PREFACE

Understanding the medical challenges in tropical regions is important, particularly for personnel in the Armed Forces, who are often stationed abroad in far flung areas. In fact, the knowledge of tropical medicine can be as important as the ability to handle armed conflict and war. Tropical medicine encompasses not only the diseases found in tropical areas, but also diseases that occur among travellers arriving in Norway. Awareness about these topics is therefore of utmost importance. This has become increasingly evident in connection with recent outbreaks of the Ebola and Zika viruses.

Prevention is imperative, BEFORE travelling, DURING the assignment, and AFTER returning, when it is important to know which signs to look out for. Knowledge of tropical medicine is a prerequisite for understanding which precautions to follow, and which information to gather. Prevention of disease is vital for a good and healthy life, which in turn is important for combat capability. Equally important is the ability to ensure good health care after service or combat. This is summed up in FSAN’s vision:

Health for combat capability.

Based on their deep and broad scientific knowledge, as well as their practical experience from national and international operations, Fredrik S. Thorn and Harald Wiik have written a handy book that gives a clear and easily comprehensible summary of the subject. The book uses instructive illustrations and concise explanations. Antidotes and treatments are comprehensively listed, to facilitate user friendliness.

Enjoy reading about this important topic!

Jan Sommerfelt-Pettersen
Rear Admiral, Norwegian Surgeon General,
Norwegian Armed Forces Joint Medical Services
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**Cover Illustration:** Outside the hospital in Haydom, Tanzania.
Since the 1950s, the Armed Forces have served in many parts of the world where the risk of infection and disease is at least as great as the risk of direct harm from the armed conflict. As Norway continues to contribute to international operations, Norwegian soldiers and civilians will be exposed to sources of infection that they would not normally encounter at home. To reduce the risk of infection, it is important to understand how diseases are transferred and how they can be avoided. Because the sources of contagion are many and ubiquitous, it is imperative for everyone to be familiar with this information. The Armed Forces ensure that food and drink are as safe as possible, but outside the base additional care must be taken, because hygiene, sanitary conditions, food and drink may be very different from that in Norway.

The next sections give a brief description of sources and routes of infection.

The clearly dominant cause of tropical disease is the numerous types of parasites.

Endoparasites reside within the host’s
digestive tract, inner organs, or body fluids. They can be found in blood cells, lymph nodes, liver, brain, muscles or skin.

Endoparasites are usually protozoans (single cell organisms), such as plasmodia (malaria), trypanosomes (sleeping sickness) or leishmania. Multicellular endoparasites can be worms (helminths), such as adult round worms or larvae (microfilaria). Microfilaria can easily spread to lymph vessels (elephantiasis) or to the eye (River Blindness, also known as onchocerciasis). Schistosomes are a type of flatworms that lay eggs in the urinary tract, and give rise to schistosomiasis (also known as snail fever or bilharzia).

Ectoparasites are found on the outside of the body. They are usually blood sucking arthropods, which include both insects (mosquitoes, sand mosquitoes, fleas and lice) and arachnids (ticks and mites).

Arthropods can transfer disease from one host animal to another by stinging or biting. This type of infection is termed «vector borne» (vector = to carry along).

Protozoans and microfilaria are transferred by vectors.

Yellow fever is a viral disease transmitted by mosquitoes, whereas the bacterial disease Borelliosis is transmitted by ticks.

Humans are infected directly with Schistosomiasis when the free-swimming larvae penetrate the skin of people who are swimming or bathing.

There are also several bacterial and parasitic diseases that are transmitted by direct contact or through water or food; hepatitis, haemorrhagic fever, typhoid fever, amoeba dysentery (shigellosis) and cholera.

The spectre of diseases is large and complex, but important!

Prophylaxis, and avoiding infection, are often the only means of protection. There are no vaccines against parasitic diseases, and there is often no treatment. Unfortunately, when a parasite has entered the body, it stays there for life, even if the symptoms may disappear for shorter or longer periods of time.

This booklet is meant to prepare you for what may come, BEFORE it is too late. There are more diseases than those included in the book, but we have included this selection because we feel it is the most relevant for postings in the pertinent regions.

Read the book thoroughly, think prevention, and enjoy your trip!

Fredrik S. Thorn

Harald Wiik
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PARASITIC DISEASES
1.1. MALARIA

CAUSE:
The disease is caused by a microscopic single cell parasite (protozoan) named *Plasmodium*. There are four species that give rise to disease in humans: *P. falciparum*, *P. vivax*, *P. ovale* and *P. malariae*; the two former being the most common. In addition, a recently discovered species, *P. knowlesi*, has been found in apes in Southeast Asia. This parasite must evolve through a stage in certain types of mosquitoes (*Anopheles*), before it can infect humans, which are considered to be the main host for this plasmodium.

PARASITE LIFE CYCLE:
The parasite has a simple life cycle. Humans are infected by mosquitoes carrying the parasite. The mosquito spits an anticoagulant into the bite site before biting, and the parasite enters with the mosquito saliva. In humans, the parasite will initially reproduce in the liver, thereafter in the red blood cells. The red blood cells will be destroyed (“bursting”), as the parasite reproduces itself. This phenomenon is the main cause of the disease symptoms.
Subsequently, new red blood cells will be affected. When people who have the disease in the relevant stages are bitten by blood sucking mosquitoes, the mosquito will absorb the parasite. The parasite will then go through a new cycle of development and reproduction in the mosquito. After 10-18 days, the mosquito will contain infective stages of the parasite, and humans can again be infected when these mosquitoes bite and suck blood.

**GEOGRAPHIC DISTRIBUTION:**
Malaria is the infectious disease which causes the most fatalities in the poorer regions of the world. On average, one person dies of malaria every 30 seconds, i.e. 1 million deaths per year. Malaria exists wherever the climate (temperature, precipitation, and altitude) are conducive to the anopheles mosquitoes, and where the temperature is warm enough for the parasite to reproduce inside the mosquito. This requires temperatures above 15-20 °C. All tropical and sub-tropical regions satisfy these conditions. However, there are seasonal variations that influence climate. The disease has been eradicated in many areas by destroying the habitat of the mosquitoes (draining of marshlands). In general, the prevalence can be said to increase, and to be less seasonal, closer to the equator.

In central, sub-Saharan Africa, malaria presence must be expected everywhere and in all seasons, even though there may be local variations. The different plasmodium species have varying geographic distribution, and cause disease of varying severity.

**MALARIA RISK**

<table>
<thead>
<tr>
<th>Countries without risk of malaria</th>
<th>Countries with risk of malaria</th>
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</table>

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8 Tropical medicine - Norwegian Armed Forces Joint Medical Services
SYMPTOMS AND COURSE OF DISEASE:
The symptoms and disease courses of malaria vary from mild symptoms or lack of symptoms, to serious illness which may be fatal. This depends on the type of plasmodium involved. *P. falciparum* is considered to be the most dangerous. For non-immune visitors to Central Africa, the risk of catching falciparum malaria is up to 30%. Even with appropriate treatment following the return to Europe, the average mortality is 0,5-2%. Health care personnel often neglect to ask febrile patients if they have been to malaria regions during the last year.

The disease courses can be divided into three types:

- **Uncomplicated malaria**: The incubation period varies from 1 to 4 weeks, but in some cases can be up to 1 year. The symptoms are similar to other types of general infections: fever, perspiration, chills, headache and general malaise. The fact that the patient is, or has recently been, in a region where malaria occurs, is an important indication. The symptoms appear in bouts lasting a few hours, and they reappear after 3-4 days. However, this is not a consistent rule; the fever attacks may last considerably longer. If *P. falciparum* is the cause, the patient may also develop jaundice.

- **Severe malaria** occurs mainly when *P. falciparum* is the cause of the disease. Damage to internal organs such as liver and kidneys may occur; these may in turn cause serious symptoms such as anemia, blood in the urine, and pulmonary edema. Other symptoms may originate in the central nervous system (unconsciousness and convulsions). Without timely medical treatment, severe malaria may be fatal.

- **Recurrent malaria**: Two plasmodium species, *P. vivax* and *P. ovale*, may develop dormant stages in the patient’s liver. These dormant parasites may «reawaken» and cause new disease episodes several months or years after the original disease outbreak.

DIAGNOSIS:
Accurate diagnosis can only be achieved by identification of the parasite in blood through microscopy. There are stix (Parasight® eller Malaquick®) for quick diagnosis, but these may result in false negatives and should not be relied upon.
PREVENTION AND TREATMENT:
There are two types of prophylactic measures: exposure prevention and chemoprophylaxis. Local knowledge about the area you are in, which plasmodium species to expect, and their resistance to medicines, is absolutely necessary in order to achieve effective medical prophylaxis. Effective prophylaxis also requires very careful adherence to the dosing regimen for each drug. Missing the prescribed dose just a single time may be enough to allow infection to occur. Since medical prophylaxis does not provide 100% protection, it is equally important to avoid mosquito bites. The mosquito is usually active at night; i.e. it seldom bites during daylight, but it will bite in a dark room during daytime!

■ A vaccine for children has been developed in 2016. Just how effective the vaccine will be is currently unclear, but this is good news in the work to reduce the spread of malaria, and to reduce the mortality.

■ Exposure prophylaxis means preventing the Anopheles-mosquito from biting. This involves the use of mosquito oil (DEET – diethyl methyl toluamide - based), use of protective clothing from sunrise to sunset, use of mosquito nets over sleeping places, and covering windows and doors with adequate screens. Clothing (uniformand mosquito net) can be treated with Permetrin®.

■ Chemoprophylaxis is prophylaxis through medication.

Malarone® (combination of Atovaquone 250 mg and Proguanil 100 mg) is the newest drug against malaria. It is effective against chloroquine- and multiresistant strains of P. falciparum. Malarone® is active against live parasites and must be used for short term treatment before and after visits to risk areas. Adults take 1 tablet daily, starting 1-2 days before arrival, and continuing until 7 days after departure. In case of vomiting within one hour of medication, a new dose must be administered. In case of diarrhoea, intestinal absorption may be considerably impaired, therefore other medication should be considered.

Doksycykline (a tetracycline derivative) is more than 90% effective against chloroquine resistant falciparum malaria. Adult dosage is 100 mg daily. Medication should start 1-2 days before exposure, and continue until 4 weeks after departure. Doxycycline is generally well-tolerated for periods over several months to a year or longer. However, as with other anti-malaria prophylaxes, doxycycline may cause photosensitisation (1-2%). The risk may be reduced by wearing a hat and using a high factor sunscreen. Doxycycline should be taken together with a light meal and plentiful liquid to facilitate absorption.

Lariam® (mefloquine) is recommended for both long and short term stays. Adult dosage is 250 mg once a week. Medication should start at least 1 week before exposure, and continue until 4 weeks after departure. Lariam® is generally well-
tolerated. For observation of adverse reactions to the treatment should start 2-3 weeks before entry into malaria regions. Chloroquine/proguanil may be used in combination, since 3% of the population are unable to transform proguanil to its active form (cycloguanil). In Africa, *P. falciparum* has usually developed immunity toward chloroquine, therefore this prophylactic treatment is no longer recommended.

**Chloroquine® (Chloroquine phosphate)**
500 mg (2 tablets) once weekly, from 1 week before arrival to 4 weeks after departure from the risk area.

Proguanil (Paludrine®) 200 mg (2 tablets à 100 mg) once daily, From the day before arrival to 4 weeks after departure from the risk area.

<table>
<thead>
<tr>
<th>Prophylaxis</th>
<th>Self-treatment</th>
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<tbody>
<tr>
<td>Chloroquine/proguanil</td>
<td>Lariam® <em>(15 mg/kg bw)</em> 1000 mg: 250 mg tablets x 4 as a single dose (WHO)</td>
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<td>Or:</td>
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<td></td>
<td>1000 mg: Initially 750 mg (three 250 mg tablets), followed by 1 tablet 8 hours later</td>
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<td>Four tablets once daily for 3 days.</td>
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<td>Malarone®</td>
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<tr>
<td>Doxycycline</td>
<td>Malarone® Four tablets once daily for 3 days.</td>
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<td>Or:</td>
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<td>Quinine® Two 250 mg tablets, 3 times daily, for 7 days.</td>
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<td>Or:</td>
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<td>Arthemeter® *(Artemesinine) 300 mg as a single dose on the first day, followed by 150 mg daily for 7 days.</td>
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<td>Riamet® Four tablets daily for 6 days.</td>
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<tr>
<td>Lariam®</td>
<td>Malarone® Four tablets once daily for 3 days.</td>
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<td></td>
<td>Lariam® *(25 mg/kg bw) divided as follows: 750 mg (three 250 mg tablets) initially, followed by 2 tablets 6-8 hours later, and one additional tablet after another 6-8 hours.</td>
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<td></td>
<td>Or:</td>
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<td></td>
<td>Quinine* or Arthemeter® may also be used.</td>
</tr>
<tr>
<td>Malarone®</td>
<td>Lariam® <em>(15 mg/kg bw)</em> 1000 mg: 250 mg tablets x 4 as a single dose (WHO)</td>
</tr>
<tr>
<td></td>
<td>Or:</td>
</tr>
<tr>
<td></td>
<td>1000 mg: divided such that the initial dose is three 250 mg tablets, followed by 1 tablet 8 hours later.</td>
</tr>
</tbody>
</table>

*Malaria: shifting from prophylaxis to self-treatment.*
Self-medication: Malaria infection must be suspected whenever a fever episode occurs between one week after arrival, and 2 months after departure from the malaria region. Treatment must start as soon as possible. If diagnostic facilities are not available, self-medication should be initiated based on suspicion of malaria. In such cases a different drug from that used in the prophylactic regime should be chosen. In case of self-medication contact a medical doctor!

Artemisinin (Arthemet® or Artesunat®)
The combination drug (Riamet®), is no longer available in Norway, only in central Europe. Rapid onset and probably with few side effects.

Arthemet® (artemesinin) dosage regimen: 300 mg on day 1, followed by 150 mg daily for 7 days.

Riamet® (CoArtem® in Africa) (artesiminin 20 mg/lumefantrine 120 mg) is highly effective against falciparum malaria. The treatment consists of 6 doses of 4 tablets, the first dose is given at diagnosis, followed by 5 consecutive doses after 8, 24, 36, 48 and 60 hours. An additional dose should be given in case of vomiting within one hour after administration.

Malaria drugs that should be avoided (but which may appear locally in malaria areas):

- Amodiaquine® (chloroquine-like), must neither be used as prophylaxis nor as treatment, because of the risk of fatal agranulocytosis.

- Daraprim®, should only be used as prophylaxis if hemoglobin, leucocyte, and thrombocyte levels can be measured at regular intervals. Megaloblastic and aplastic anemia, as well as granulocytopenia have been observed.

- Fansidar® (pyrimethamine-sulfadoxine), should not be used as prophylaxis, because cases of fatal agranulocytoses have been reported. Fansidar® may still be used for treatment.

- Halfan® (halofantrine), should not be used. Although it is a very effective treatment for malaria, even slight over dosage may result in fatal ventricular arrhythmias.

- Maloprim® (pyrimethamine-dapsone), should not be used as prophylaxis, due to the risk of fatal agranulocytosis.

Correct use of mosquito net is important to stop the mosquito. Photo: FSAN.
1.2. LEISHMANIOSIS

**CAUSE:**
The disease is caused by single cell parasites of the species *Leishmania*. The parasite resides in the cells of humans and animals, mainly in the blood-forming organs and lymphatic tissues. The infection is transmitted between humans or from animals to humans through bites from the sand mosquito *Phlebotomos* (1,5-2,5 mm).

The sand mosquito reproduces in forested areas, in caves, or in the burrows of small rodents. Sand mosquito larvae thrive in areas with nutrient-rich soil or waste. In particular, the larvae need these conditions to develop. Therefore, there may be many larvae in areas where the soil has been dug up, or in waste disposal areas. Adult mosquitoes want blood. The mosquitoes rarely fly higher than 1-2 m above the ground, and they are nearly silent.

There are several species of *Leishmania*; *L. tropica*, *L. major*, *L aethiopica* and more. These have varying geographic distributions and often different animals or humans as hosts. Small rodents are often preferred hosts.
PARASITE LIFE CYCLE:
The life cycle is simple. The infection is transmitted by sand mosquitoes. Within the mosquito, the parasite goes through a differentiation process which can take 2-3 weeks. The next time the sand fly bites a host animal, the infection can be transmitted through the bite.

GEOGRAPHIC DISTRIBUTION:
The disease is spread throughout large parts of the world in tropical and sub-tropical areas. Sometimes more temperate areas, such as southern Europe around the Mediterranean Sea, have Leishmaniosis. The limits for the spread of Leishmaniosis have been established at around 45°N and 32°S. The disease is endemic in 88 countries, which puts 350 million people at risk. The main areas are dry savanna and semi-arid areas near the equator. Sudan, Iraq, Afghanistan and Brazil are the areas with the highest infection rates. Globally, between 1.5 and 2 million new cases are reported annually.
SYMPTOMS AND COURSE OF DISEASE:
There are two different forms of the disease:

- **Cutaneous leishmaniosis** results in rashes and skin lesions of varying severity.
- **Visceral leishmaniosis** involves internal organs, mainly the blood-forming organs and lymphatic tissues. This is a serious disease; it may be fatal if left untreated.

**Cutaneous leishmaniosis:**
The insect bite itself often goes unnoticed, but it can subsequently itch very badly. A small hard lump can last a few days, followed by a silent incubation period, which may last from one week to several months after the bite. Gradually an itchy swelling develops, increasing in size with an inflammatory reaction around the edges (see photo).

The cutaneous type can be sub-divided into three groups:
- The most common type heals without any intervention. From 1-200 simple ulcerations appear on the face, arms and legs. Spontaneous healing takes place within a few months, but scars remain. The lesions have a characteristic shape, with raised edges and ulcerations in the centre. The condition is not serious, and usually the patient’s general health is not affected.
- The muco-cutaneous type is more serious. This type affects the cutaneous membranes of the upper respiratory system, resulting in damage to the soft tissues in the nose, mouth, and throat. This may lead to considerable facial disfiguration.
- The diffuse type is a widespread, chronic skin disease which may resemble multibacillary leprosy. This type never heals spontaneously, and often recurs after treatment.

**Visceral leishmaniosis:**
This is the most serious type of leishmaniosis. The incubation period is often long, commonly from 2-6 months, but varying widely from 10 days to 10 years. Left untreated the disease will usually be fatal within 2 years. The most severe damage occurs in the liver, spleen, and bone marrow. Early symptoms are often non-specific, including fever, anaemia, lethargy, and weight loss. Later, skin pigmentation can appear (in combination with anaemia) on the hands, feet, face, and abdomen. This has given rise to the Hindi name “kala azar” (black fever). In later stages emaciation with severely distended abdomen due to hepatosplenomegaly may occur.
**DIAGNOSIS:**
Any skin lesions that do not heal within 3-4 weeks in patients who have been in tropical or sub-tropical regions, including areas around the Mediterranean, may be due to cutaneous leishmaniosis. The diagnosis may be verified by histologic examination of a biopsy from the outer border of the lesion, using Giemsa staining. Visceral leishmaniosis must be verified by serology at specialised laboratories.

**TREATMENT:**
- **Pentostam**® (sodium-stibogluconate), i.v. injection 5-10 mg pr. kg body weight, morning and evening for 10-20 days, depending on the type of parasite.
- All medications currently available for the treatment of leishmaniosis have serious side effects, e.g. pancreatitis and cardiovascular complications. Correct dosing is therefore of utmost importance.
- Cutaneous leishmaniosis must always be treated, and referred to a Centre for Tropical Medicine, for further assessment.
- Visceral leishmaniosis must always be treated.

**PROPHYLAXIS:**
There is no vaccine against leishmaniosis. Prophylactic measures include reduction or elimination of sand mosquitoes, and use of mosquito nets (treated with Permethrin®) at night. Permethrin® treatment of the nets is important because the mosquitoes may pass through the holes in the net. Clothing with long sleeves and trousers should cover as much skin as possible. Use mosquito repellent which contains DEET or other recommended repellent on bare skin.

*Rock badger (rock hyrax). Reservoir for Leishmania in Africa.* Photo: FSAN.
1.3 SLEEPING SICKNESS (AFRICAN TRYPANOSOMIASIS)

**CAUSE:**
Sleeping sickness is caused by a microscopic parasite, a sub-species of the flagellate *Trypanosoma brucei*; either *Trypanosoma brucei gambiense* or *Trypanosoma brucei rhodesiense*. These parasites are transferred through bites from the tse tse fly (*Glossina*) from human to human, or from animal to human. The parasites is found in blood and body fluids.

**PARASITE LIFE CYCLE:**
The parasite has a simple life cycle. The tse tse fly gets infected by biting infected animals or humans. The parasite then undergoes a 3-week long development process within the fly, which subsequently allows them to infect humans or animals when the tse tse fly bites. Humans are the primary reservoir for *T. gambesiense*. Livestock and antelopes are the primary reservoir for *T. rhodesiense*.
SLEEPING SICKNESS IN AFRICA

GEOGRAPHIC DISTRIBUTION:
Sleeping sickness is only found in Africa. The disease occurs between 15° N and 20°S, i.e. south of the Sahara, and as far south as South Africa. It is limited to areas inhabited by the tse tse fly; mainly rural areas. Therefore, there is no risk of infection in cities and urban areas. The areas inhabited by the tse tse fly are well delimited and known to the local people.

The two sub-species have different geographic distributions. T. gambiense is widespread in western Africa, and is the cause of West-African sleeping sickness. This disease develops slowly, over months to a year, as an encephalitis. T. rhodesiense is found in Eastern Africa, and gives rise to East-African sleeping sickness. The spread of the disease is increasing, and poses a risk to hunters and safari tourists in the area. Between 50,000 and 100,000 cases of sleeping sickness are reported every year.

SYMPTOMS AND COURSE OF DISEASE:
The tse tse fly bites in day time, with a painful bite.

T. gambiense has an incubation period of 2-3 weeks. The first symptoms are usually non-specific. Irregular bouts of fever may be the only symptom, with cycles lasting from one to several days, and not responding to anti-malaria medication. Subsequently swollen lymph nodes, headache, muscle pain,
and asthenia may occur. Following this initial phase, a relatively asymptomatic phase lasting from several months to a few years may occur. The second phase is characterised by symptoms related to the trypanosomes migrating into the central nervous system. This gives rise to chronic meningoencephalitis with increasing apathy and other neurologic symptoms, which end in coma and death.

**T. rhodesiense:** A severe local swelling appears within a couple of days following infection, and a chronic lesion (chancre) develops at the site of the bite.

The first symptoms appear from 1-4 weeks after the bite. These include fever, muscle and joint pain, and feeling unwell. The classical symptoms of fatigue, inability to concentrate, apathy, and later convulsions appear when the parasite has reached the brain. The East African type has a more rapid course than the West African variety. When the disease has reached the brain, it is difficult to treat and usually fatal. Death may also occur within the first weeks due to myocarditis.

**DIAGNOSIS:**
*T. gambiense:* Suspected cases should undergo serologic testing (CATT; card-agglutination-test for trypanosomiasis). When a reaction is observed, further parasitological testing of blood and/or lymph should be performed. In cases with positive findings, CSF must be examined to assess which stage the disease is in.

*T. rhodesiense* can usually be found by microscopy of blood smears.

**TREATMENT:**
There are drugs that can cure the disease. Early stages of the disease are easier to treat. The treatment requires hospitalisation and specialist treatment, due to drug administration, observation for side effects, and different treatment regimes related to the stage of the disease. Without treatment, the disease is fatal.

**PREVENTION:**
There are no drugs that can prevent the disease.

Avoid insect bites!
The tse tse fly is about the size of a bee, easy to see and hear.

The best way to avoid bites is to avoid areas with tse tse flies. The flies are attracted to motion and dark colours that give a contrast to the environment. Use light coloured clothing, or clothing that blends well with the surroundings. The fly can bite through thin textiles. Medium thickness clothing (treated with Permethrin®) with long sleeves and mosquito net in front of the face, are recommended in tse tse fly regions. Military uniforms are considered to have adequate thickness. Mosquito repellent has no guaranteed effect.
FILARIASIS

Filariasis is caused by round worms that reside in subcutaneous and lymphatic tissues. There are eight types that can infect humans. Of these, there are three types that cause most of the disease cases. *Wuchereria bancrofti* and *Brugia malayi* cause lymphatic filariasis (elephantiasis) and *Onchocerca volvulus* causes Onchocerciasis (river blindness).

1.4. ELEPHANTIASIS (LYMPHATIC FILARIASIS)

**CAUSE:**
The disease is caused by parasites; the round worms *W. bancrofti* and *B. malayi*. Humans are the main host for these parasites, which are transmitted from person to person by mosquito bites. Many mosquito species can transmit the parasites. Culex or Anopheles mosquitoes are both nocturnally active, *Aedes* mosquitoes bite in the early morning and late afternoon, while the Mansoni mosquito bites both day and night.

**PARACITE LIFE CYCLE:**
The parasite has a relatively simple life cycle. The adult stages end up in the lymphatic system of humans, where they can reside for years. They produce larvae which eventually find their way into the general circulation. When the mosquito sucks blood, the larvae are transmitted to the mosquito, where they develop during a period of 1-3 weeks. When the mosquito bites a human again, infective larvae are transmitted. These larvae then develop into adult worms in the human lymphatic system. The whole maturation process can take up to 18 months.

**GEOGRAPHIC DISTRIBUTION:**
The disease is widespread in most tropical regions. About 120 million people are affected by the disease, and 40 million are crippled by it. One third of the infected persons live in sub-Saharan Africa, one third in India, and most of the remainder live in South Asia, the Pacifics, and South America. The disease can be found in 80 countries, and about 1 billion people live in risk areas. The highest prevalence is in poor and rapidly growing communities with poor sanitary conditions and environments conducive to mosquitoes.

*Wuchereria bancrofti* mikrofilarie. Photo: © CDC.
SYMPTOMS AND COURSE OF DISEASE: The disease follows a chronic course, where mainly adult humans exhibit clinical symptoms. Men are affected more often than women. The symptoms appear as edema, particularly affecting the legs. Arms, scrotum, penis, vulva and breasts are less often involved. The symptoms are caused by blocked lymph drainage due to the presence of the parasite in lymph vessels and lymph nodes.

DIAGNOSIS:

Clinical examination: Sudden fever with acute abdominal pain and swollen, sore lymph nodes and edema of the lower extremities should lead to suspicion of filariasis.

Microscopic examination: The microfilaria W. bancrofti and B. malayi can be identified through microscopy of blood smears. Antigen testing can be useful because the number of microfilaria in the blood may be low or variable.

Biogenetic diagnosis (PCR – polymerase chain reaction) is available for both species.

TREATMENT:
The disease is relatively easy to treat in early stages. The correct diagnosis must be ascertained before treatment, and infection with Onchocerca (River Blindness) or Loa Loa must be ruled out, because these can lead to serious complications if Diethylcarbamazine (DEC) treatment is used.
**Parasitic Diseases**

*Diethylcarbamazine (DEC)* kills adult worms and impairs microfilaria, so that they are removed from the host’s immune system. Recommended DEC dosage is 6 mg/kg body weight/day in 3 doses following meals for 12 days. The treatment can be repeated every 6 months as long as microfilaria can be detected, or the patient has symptoms.

**Prevention:**
In some regions, the disease can be prevented by chemoprophylaxis using DEC for the whole population. This may be used in regions with high disease prevalence. WHO uses drug treatment and prophylaxis in its programmes to eradicate the disease. Control of vectors is also an important factor in reducing the spread of the disease.

**Practical Measures to Avoid the Disease:**
Protection against mosquito bites, day and night, use of mosquito repellents, covering up skin, and the use of mosquito nets at night are the simplest protective measures.
1.5. RIVER BLINDNESS (FILARIASIS, ONCHOCERCIASIS)

**CAUSE:**
The disease is caused by a round worm (*Onchocerca volvulus*) which resides in the connective tissues, particularly sub-cutaneously in humans. In the larval stage, the parasite can wander to the eye and cause reactions that result in blindness. This has given the disease the name "River Blindness". The adult stage of the parasite is relatively large; a few centimetres in length, but the larvae (microfilariae) are microscopic. The parasite is transmitted from human to human as microfilariae through the bite of a type of black mosquito (*Simulium*), which functions as an intermediary host for the parasite.

**PARASITE LIFE CYCLE:**
The life cycle is simple. Adult worms lay eggs under the skin in humans. The eggs develop into microfilariae, which then migrate within the host's body. The black mosquito acquires the infection when it bites and sucks blood from the human host. Following a development period in the mosquito, the microfilariae may again be transmitted to humans when the infected mosquito bites.

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**FILARIASIS**

**Stages in the mosquito**

1. The mosquito (genus *simulium*), bites and sucks blood. Larvae in the L3 stage enters the bite wound
2. Larvae in the subcutaneous tissues
3. Adult stages in subcutaneous nodules
4. Adult stages only produce microfilaria, which are typically found in the skin, connective tissue, and lymph vessels, but sometimes also in the blood, urine, and saliva
5. The mosquito bites and sucks blood containing microfilaris
6. Microfilarias penetrate the intestines of the mosquito, and wander to the chest muscles
7. L1 Larva
8. L3 Larva
9. Migration to the mosquitoes head and mouth parts

**Stages in the humans**

© CDC.
PARASITIC DISEASES

GEOGRAPHIC DISTRIBUTION:
The disease is mainly spread in Sub-Saharan Africa, throughout the regions on both sides of the equator. It can also be found in South America. *Onchocerca volvulus* and the simulium mosquito, which transmits River Blindness, reside mainly in the damp areas along rivers and waterways, hence the name of the disease. Fifteen to twenty million people suffer from the disease, 500,000 have lost their eyesight.

River Blindness has a chronic, non-fatal course. The incubation period is long. The first symptoms appear one year or longer after the bite from an infected mosquito.

Visible lumps appear in the skin where the adult worms are located. They can also be in connective tissue, in musculature, or close to joints, which can be painful. The larvae will

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**Countries with River Blindness in Africa**

[Map showing African countries with River Blindness]
migrate to the skin surface, where they give rise to skin irritation and lesions. In some cases, the larvae will migrate to the connective tissues of the eyes. The resulting reaction in the connective tissue may lead to blindness.

The disease is mainly contracted by people who live for longer periods (months) in areas where the disease is found. Shorter stays are associated with lower risk of infection.

**DIAGNOSIS:**

**Clinical:** Skin and eye reactions, with or without subcutaneous lumps, in persons who live in, or have visited, the affected regions, may be signs of River Blindness.

Also, severe itching, if other disease such as scabies, reactions to insect bites, or contact dermatitis, have been excluded.

**Ultrasound** is an important diagnostic tool in the search for onchocercid nodules, particularly deep, non-palpable nodules.

**Mazzoti’s test:** involves giving a low oral dose of DEC, preferably 50 mg for adults. After 20 minutes to 24 hours, intense itching will occur, due to a reaction to dead microfilariae in the skin. This test may cause serious side effects, and should therefore only be used when skin biopsy is negative but the diagnosis is still suspected.
**Parasite identification**: Histologic identification of microfilariae in a biopsy specimen void of blood.

**Immunologic diagnosis**: There are several different immunoassays that may be used.

**TREATMENT**:  
*Ivermectin (Mectizan®)* given as a single, oral dose; 150 μg/kg body weight. Eighty percent of the microfilariae in the skin will be eliminated within 48 hours. Re-treatment may be given 6-12 months later.

*Diethylcarbamazine (DEC)* is no longer recommended because of the side effects previously mentioned under Mazzoti’s test.

WHO has implemented a comprehensive eradication programme which includes among others, drug treatment (one dose annually over long periods). Subcutaneous nodules with parasites should be removed.

**DRUGS TO BE AVOIDED**:  
*Suramin*: This drug is no longer recommended due to fatal toxic effects.

**PREVENTION**:  
The main focus in prevention of River Blindness is to reduce or eliminate the occurrence of mosquitos which carry the disease, and to prevent bites. In areas with high risk of infection, insecticides may be added to the waterways where the mosquitos lay their eggs.

**PRACTICAL MEASURES TO AVOID THE DISEASE**:  
The risk of mosquito bites can be reduced by wearing long trousers and long sleeves. The mosquito is deterred by repellents, and these should be used in risk areas.
1.6. SCHISTOSOMIASIS (BILHARZIA)

**CAUSE:**
The disease is caused by a parasite (*Schistosoma*), a fluke. There are 5 species that infect humans (*S. mansoni*, *S. japonicum*, *S. haematobium*, *S. mekongi* og *S. intercalatum*). They have varying prevalence and clinical presentations. In their adult stage the flukes reside in venous blood vessels where they produce eggs. Predilection sites in the body vary from species to species.

**PARASITE LIFE CYCLE:**
The fluke has its adult stage in humans, which are the main host. Some animal species may also become infected, thereby spreading the infection. Infected humans and animals excrete the parasite eggs through the urine or faeces, depending on the parasite species. When the eggs reach fresh water, they are absorbed by certain snails and the eggs develop into infective stages. Finally, thousands of the infectious stage, called cercaria, are excreted into the water. Cercaria are less than 1 mm in diameter and cannot be seen with the naked eye.

Humans are infected in fresh water by free swimming cercarias. The cercarias penetrate the skin or mucosal membranes of people who are bathing or washing themselves. The
Cercarias then migrate to the portal circulation. Within the liver, they develop into adult flukes. These migrate on to venous plexi, mainly around the gut or bladder. All of this happens within a few weeks. The adult flukes will then lay eggs which eventually will be excreted through urine or faeces. When these eggs reach fresh water, the cycle starts again.

**SYMPTOMS AND COURSE OF DISEASE:** Schistosomiasis is both an acute and a chronic disease. The time from infection to the appearance of symptoms may be 2-3 weeks, but may also be much longer. In early stages the symptoms are non-specific, and can include fever, abdominal pain, blood in the urine, reduced appetite, nausea and weight loss. In some cases, the symptoms are so mild that they are not discovered. These persons become chronic infection carriers.

In severe cases, the parasite may affect the cardiovascular system, lungs, the central nervous system (CNS), kidneys, etc., depending on where the parasite is located. Without effective treatment, infected people will become chronically ill. The disease is normally not considered to be fatal.

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**SCHISTOSOMIASIS/BILHARZIA**

1. Infectious stage
2. The eggs hatch and release miracidia larvae
3. Miracidia larvae enter into the snail body tissue
4. Several generations of sprocyists develop in the snail
5. The free-swimming cercaria stage is released into the water
6. During skin penetration, the cercarias lose their tails and become schistosomules
7. Schistosomules circulate
8. They migrate to the portal vein of the liver and develop into the adult stage
9. The adult male and female intertwine and migrate to the veins of the large intestine and liver
10. The eggs circulate to the liver and sometimes the bladder veins

©CDC.
Clinical manifestations:
Swimmers itch: Itchy dermatitis (similar to mosquito bite) which occurs from minutes up to 24 hours following exposure to the parasite. Every year bird schistosomes (these die shortly after penetrating human skin and are harmless) get news coverage related to people swimming in Norwegian lakes.

Acute schistosomiasis (Katayama Fever): This is a reaction mediated by immune complexes. It can start 4-80 days (usually 3-7 weeks) following exposure. The most common source is S. japonicum, but it can also occur with S. mansoni, particularly in non-immune visitors who are infected for the first time. Symptoms include acute onset of fever, chills, nausea, headache, and diarrhoea. In blood stains eosinophilia is often observed.

Chronic schistosomiasis: This condition involves chronic granulomatose inflammation with fibrosis, as a reaction to the schistosome larvae penetrating through tissues. The condition is often asymptomatic, but can also involve fatigue, fever, abdominal pain, and diarrhoea.

The following organs may be involved, leading to organ specific symptoms:

- **Chronic Pulmonary Disease:** Embolising eggs give rise to arteritis and secondary pulmonary hypertension, eventually resulting in heart failure.

- **Hepatosplenic schistosomiasis:** Embolising eggs give rise to periportal fibrosis and portal hypertension with hepatosplenomegali.

- **Intestinal schistosomiasis:** Eggs reach the gut and cause inflammatory changes, hemorrhagic diarrhoea, appearance of pseudo-polyps and findings of ulcerating colitis. Eggs can be detected in mucous membranes and faeces.

- **Vesicular schistosomiasis:** By S. haematobium infection, eggs may penetrate the bladder wall, leading to hematuria, fibrosis, and hydronephrosis. This can lead to the development of carcinoma of the bladder.

- **CNS involvement:** In rare cases eggs can embolise in the brain, causing local inflammation and focal epilepsy. One of the most feared complications is caused by eggs embolising in the spinal medulla, with subsequent paraplegia.

GEOGRAPHIC DISTRIBUTION:
The disease is widespread in tropical regions all over the world, and to a lesser extent in sub-tropical regions. Sub-Saharan Africa is a central region, where some 200 million people in 74 countries have the disease, and about 20,000 related deaths per year. Schistosomiasis is the second most serious parasitic disease, next to malaria. The risk of infection is highest in the areas with non-existent or poor sanitary conditions. The risk of infection is independent of seasons.
**DIAGNOSIS:**
Eggs can be found in urine or faeces, and in biopsies from intestines or bladder. Serologic examination has high sensitivity as a screening tool in visitors with limited exposure, but specificity is limited due to cross reactions with non-human schistosomes (e.g. bird schistosomes).

**TREATMENT:**
*Praziquantel (Biltrizide®)* 600 mg tablets
40 mg/kg body weight as a single dose, or in divided doses at 4-6 hour intervals. Dosage for children and adults is the same. The efficacy is about 70%, therefore the treatment should be repeated after one month.

**PREVENTION:**
Reduction in the spread of eggs from human faeces to water containing host snails is key in all efforts to stop the spread of this disease. Eradication of snails is also being attempted.

All contact with fresh water (bathing, wading, washing) in affected regions must be avoided. Boiling water, or heating it to 50 °C for 5 minutes, kills the cercarias. Disinfection with chlorine has little or no effect on drinking water. Chlorination of swimming pools works. The cercarias die after 2-3 days, therefore storage of fresh water for more than 48 hours, without pollution by snails, is considered to provide sufficient safety from schistosome infection.

The use of mosquito repellents containing DEET has been shown to have a protective effect on exposed areas. When exposure occurs, rapid, strong drying is recommended.
1.7. DRACUNCULIASIS ("GUINEA WORM")

CAUSE:
The disease is caused by a round worm (*Dracunculus medinensis*), which has its adult stage in the subcutaneous connective tissues in humans. Of all known parasites in humans, this is the largest. It can reach a length of 80 cm, with a diameter of 1.5-2.0 mm.

PARASITE LIFE CYCLE:
The round worm has its adult stage in humans, the main host for this parasite. Humans get infected by drinking surface water from sources such as ponds, small lakes, and shallow wells. The water may contain water fleas (Cyclops, 1-2 mm of size) which act as an intermediate host for the parasite. When the fleas are ingested and arrive in the stomach of humans, the parasite larvae are released. The larvae migrate through the intestinal wall and into body tissues. Three months later adult stages will mate. The pregnant females will migrate through the body to the subcutaneous connective tissue, arriving 8-10 months later in the lower extremities of their host. They will

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**DRACUNCULIASIS (GUINEA WORM)**

1. Human drink unfiltered water containing fleas with L3 infection
2. When the water flea dies, the larvae are released. The larvae penetrate the stomach/intestinal wall of their host, where they develop and reproduce
3. Fertilised female larvae will migrate toward the skin surface to lay eggs/larvae that can leave host through a lesion in the skin. One year after the infection, the female larva emerge through the skin
4. The female worm releases L1-larva into the water
5. L-1 larvae are eaten by a water flea
6. The larvae go through two stages within the water flea before reaching the larval stage 3 (L3)

© CDC.
then break through the skin to lay eggs. This results in a small, chronic lesion on the calf. When the eggs reach water, they will be eaten by water fleas. After a couple of weeks in the water flea, infective larvae will develop. Dracunculiasis is then again transmitted to humans when a person drinks water containing water fleas.

**GEOGRAPHIC DISTRIBUTION:**
Dracunculiasis is found in 13 countries in the sub-Saharan region. Most cases are located in Sudan, Nigeria and Ghana. Following diligent efforts to reduce the occurrence of the disease, the global incidence has been reduced by 97%, from 3.2 million cases in 1986 to fewer than 80,000 cases in 2005. In recent years fewer than 50,000 cases per year have been reported in Sudan, but there is reason to assume that the actual number is higher.

Most cases become infected in the period from May to September.

**Risk of Dracunculiasis**

![Map showing countries with and without risk of Dracunculiasis](image-url)
SYMPTOMS AND COURSE OF DISEASE:
The symptoms are caused by the round worm’s migration in the host’s connective tissues. In the early stages the symptoms are non-specific, and will mainly consist of pain, particularly in the vicinity of joints, when the round worm migrates to the area. When the female worm breaks through the skin to lay eggs, painful lesions appear. The lesions may become infected by bacteria. Typically, the affected persons dip their feet in water to alleviate the pain. Thus, the eggs can be transferred directly to the water, where they are swallowed up by water fleas.

The course of the disease is long and painful. It may also be crippling. In infected patients, there is usually one outbreak per year, with one worm. However, cases with up to 20 worms at once have been observed. The disease is non-fatal.

DIAGNOSIS:
Infected persons cannot be diagnosed before the female worm penetrates the skin. Immediately before this, it is sometimes possible to see and feel the worm under the skin.

Clinical diagnosis:
Examination of the ulceration where the
A female worm has penetrated the skin. Formation of blisters with localised itching, and burning pain.

Microscopic findings of larvae in water contaminated by the female worm. This is achieved by holding the ulcer under water.

**TREATMENT:**

There is no drug treatment that kills the round worm in the body. The only treatment is to slowly and carefully pull the worm out; i.e. to remove the adult round worm after it breaks through the skin (see photo). The lesion will heal spontaneously once the round worm has been pulled out. If the round worm comes apart it will recoil into the host tissue, resulting in a severe inflammatory reaction and subsequent scarring.

**PREVENTION:**

Infection can easily be avoided. The only source of infection is untreated drinking water. Simple filtration is enough to avoid infection.

In principle, the disease is easy to control and eradicate. WHO has conducted a successful programme to eradicate the disease. Filtration of drinking water is the most important step. However, in southern Sudan this programme has been stopped because of the civil war, and the number of reported cases has been rising rapidly since the year 2000.

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The guinea worm is the source of the symbol used to depict medical science and the Norwegian Medical Association. This has in turn inspired the design of FSAN’s emblem.
In the present context, the term "vector" refers mainly to insects and other arthropods which transmit infectious material from one individual to another, usually through bites. In central Africa, a large number of dangerous, infectious diseases are transmitted by vectors. Some of the most important ones (malaria, leishmaniosis, and sleeping sickness) are parasitic diseases that have been described previously. In this chapter, the viral diseases yellow fever, dengue fever, and chikungunya fever will be discussed.
2.1. YELLOW FEVER

CAUSE:
The disease is caused by a flavi virus. The virus can survive at 4°C for one month.

ROUTE OF INFECTION:
The mosquito species *Aedes aegypti* transmits the disease from humans to humans. Infection from apes are transmitted via bites from either *Aedes* or *Haemagogus* mosquitoes to humans. The mosquitoes often bite around sunrise and sunset, but they may also bite during the day.

The mosquito becomes infected following bites on infected people who are in their first to third febrile day. The virus then undergoes a development process within the mosquito, which lasts from 4 days at 37°C to 18 days at 18°C.

The mosquito remains infected for the rest of its life, which is about 2-4 months long. Transmission from one generation to the next (transovarian transmission) seems to be an important factor that enables the virus to survive the arid season.
**GEOGRAPHIC DISTRIBUTION:**
Yellow Fever is found in tropical regions in central Africa, and in the rain forests, and in urban areas of South America. In Africa, the disease is most common in the western regions. Yellow Fever often occurs in epidemic outbreaks. This is because the risk of infection increases when the virus reservoir increases. WHO estimates annual rates of yellow fever at 200,000 cases, and 30,000 deaths from the disease.

**SYMPTOMS AND COURSE OF DISEASE:**
The incubation period is short (3-6 days). Initial symptoms include sudden fever, chills, headache, muscle pain, nausea, and vomiting. This phase lasts up to 6 days, and this is the period when the patient may infect the vector mosquito, because the virus is present in the blood. Following a 24-hour period, 5-20% will develop severe symptoms such as jaundice (which has given the disease its name), renal insufficiency, and increased bleeding tendency. During epidemics in endemic regions, the disease is generally mild, and mortality lies around 5-10%. Mortality can be considerably higher though, and in southwest Ethiopia, along
the Omo River, the mortality rose to 85% during the epidemics of 1960-62.

Patients who survive a serious episode experience long convalescence periods, usually without sequelae.

**DIAGNOSIS:**
Virus can be isolated during the first 3 days after the outbreak of the disease. After one week antibodies (IgG) can identified.

**TREATMENT:**
Viral diseases are, for all practical purposes, untreatable. There are inadequate and ineffective treatments against a few viral diseases, but none that help against yellow fever.

It is important to be aware of the risk of dehydration; patients must receive enough fluids. It is not necessary to isolate patients, but contact with mosquitoes should be avoided, to prevent secondary spreading.

**VACCINE:**
There is an effective vaccine against yellow fever. The vaccine provides excellent protection, and is included in the vaccination regimen for all foreign operations.

Refugee Camp in East Congo. High risk of disease spread. Photo: FSAN.
2.2. DENGUE FEVER

CAUSE:
Denguefeber is caused by an arbovirus (dengue virus) which is transmitted by an Aedes mosquito, mainly *A. aegypti*. There are 4 different types of Dengue Fever virus (DEN-1 to DEN-4).

INFECTION ROUTE:
The infection is transmitted by bites from mosquitoes which have previously bitten an infected person. *A. aegypti* prefers to live close to humans. It hatches in small water puddles, for example in tyres or other places. The mosquito often bites around sunrise and sunset, but it may also be active during the day. The virus is also transmitted from adult mosquitoes to the next generation through the mosquito ovaries. Infected mosquitoes are considered to carry the infection throughout their life.

GEOGRAPHIC DISTRIBUTION:
Dengue Fever originated in South-eastern Asia. Until about 1970 Dengue Fever was limited to this part of the world. During the past 20 years the disease has spread to most tropical and partially to sub-tropical regions, but it is still most common in South-eastern Asia. The disease is mainly found in cities and urban areas, because the habitat for

*Aedes aegypti* mosquito. Photo: © CDC.
A. aegypti is enhanced in densely populated areas with many small water puddles. The disease has spread rapidly to other more tropical regions in Latin and South America. Currently, an estimated 50 million new cases occur annually on a global basis. About 500,000 of these require hospitalisation.

**SYMPTOMS AND COURSE OF DISEASE:**
The disease is mainly found in older children and adults. When younger children are infected they generally exhibit few, mild symptoms, which may often not be noticed. The incubation period is short, 2-7 days. The symptoms are non-specific, including rapid onset of fever which may last a few days. This is accompanied by muscle pain, headache, reduced appetite, and general weakness. Sometimes a rash may occur. Given adequate treatment and care, the expected mortality is 1-2%. However, one variety of Dengue Fever, Dengue Haemorrhagic Fever (DHF), is much more serious, with mortality rates up to 20%. It is currently unknown why some patients develop this disease course. Among naïve patients 0.2% develop DHF, whereas among those who have previously had Dengue Fever, 2% develop DHF. Mortality increases when previously infected individuals become infected with a different DEN type.

**DIAGNOSIS:**
Serologic tests can be performed from the first days after onset of disease symptoms. These tests are offered by larger hospitals in the affected regions.
The virus can be identified in early stages of the disease, but this is seldom carried out.

**TREATMENT:**
As for other viral diseases, there is no specific treatment. Symptomatic treatment and good patient care are recommended.

**PREVENTION/VACCINE:**
A vaccine against Dengue Fever is currently in development, with promising results.

The most important prophylactic measure is to avoid mosquito bites. The difficulty with Aedes mosquitoes is that, in contrast to other mosquitoes, they also bite during daytime. Clothing and mosquito repellents are therefore the most important prophylactic measures.

It is imperative to empty water from places where the mosquito may lay its eggs.

**IMPORTANT!**
Avoid the use of acetyl salicylic acid (e.g. Aspirin) in regions with endemic Dengue Fever, because Dengue Fever may cause thrombocytopenia, which in turn results in an increased bleeding tendency in case of Dengue Fever infection.
Some mosquitoes can pass through mosquito nets. Photo: © CDC.

High risk area for vector borne diseases. Photo: © FSAN.
The name means «bent hands / joints» in Kiswahili (a Tanzanian language), because patients assume contorted poses due to the severe joint pain caused by the disease.

**CAUSE:**
Chikungunya fever is caused by an alpha virus.

**ROUTE OF INFECTION:**
Chikungunya is mainly transmitted by mosquitoes of the \textit{A. aegypti} species, but it can also be transmitted by a large number of other species of \textit{Culex} and \textit{Mansoni Africana}, and Anopheles.
GEOGRAPHIC DISTRIBUTION:
The first observation of Chikungunya Fever was as an epidemic in East Africa (Tanzania) in 1952-53. The disease is widespread in Africa, and is also found in Saudi Arabia, India, and Southeast Asia.

SYMPTOMS AND COURSE OF DISEASE:
The duration of incubation is 1-12 days (average 2-3 days). Classical symptoms start with acute joint and back pain, in addition to muscle pain, high fever, and conjunctivitis. The condition improves after 2-3 days, following which a widespread maculopapillary rash develops. The fever may recur after a remission lasting 1-2 days.

Most patients recover after a few weeks, but 5-10% can have chronic joint trouble with pain, stiffness and swelling that may persist for several years.

Children often have less typical clinical symptoms.

DIAGNOSIS:
The virus can be isolated during the first 3-4 days of the disease. IgM can be measured in the acute phase, and can be detected from several weeks to months later.

The disease is in itself not fatal, but impaired immune responses may lead to fatal outcomes.
TREATMENT:
Symptomatic, as for other viral diseases.

PREVENTION/VACCINE:
Avoid mosquito bites. The same principles as with Dengue Fever apply. Fight environmental factors conducive to mosquito reproduction and growth. There is no vaccine against Chikungunya Fever.
2.4. RIFT VALLEY FEVER (RVF)

Rift Valley Fever was first described in Kenya in the early 1900s, as a disease affecting sheep and humans. In animals the symptoms are diffuse, and mortality among young animals is high. The disease is a vector-borne zoonosis; i.e. it is transmitted from animals to humans. Until 1977 the disease was limited to the regions south of the Sahara. In 1977 RVF spread to Egypt.

CAUSE:
Rift Valley Fever is caused by a phlebovirus.

ROUTE OF INFECTION:
The virus can infect many different animals. The virus is transmitted between hosts by mosquitoes of the species *Eretmapoides, Mansonia, Anopheles, Aedes,* and *Culex.* Other biting insects may also spread the disease by simple physical contact. Infection directly by contact with droplets from animal carcasses may occur.

GEOGRAPHIC DISTRIBUTION:
Rift Valley Fever can be found all over Africa, except the northwestern regions (Morocco, Algeria, and Tunisia). In 2000 an outbreak was recorded on the Arabian peninsula.

SYMPTOMS AND COURSE OF DISEASE:
The incubation period lasts 2-6 days. The disease starts suddenly, with fever, headache, muscle and joint pain, conjunctivitis, and oversensitivity to light. Most patients recover completely after the first acute phase, but in a few cases the symptoms recur. In 5-10% of the cases, kidney disease may develop 1 to 3 weeks after the initial febrile phase. Macular exudates may occur, and in some cases renal bleeding and vasculitis. Half of these patients develop sequelae, such as impaired ability to focus the eyes, and in 5% encephalitis occurs. The encephalitis is rarely fatal. One % of these cases develop hemmorhagic fever, very similar to yellow fever, with a mortality of 10%.

DIAGNOSIS:
Virus can be found in blood cultures, or by PCR, during the first week following the onset of symptoms. Methods for measuring antigen are available.

TREATMENT:
For the uncomplicated cases, symptomatic treatment and observation may suffice. For more serious cases, intensive care may be necessary. Ribavirin has been shown to be efficacious against this virus, and is recommended in cases where haemorrhagic conditions develop.

PREVENTION/VACCINE:
There is a vaccine, and it is recommended for exposed veterinarians and laboratory personnel. The spread of the disease may be prevented by isolating sick animals. The animals must run the full course of the disease; i.e. either die or recover. They must not be slaughtered, because of the danger of contagion from opened carcasses. Control of vectors is recommended.
3 DISEASES THAT ARE TRANSMITTED THROUGH FOOD AND WATER
Most diseases that are transmitted through food or water are caused by pollution from excrements, urine, or carcasses from infected people or animals. Food or water can be directly or indirectly contaminated by faeces or urine containing the infectious agent (faecal – oral route of infection). Diseases that spread in this manner are seldom caused by direct infection between humans, or between animals and humans.

In principle, it is easy to protect against food and water borne diseases, by following certain basic rules of hygiene. These consist in interrupting the «connection» between human and animal faeces on the one hand, and whatever is ingested through the mouth on the other.

In our part of the world, the establishment of adequate sewage systems and sources of drinking water are the most important measures that protect us from large outbreaks of such diseases. In addition, strict regulation of processing, preparation, and distribution of food are important. It is when these safety measures are neglected, that diseases break out.

In other parts of the world where there are inadequate resources to ensure satisfactory hygienic standards, or where there is inadequate understanding of how diseases spread, the food and water borne diseases pose large threats to public health. As foreigners in these areas, we must therefore be aware that most of the food and water may potentially be a source of infection.

The most important safety measures include the following:

- Only drink water that comes from an established supply system.
- If safe water is not available, the water must be boiled or microfiltered before drinking.
- Avoid ice cubes in any drink. Bacteria easily survive being frozen in ice, and you have no control over the source of water for ice cubes.
- Avoid food that has not been boiled, or that is no longer hot.
- Only eat fruit that you have peeled yourself: "if you can’t peel it, don’t eat it".
- Do not eat raw salad.
- Avoid all food and drink from the local population (when practically possible).
- Always bring your own cup.
- Always wash your hands before eating.

It is often up to each person to employ a few simple safety measures in order to avoid infection.

The following pages will provide an overview of the most common food and water borne diseases.
DESCRIPTION:
Cholera is an acute intestinal infection caused by the bacteria *Vibrio cholerae* (serotype 0-1 and 0-139). Other serotypes cause milder disease. The bacteria produce a toxin that affects the intestinal epithelium, so that large amounts of fluid are excreted through the intestines. The disease can be mild, but in many cases, particularly in children, the disease can be life threatening, mainly because of rapid dehydration.

As for *Yersinia pestis* and Yellow Fever, Cholera is a disease with mandated reporting to WHO, and extensive resources are invested to limit the spread of the disease whenever an outbreak is reported. Every year outbreaks are reported, and many thousands of people are affected.

In one outbreak in a refugee camp in Congo in 1994, 50,000 people became ill, and 24,000 died.
DISEASES THAT ARE TRANSMITTED THROUGH FOOD AND WATER

GEOGRAPHIC DISTRIBUTION:
Cholera is mainly found in tropical and underdeveloped regions in the world. The disease may also appear anywhere in the world, since it can be spread by people who excrete the bacteria through their faeces. However, larger outbreaks of the disease occur mainly where the sanitary conditions are inadequate, e.g. in disaster areas following floods, or in refugee camps.

SYMPTOMS AND COURSE OF DISEASE:
Most infected people will not develop severe symptoms. In otherwise healthy individuals, on average 5% will develop serious symptoms following infection. The incubation period is short, often less than 24 hours, but usually 2-3 days. Patients get very severe diarrhoea, and often also vomiting. Because of dehydration, death may occur within hours, if the patient is left untreated.

DIAGNOSIS:
Violent diarrhoea considered together with the environment should trigger suspicion. The bacteria are excreted in large amounts, and the diagnosis is confirmed by identification of the bacteria in a faecal sample.

TREATMENT:
Rapid intravenous fluid treatment (sugar or electrolyte solutions) is essential. Drinking large amounts of fluids is not enough, but should always be given in addition to intravenous fluid.

VACCINE:
Vaccine is available and is included in the standard INTOPS vaccine package. Unfortunately, it is not as good as one might wish.

Vibrio cholerae bacteria (x 1,000 magnification).
Photo: © WHO.
3.2 TYPHOID FEVER AND PARATYPHOID FEVER

DESCRIPTION:
Typhoid Fever and Paratyphoid Fever are two different infectious diseases, caused by two different bacteria, *Salmonella typhi* and *Salmonella paratyphi*, respectively. Typhoid Fever is a slightly more severe disease, but otherwise the two infections will appear to be fairly similar. They will therefore be presented together.

Particularly Typhoid Fever is a very serious disease which is often fatal if treatment is not initiated immediately. In areas where the disease occurs frequently, most infections will only result in mild symptoms that do not require medical attention. In contrast, people who are naïve to the disease can become very ill. In endemic areas, many people can be infected without showing any signs of disease. They will serve as sources of infection over long periods of time. On a global basis, between 15 and 20 million people are infected every year, and about 600,000 people die. In contrast to other salmonella infections, typhoid and paratyphoid fever only affect humans.

GEOGRAPHIC DISTRIBUTION:
The disease is mainly found in underdeveloped tropical countries, and is related to poor or lacking sanitary conditions. Central Africa is a focal area. In our part of the world only a few cases occur, and these are nearly all persons who were infected in areas where the disease is endemic.

SYMPTOMS AND COURSE OF DISEASE:
The incubation period is relatively long compared to most other food and water borne diseases; on average 7-8 days. The symptoms include high, long lasting fever, headache, dry cough, lack of appetite, and feeling ill. Typhoid fever simply a form of septicaemia. In contrast to other food and water borne infections, diarrhoea is uncommon. On the contrary, constipation is typical. However, the symptoms vary widely, from the symptom free bearer of the disease, to patients in need of acute intensive care. After the disease has run its course, the patient may excrete bacteria for several weeks. This makes it difficult to prevent the spread of the disease.

DIAGNOSIS:
Diagnosis based on clinical findings alone is difficult. In outbreaks where many people become ill at the same time, with high fever, Typhoid Fever must be suspected. A conclusive diagnosis may be reached by identification of the bacteria in blood samples from the acute stage, or by examination of faeces or urine samples in the later disease stages.

TREATMENT:
Antibiotic treatment is, in principle, efficacious. Previously chloramphenicol was used, with excellent results. Today other antibiotics are tried first. In any case, the patient should be hospitalised for intensive care.
VACCINE:
The vaccine is good. It is included in the standard vaccination package for personnel traveling to areas where the disease is common, such as central Africa. The vaccine provides 80% protection, but is no guarantee against infection.

FOLLOW UP:
Persons who have had the disease must be followed up with faeces specimens to check that they no longer are sources of infection. This is particularly important for all personnel involved in handling or preparation of food.

Water quality inspection in the Niger River.
Photo: FSAN.
3.3. OTHER SALMONELLOSES

DESCRIPTION:
Many other types of salmonella bacteria may cause disease. In contrast to the serious diseases Typhoid and Paratyphoid Fever, these are usually much less severe. These infections primarily affect the intestines, and the symptoms are mainly diarrhoea, often with fever. “Salmonelloses” are, in contrast to Typhoid and Paratyphoid Fever, classified as zoonoses, which means that the reservoir of infection is found in animals. The route of infection goes from animals to humans, either by contamination of drinking water with faeces from infected animals, or through consumption of food products from infected animals. However, infected humans may also excrete bacteria, and thereby serve as a source of infection.

GEOGRAPHIC DISTRIBUTION:
Salmonella infections are common all over the world where the general hygienic standards can be described as poor. In our part of the world, in particular in Scandinavia, where animal husbandry is a fairly uncommon occupation, salmonella infections are relatively rare. In Norway there are about 1,000 cases per year.
SYMPTOMS AND COURSE OF DISEASE:
The incubation period is usually 1-2 days. Symptoms will vary according to the type of bacteria involved. The symptoms develop rapidly. They will mainly include severe diarrhoea with loss of fluids and dehydration, abdominal pain, and often mild fever, which may last for a few days. In rare cases, the bacteria may spread from the intestines and cause severe sepsis, or they can move to various other tissues, causing other afflictions such as joint pain, or abscesses.

DIAGNOSIS:
Accurate diagnosis can only be confirmed by detection of salmonella bacteria in the faeces.

TREATMENT:
Drinking large amounts of fluid is important to prevent fluid loss due to diarrhoea. Uncomplicated salmonellosis is generally not treated with antibiotics.

VACCINE:
There is no vaccine against common salmonellosis.

FOLLOW UP:
Persons who have been ill must be followed up with 3 negative faeces specimens in order to ensure that they are no longer sources of infection.
3.4. DYSENTERY (BACILLARY DYSENTERY – SHIGELLOSIS)

DESCRIPTION:
Bacillary dysentery is an intestinal infection caused by Shigella bacteria. The term dysentery is also used with regard to intestinal infections caused by amoebas (amoeba dysentery) which will be described later. "Dysentery" actually means "disruption of intestinal function", and in this sense the term is used to describe medical conditions that result in diarrhoea with blood. The most serious type of this disease is caused by the bacteria *Shigella dysenteriae*.

This is a serious disease which, on a global basis, costs several hundreds of thousands of lives every year, mainly children less than 10 years of age.

GEOGRAPHIC DISTRIBUTION:
Shigella infections are found all over the world, but in our part of the world large outbreaks seldom occur. The cases that do appear are usually related to "import" by people who have been infected in areas where the disease is endemic. These regions
are mainly in countries in central and southern Africa. Larger outbreaks occur mainly where people live under crowded conditions with poor sanitation, particularly in refugee camps. The infectious dose of Shigella bacteria is very small. While other intestinal infections require infectious doses in a range of 10^5-10^6 bacteria, an intake of only a few dozen shigella bacteria can be enough to cause disease. Shigella bacteria are generally transmitted through water. Shigella bacteria only cause disease in humans, and humans are considered to be the only reservoir of infection.

**SYMPTOMS AND COURSE OF DISEASE:**
Shigellosis is mainly a paediatric disease, since it is mainly contracted by children less than 10 years of age. However, people of all ages may get the disease. The incubation period is short (1-3 days). The symptoms develop rapidly. The most important symptom is diarrhoea, usually bloody and accompanied by fever. Dehydration is often less pronounced than with other diarrhoea conditions. In addition to diarrhoea with blood, there are often symptoms originating from other organ systems. The most common, and also the predominant cause of death in children, is kidney damage with haemolysis (haemolytic-uremic syndrome). Kidney damage becomes evident several days after the diarrhoea symptoms.

**DIAGNOSIS:**
Diarrhoea with blood, accompanied by fever, should raise suspicion. Accurate diagnosis can only be confirmed by identification of the bacteria in faeces.

**TREATMENT:**
Fluid replacement is important. In severe cases intensive care is required.

In case of outbreaks it may be difficult to identify the source of infection, e.g. drinking water, because there may be only a few bacteria in the drinking water.

**VACCINE:**
There is no vaccine against Shigellosis.
3.5. ESCHERICHIA COLI INFECTIONS

**DESCRIPTION:**
*Escherichia coli* is normally found in large amounts in the intestines of both humans and animals. They are an integral part of the normal intestinal flora. However, some varieties of the bacteria (enteropathogenic *E. coli*) may cause disease. On a global basis, *E. Coli* is the most common cause of diarrhoea. A few types of *E. coli* may cause serious disease, with symptoms originating from other organs than the intestines.

**GEOGRAPHIC DISTRIBUTION:**
*E.coli* infections occur in countries with warm climates, poor hygiene, inadequate drinking water and sanitation, combined with high population density and generally poor health conditions. There are exceptions, for example the *E. coli* type 0157:H7, the so-called «hamburger bacteria» that has caused outbreaks in western, industrialised countries. For this *E. coli* type, cattle were the source of infection.

**SYMPTOMS AND COURSE OF DISEASE:**
The symptoms are diarrhoea, sometimes with blood, fever, stomach cramps, and dehydration. These symptoms will generally last a few days. The so-called "hamburger bacteria" can also cause haemolysis and kidney damage, so-called haemolytic uremic syndrome. This is a very serious condition which requires intensive care.

**TREATMENT:**
Fluid replacement is important. In some cases intensive care is indicated.

**DIAGNOSIS:**
Accurate diagnosis can only be confirmed by identification of the pathogenic *E. coli* bacteria in faeces.

**VACCINE:**
As for Shigellosis, there is no vaccine against *E. coli*-infection. Cholera vaccine may provide a degree of protection against strains with cholera plasmids that are transmitted by *E. coli*.

Outdoor toilet in Congo. Photo: FSAN.

Rat in Mali – disease spreader. Photo: FSAN.
3.6. OTHER BACTERIAL INTESTINAL INFECTIONS

Unhygienic conditions are important in the spread of diseases. *Photo: FSAN.*

*Campylobacter* and *Yersinia*. These bacteria are found all over the world, and their most common reservoirs of infection are chickens and pigs, respectively. They do not pose particular threats in Central Africa, but as with other food and water borne intestinal diseases, they can be found more frequently in areas with poor drinking water and sanitation. Yersinia infections are more common in temperate climate zones than in the tropics. They are generally considered to be less serious than the previously discussed infections. Mainly children are affected.
FOOD- AND WATER BORNE PROTOZOAN DISEASES

Protozoans are microscopic, single cell organisms, which are capable of reproduction within a host body, preferably in the gut. In this respect they resemble bacteria, but they are much larger, and can easily be seen under a microscope. They are much more resistant to disinfectants, and normal chlorination of drinking water is not enough to kill them.

The three most common food and water borne protozoan diseases will be presented here: Amoeba Dysentery, Giardiasis, and Cryptosporidiosis.
4.1. AMOEBA DYSENTERY

DESCRIPTION:
Amoeba Dysentery is caused by a single cell parasite, *Entamoeba histolytica*. The disease bears the name dysentery because it displays similar symptoms to the type of dysentery caused by Shigella bacteria, i.e. faeces with blood and mucous. Amoeba Dysentery is a serious disease, causing 100,000 deaths annually on a global basis. A large part of the population in impoverished parts of the world are healthy carriers of the disease, since only 10% of people infected with the disease, actually develop symptoms.

GEOGRAPHIC DISTRIBUTION:
Although the disease may be found anywhere in the world, Amoeba Dysentery is most often a problem in areas with poor hygienic conditions. In the cases where the disease appears in our part of the world, it has often been imported by people who have contracted the disease in "southerly" countries. In central Africa the disease is widespread. The feature that makes this disease so common, and so difficult to combat, is that most infected people show no symptoms, so they serve as reservoirs for the infection. Amoeba Dysentery involves mainly young adults and is seldom seen in children below the age of 5.

MEANS OF INFECTION:
The source of infection comes in the shape of very resistant cysts that are excreted in faeces from infected people. The cysts can survive for a long time in the environment; in water, soil, and in dust. Humans are infected by consumption of food or water.
containing the amoeba cysts, or by hands that have been in contact with infectious sources. People who show symptoms of Amoeba Dysentery are not considered to be particularly contagious, since the vegetative stages of the amoeba have limited viability in the environment. Therefore, it is mainly the silent bearers of the disease who serve as the main source of infection, because they excrete the parasite as resistant cysts.

SYMPTOMS AND COURSE OF DISEASE:
The incubation period is on average 2-4 weeks, but may be significantly longer. The symptoms will vary. The most common is an intense course, with bloody, slimy faeces, intense stomach pain, and fever. However, there may also be a more protracted course, with moderate belly ache, no fever, and alternating bloody diarrhoea and constipation. Although a certain degree of spontaneous recovery may be expected, Amoeba Dysentery should be approached as a chronic disease which must be treated. In rare cases an amoeba can enter the bloodstream, subsequently giving rise to abscesses, particularly in the liver. This will present as a chronic condition which must be treated.

TREATMENT:
Fluid replacement is important. There are specific drugs which are fairly efficacious.

DIAGNOSIS:
Amoeba Dysentery may be difficult to diagnose. Accurate diagnosis can only be confirmed by identification of the amoeba in faeces. Faecal specimens must be fresh and warm, in order to identify the parasite (use a heated bed pan for the faeces sample).
These diseases are caused by the single cell parasites *Cryptosporidium* and *Giardia*, respectively. To a large degree, the diseases resemble Amoeba Dysentery, with similar geographic spread and means of infection. As a rule, they cause much milder disease. In contrast to Amoeba Dysentery, mainly children are affected.
A large number of viral diseases can be transmitted through food, water, or body fluids. For simplicity, they may be divided into two groups:

Diseases that primarily exhibit symptoms from the digestive tract, such as diarrhoea and fever: These are relatively harmless, and often occur in our part of the world. This group will be discussed together later in this chapter. An example is Norwalk (=Norovirus), which is often the cause of large outbreaks of diarrhoea and vomiting at institutions, such as nursing homes, and some times military bases.

Diseases that cause symptoms from other organs: Viral hepatites are the most important among these diseases. Poliomyelitis spreads through soil and water, but due to vaccination programmes this disease is nearly eradicated, therefore it is less important.

Viral hepatitis is a common term for viral infections that lead to liver disease. Such diseases may be caused by widely different viruses, resulting in disease of varying severity. They also spread in different ways. There are four viral hepatites: A, B, C and E.

Type A and E are spread by the faecal – oral route, type B spreads via blood or sexual contact, and type C is transmitted through blood transfusions. However, there is currently less knowledge about hepatitis C.
VIRAL HEPATITIS

GEOGRAPHIC SPREAD:
The disease is endemic in all continents, but less in Europe, North-America, Australia, New Zealand and Japan. It is particularly widespread in the poorer regions of the world. Most cases occur in areas with inadequate sanitary conditions and poor hygiene.

MEANS OF INFECTION:
The virus is excreted in the faeces of infected people, and the disease is spread when food or water is contaminated by faeces or sewage containing the virus. There are no insects or other vectors that transfer the disease. Humans are the only disease reservoir.

It must be emphasised that infected individuals may excrete the virus, thereby posing a risk of contagion, long before they show any sign of the disease.

SYMPTOMS AND COURSE OF DISEASE:
Mainly adults develop symptoms of the disease. Children may also be infected, but they exhibit few, if any, symptoms. However, they will excrete virus, and thus be a reservoir of the disease.

The incubation period is relatively long, varying from at least one week, to 6-7 weeks. The disease starts with high fever, fatigue, nausea, abdominal pain, and diarrhoea. After a few days, icterus will commonly develop, as a result of liver damage. The disease is seldom fatal in individuals without concomitant disease. Convalescence is fairly long (weeks to months). About 10-15% of persons who have suffered the disease will experience relapses up to as long as one year after the first appearance of symptoms.

TREATMENT:
As for other viral diseases there is no specific treatment. Antiviral drugs and corticosteroids have no effect in the acute phases of the disease.

VACCINE:
There is an efficacious vaccine against Hepatitis A. The vaccine is included in the standard regime for foreign assignments.

PRACTICAL MEASURES:
Infection can be avoided through strict personal hygiene, particularly washing hands. In areas where the disease is widespread, it is important to avoid food and water that has not been prepared or served in an acceptable manner. Boiling food and water kills the virus. Safe handling of waste from latrines and rubbish is important.

5.1. HEPATITIS A
5.2. HEPATITIS E

This disease has a similar geographic spread, means of infection, and disease course, to those of hepatitis A. However, there is a higher prevalence in central Africa than in the rest of the world. A large outbreak was reported in South Sudan in the autumn of 2004.

The disease primarily affects children. Also pregnant women in the last trimester are particularly vulnerable, with relatively high mortality rates.

**PREVENTION:**
The same measures as those recommended for hepatitis A. Food and drink must come from a reliable source. Good hygiene, particularly hand hygiene. Appropriate waste treatment from latrines and refuse.

**VACCINE:**
There is no vaccine against hepatitis E.
5.3. HEPATITIS B

GEOGRAPHIC SPREAD:
Hepatitis B is hyperendemic in Africa, Southeast Asia, and South America, and the risk is high in Russia, eastern Europe, Canada, Alaska and Greenland. Globally about 2 billion people are constantly exposed to the infection, and 350 million have chronic infections. In Europe, about 900,000 new cases are reported annually, of which 90,000 are considered to be chronic infections.

MEANS OF INFECTION:
Humans are considered to be the only source of infection. The infection is sexually transmitted through body fluids, or through blood and blood products. The infection can also be transmitted through other tissue fluids. Sexual transmission is the most common means of infection. Other routes include blood transfusion, surgery or dental procedures, or through acupuncture, tattooing or piercing.

SYMPTOMS AND COURSE OF DISEASE:
The incubation period for hepatitis B varies from 1 to 6 months. The initial symptoms are similar to those of acute hepatitis, with severe nausea and vomiting, abdominal pain and weight loss, fever, and sometimes jaundice. However, sometimes patients may be asymptomatic and without jaundice. The disease may progress to a chronic disease. Among patients with chronic hepatitis, 25% develop liver cirrhosis, and 20% of these will in turn develop hepatocellular carcinoma. Patients who have had hepatitis B and cleared the infection, will have life-long immunity.

TREATMENT:
Interferon α treatment may be employed in cases of chronic hepatitis B, but it is only effective in half of the cases.

There is no specific treatment for acute hepatitis. General recommendations include rest and a healthy diet. Exercise is recommended when the patient feels better.

Alcohol must be avoided in the acute phase.

VACCINE:
There is an efficacious vaccine against hepatitis B, and it is included in the standard regime for foreign assignments.

PRACTICAL ADVISE:
Avoid unsafe sexual activity, use condoms. Only visit clinics approved by your national authorities.

In case acute treatment is required, it is sensible to bring your own sterile equipment. Any dental treatment should be taken care of before leaving Norway.

Never undergo acupuncture, tattooing, or piercing abroad (even in Norway this is risky).
5.4. HEPATITIS C

GEOGRAPHIC SPREAD:
Hepatitis C is found all over the world. Among blood donors, high antibody titres have been found in Italy, Spain, Central Europe, Japan, and in certain areas in the Middle East; including 19% of Egyptian blood donors.

MEANS OF INFECTION:
Information about hepatitis C is limited, and in about 30% of the patients, the route of infection is unknown. It may be assumed that blood transfusion and infection by contact with blood products are important sources of infection. About 35% of patients with hepatitis C are known intravenous drug abusers.

SYMPTOMS AND COURSE OF DISEASE:
Most infections are asymptomatic. Fewer than 30% have non-specific symptoms. Some develop a mild jaundice. Fulminant hepatitis has been described. Between 50% and 80% cannot clear the virus within 6 months, and they develop chronic hepatitis C. After 20 years, chronic hepatitis C will lead to liver cirrhosis in more than 20% of the patients. The risk of hepatocellular carcinoma after 20 years is 1-5%, and is more likely to develop in men than in women.

TREATMENT:
Chronic hepatitis C is treated with interferon α, just as hepatitis B.

VACCINE:
There is no vaccine against hepatitis C.

PRACTICAL MEASURES:
Only use tested blood products. Avoid contact with any material that may be contaminated (use gloves, masks, etc.). Safe sexual contact (condoms reduce the risk). Use sterile, single use equipment for injections.

Animals from the bush may spread disease.
Photo: FSAN.
5.5. TRAVELER’S DIARRHOEA

Traveller’s diarrhoea is a common term for several less severe types of diarrhoea, sometimes accompanied by fever and other signs of intestinal infections. Traveller’s diarrhoea is defined as 4 bouts of diarrhoea within 24 hours, accompanied by at least one of the following symptoms: Nausea (10-70%), vomiting (10%), abdominal pain (80%), fever (10-30%), blood in the faeces (10%).

CAUSE:
Traveller’s diarrhoea is caused by “all sorts” of microorganisms; ETEC (enterotoxin producing E. coli), Shigella, Salmonella, Campylobacter, Norwalk/Norovirus, Rotavirus, Cryptosporidium, Giardia and more. Therefore, diarrhoea may be caused by toxins, bacteria, virus or parasites, but in most cases, the cause remains unknown. In simple terms, traveller’s diarrhoea is caused by a temporary adjustment of the so-called normal intestinal flora, because of moving to a foreign environment.

GEOGRAPHIC SPREAD OF DISEASE:
Traveller’s diarrhoea is the most common disease in travellers outside of industrialised countries. Between 20-60% contract traveller’s diarrhoea, usually by infection during the first week. Children and young adults (20-29 years of age) are most commonly affected. The risk is highest in Africa, India, Southeast Asia, and South America.

DIAGNOSIS:
The diagnosis is determined by clinical examination.

TREATMENT:
Symptomatic treatment with fluid and electrolyte replacement is recommended. Intestinal motility regulating drugs may be used. Loperamide (Imodium® 2 mg tablets) may be given in cases of uncomplicated diarrhoea (no fever, minor abdominal pain, and no blood in stools) in order to reduce bowel movement. Four mg (2 tablets) should be given initially, followed by 2 mg (1 tablet) after each bout of diarrhoea, up to a maximum dose of 16 mg (maximally 8 tablets) in 24 hours. Antibiotics are often used for self-medication. In such cases, treatment should start immediately, preferably within 24 hours from the appearance of the first symptoms. Ciprofloxacin (Ciproxin® 500 mg tablets) may be taken once daily with good effect.

PREVENTION:
The risk of infection can be reduced by drinking bottled water from an approved supplier, avoiding ice cubes, not eating salads, only eating fruit that can be peeled, and eating food that is well cooked or fried, and recently prepared. Avoid food that has been heated in restaurant buffets. Good hygiene and frequent hand washing is recommended.

VACCINE:
Dukoral® is a vaccine against cholera. It contains a combination of genetically engineered cholera toxin and inactivated cholera bacteria.
The term “food poisoning” is often used about illness caused by consumption of food. Most of the gastrointestinal infections described here would be described in colloquial terms as “food poisoning”, even though the correct term should be “infection”. In reality, the term “food poisoning” only refers to conditions that are caused by toxins (poisons) that bacteria produce in the food before it is eaten. However, the difference is not always clear. In classical food poisoning, caused by bacterial toxins in spoiled food, the symptoms will usually appear more rapidly, be more intense, and disappear more quickly than with actual gastrointestinal infections. Food poisoning is caused by toxins that originate from bacteria found all over the world (E. coli, staphylococci, bacillus species, and clostridia). Classical food poisoning is caused by poor kitchen hygiene, ignorance about storage temperatures, and about methods of food preparation. This can occur
just as often in our part of the world as in other regions. Food poisoning will therefore not be discussed further.
6

VIRAL HAEMORRHAGIC FEVER (VHF)
(MARBURG, EBOLA, LASSA)
Viral haemorrhagic Fever (VHF) is a common term for viral infections that often have bleeding as the most obvious symptom. They can be caused by several different viruses. The most feared, Ebola and Marburg, are very serious diseases, often with a mortality higher than 50%. They are relatively uncommon, usually with only a few hundred cases per year. Outbreaks of these diseases often get extensive media coverage, and the international community (the World Health Organization – WHO) allocates substantial resources to learn more from each outbreak.

**GEOGRAPHIC SPREAD:**
With the exception of one case of laboratory infection in Germany (Marburg) in the sixties, all reported cases are from Africa. The core area for these diseases is central Africa south of Sahara, on both sides of the equator. A somewhat milder type of VHF, but still a very deadly disease (40% mortality), Lassa Fever, is limited to western central Africa. Most of these viruses have their reservoir in animals, preferentially some ape species, but the statistics are unreliable. The main reservoir for Lassa Fever is found in small rodents. The main reservoir for Ebola is in bats. Other milder types of VHF, for example Crimean Congo Haemorrhagic Fever, are widespread over several continents.
MEANS OF INFECTION:
Ebola, Marburg- and Lassa Fever are generally less contagious. They are transmitted from person to person by direct contact, mainly by contact with blood or other body fluids from sick or dying patients. In the cases where there have been large outbreaks, mostly the relatives and local health workers have been infected. This is mainly due to inadequate hygienic measures and ignorance about the means of infection. Partially it is also due to culturally determined behavior related to the families gathering around the dead or dying family members.

COURSE OF DISEASE:
The period of incubation varies from 3 to 20 days. The diseases run a dramatic course. Initially the symptoms are non-specific, with high fever, sweating, lack of appetite, etc. The more well-known symptoms of bleeding from all body orifices, appear later. The bleeding is caused by the ability the viruses have to destroy hematopoietic tissues, and thereby also the blood factors necessary for normal coagulation.

TREATMENT/PREVENTION:
There is no specific treatment or available vaccine for most types of VHF. In connection with the large outbreak of Ebola in west Africa in 2014 and 2015, there were intense efforts to find medical treatments and a vaccine. In the autumn of 2015 a vaccine against Ebola was successfully developed, among others with Norwegian assistance. Treatment of Ebola is symptomatic, with replacement of fluids, with salt and nutrients.
Gorillas in Central-Africa can spread disease. Photo: FSAN.

Glossary/Medical terms:

**Asthenia**: Weakness

**Conjunctivitis**: Inflammation of the outer part of the white of the eye, and inner eyelid

**Encephalitis**: Inflammation of the brain

**Portal circulation**: Veins that carry blood from the gut to the liver

**Intrahepatic**: Within the liver

**Hepatosplenic**: Pertaining to the liver and spleen

**Hepatosplenomegalia**: Enlarged liver and spleen

**Hematuria**: Blood in the urine

**FSAN**: Norwegian Armed Forces Joint Medical Services

**Fibrosis**: Formation of excessive connective tissue

**Hydronephrosis**: Fluid trapped in the kidney, usually by obstruction of urinary flow

**Haemmorhagic diarrhoea**: Diarrhoea with blood

**Macular exudate**: Fluid from the macula, a structure in the eye

**Maculopapillary**: Related to the macula, a structure in the eye

**Myocarditis**: Inflammation of the heart muscle

**Pseudopolyps**: Scar tissue resembling polyps

**Ulcerating colitis**: Inflammation of the large intestine with open lesions

**Embolise**: To block a channel, e.g. a blood vessel or urinary pathway

**Sequelae**: A pathological condition resulting from a disease, injury, therapy, or other trauma

**Vasculitis**: Inflammation of blood vessels
The Norwegian Armed Forces Joint Medical Service (FSAN) was established in the UK in November 1941, and currently has its headquarters at Sessvollmoen base in Ullensaker municipality.

The organisation provides the Norwegian Armed Forces with various medical services, and ensures a strong civilian – military collaboration, in order to secure operationality and combat capability.

Since World War II, the Norwegian Armed Forces Joint Medical Service has played an important rôle in several of the international operations that the Norwegian Armed Forces have taken part in around the world.

The goal of the organisation is to strengthen the medical profession and to improve the organisation, so that FSAN can offer "Health for combat capability".