptomatic.

**Diagnosis Methods**

* **Laboratory tests**:
  + RT-PCR for viral RNA detection.
  + Serology: detection of IgM and IgG antibodies.
  + Virus isolation in high-containment labs.
* Diagnosis requires biosafety level 4 (BSL-4) facilities.

**Treatment Options**

* Early administration of ribavirin reduces mortality.
* Supportive care: hydration, electrolyte balance, treatment of complications.
* Isolation and infection control measures in healthcare settings.

**Prevention**

* Avoid contact with rodent excreta and contaminated food.
* Rodent control and improved sanitation.
* Use of personal protective equipment (PPE) in healthcare.
* No licensed vaccine currently available; research ongoing.

**Prognosis**

* Overall mortality ~1%, but up to 15–20% in hospitalized severe cases.
* Hearing loss is a common sequela in survivors.
* Early treatment improves outcomes significantly.

**Possible Complications**

* Hemorrhagic manifestations.
* Multi-organ failure.
* Neurological complications including deafness.
* Miscarriage and maternal death in pregnant women.

**When to See a Doctor / Red Flags**

* Fever with bleeding, facial swelling, or neurological symptoms after exposure in endemic areas.
* Severe abdominal pain, vomiting, or respiratory distress.

**Differential Diagnosis**

* Other viral hemorrhagic fevers (Ebola, Marburg, Crimean-Congo).
* Malaria, typhoid fever, bacterial sepsis.

**Recent Guidelines or Updates**

* WHO and CDC recommend ribavirin use and strict infection control.
* Surveillance and outbreak response critical in endemic regions.
* Vaccine development efforts ongoing.

**Epidemiology and Statistics**

* Estimated 100,000–300,000 cases annually in West Africa.
* Endemic in Nigeria, Sierra Leone, Liberia, Guinea, and surrounding countries.
* High risk for healthcare workers and pregnant women.

**References**

1. World Health Organization. Lassa Fever Fact Sheet. 2023.
2. CDC. Lassa Fever. 2024.
3. Mayo Clinic. Lassa Fever Overview. 2023.
4. McCormick JB et al. Lassa fever. Clin Infect Dis. 1987.
5. UpToDate. Lassa fever virus infection. 2024.

**Japanese Encephalitis (JE)**

**Definition**

Japanese encephalitis is a mosquito-borne viral infection caused by the Japanese encephalitis virus (JEV), a flavivirus. It primarily affects the central nervous system, causing inflammation of the brain (encephalitis).

**Causes and Transmission**

* **Cause**: Infection with Japanese encephalitis virus (JEV).
* **Transmission**:
  + Bite of infected *Culex* mosquitoes, especially *Culex tritaeniorhynchus*.
  + The virus circulates between mosquitoes and vertebrate hosts, mainly pigs and wading birds.
  + Humans are incidental dead-end hosts.
* **Epidemiology**:
  + Endemic in rural and agricultural areas of Asia and Western Pacific.
  + Approximately 68,000 clinical cases annually, with seasonal outbreaks during rainy seasons.

**Signs and Symptoms**

* **Incubation period**: 5–15 days.
* Most infections (~99%) are asymptomatic or mild.
* Symptomatic cases present with sudden onset fever, headache, vomiting, confusion.
* Neurological signs: seizures, paralysis, altered consciousness, coma.
* Mortality rate among encephalitis cases: 20–30%.
* Long-term neurological sequelae common in survivors.

**Diagnosis Methods**

* **Laboratory tests**:
  + Detection of JEV-specific IgM antibodies in cerebrospinal fluid (CSF) or serum by ELISA (gold standard).
  + PCR and virus isolation in specialized labs.
* **Clinical diagnosis** supported by epidemiological context.

**Treatment Options**

* No specific antiviral treatment available.
* Supportive care: hospitalization, management of complications (seizures, increased intracranial pressure).
* Rehabilitation for neurological sequelae.

**Prevention**

* Vaccination: several effective vaccines available (inactivated, live attenuated).
* Vector control: reducing mosquito populations and avoiding bites using repellents, bed nets.
* Public health education in endemic areas.

**Prognosis**

* High mortality among severe cases.
* Up to 30–50% of survivors have permanent neurological or psychiatric disabilities.

**Possible Complications**

* Permanent neurological deficits: paralysis, cognitive impairment, movement disorders.
* Seizures and epilepsy.
* Behavioral and psychiatric problems.

**When to See a Doctor / Red Flags**

* Sudden high fever with headache and neurological symptoms (confusion, seizures).
* Signs of altered consciousness or paralysis.
* Recent travel to or residence in endemic areas.

**Differential Diagnosis**

* Other causes of viral encephalitis (herpes simplex virus, West Nile virus).
* Bacterial meningitis, cerebral malaria.
* Other flavivirus infections.

**Recent Guidelines or Updates**

* WHO recommends vaccination in endemic areas and during outbreaks.
* Routine immunization programs have reduced incidence in many countries.
* Ongoing surveillance and vector control remain critical.

**Epidemiology and Statistics**

* Estimated 68,000 clinical cases annually worldwide.
* Endemic in 24 countries in Asia and Western Pacific.
* Most cases occur in children under 15 years.

**References**

1. World Health Organization. Japanese Encephalitis Fact Sheet. 2023.
2. CDC. Japanese Encephalitis. 2024.
3. Mayo Clinic. Japanese Encephalitis Overview. 2023.
4. Campbell GL et al. Estimated global incidence of Japanese encephalitis: a systematic review. Bull World Health Organ. 2011.
5. UpToDate. Japanese encephalitis virus infection. 2024.

**West Nile Virus Infection**

**Definition**

West Nile virus (WNV) infection is a mosquito-borne viral disease caused by West Nile virus, a flavivirus. It can range from asymptomatic infection to severe neuroinvasive disease.

**Causes and Transmission**

* **Cause**: Infection with West Nile virus.
* **Transmission**:
  + Bite of infected *Culex* mosquitoes, which acquire the virus from infected birds (primary reservoir).
  + Rarely via blood transfusion, organ transplantation, mother-to-child transmission, and laboratory exposure.
* **Epidemiology**:
  + Endemic in Africa, Europe, the Middle East, North America, and West Asia.
  + Introduced to North America in 1999; now widespread.
  + Seasonal outbreaks in summer and early fall.

**Signs and Symptoms**

* Most infections (~80%) are asymptomatic.
* **West Nile fever**: fever, headache, body aches, joint pains, vomiting, diarrhea, rash.
* **Neuroinvasive disease** (in <1%): meningitis, encephalitis, acute flaccid paralysis.
* Symptoms may include altered mental status, muscle weakness, seizures.

**Diagnosis Methods**

* **Laboratory tests**:
  + Detection of WNV-specific IgM antibodies in serum or cerebrospinal fluid (CSF) by ELISA (gold standard).
  + PCR and virus isolation in specialized labs.
* **Clinical diagnosis** supported by epidemiological context.

**Treatment Options**

* No specific antiviral treatment available.
* Supportive care: hospitalization, respiratory support, management of neurological complications.
* Rehabilitation for neurological deficits.

**Prevention**

* Avoid mosquito bites using repellents, protective clothing, and bed nets.
* Vector control: elimination of mosquito breeding sites, insecticide spraying.
* No human vaccine currently licensed; veterinary vaccines exist for horses.

**Prognosis**

* Most recover fully without symptoms.
* Neuroinvasive disease can cause long-term neurological impairment or death (mortality ~10%).
* Older adults and immunocompromised individuals at higher risk for severe disease.

**Possible Complications**

* Meningitis, encephalitis.
* Acute flaccid paralysis resembling poliomyelitis.
* Long-term neurological sequelae including cognitive dysfunction, muscle weakness.

**When to See a Doctor / Red Flags**

* Fever with severe headache, neck stiffness, confusion, muscle weakness.
* Sudden paralysis or altered consciousness.
* Recent exposure to mosquitoes in endemic areas.

**Differential Diagnosis**

* Other viral encephalitides (herpes simplex, Eastern equine encephalitis).
* Bacterial meningitis.
* Guillain-Barré syndrome.

**Recent Guidelines or Updates**

* CDC recommends mosquito bite prevention and surveillance.
* Research ongoing for vaccines and antiviral therapies.
* Public health measures focus on vector control and blood supply screening.

**Epidemiology and Statistics**

* Estimated tens of thousands of cases annually in the US alone.
* Widespread in Africa, Europe, Asia, and the Americas.
* First detected in the US in 1999; now endemic.

**References**

1. World Health Organization. West Nile Virus Fact Sheet. 2023.
2. CDC. West Nile Virus. 2024.
3. Mayo Clinic. West Nile Virus Infection. 2023.
4. Petersen LR, Brault AC, Nasci RS. West Nile virus: review of the literature. JAMA. 2013.
5. UpToDate. West Nile virus infection. 2024.