
# DIRECTORATE OF LEARNING SYSTEMS

## DISTANCE EDUCATION PROGRAMME

COMMUNICABLE DISEASES COURSE

**Unit 8**
**Vector Borne Diseases**


**Allan and Nesta Ferguson Trust**

# Unit 8: Vector Borne Diseases

A distance learning course of the Directorate of Learning Systems (AMREF)



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The African Medical Research Foundation (AMREF) wishes to acknowledge the contributions of the Commonwealth of Learning (COL) and the Allan and Nesta Ferguson Trust whose financial assistance made the development of this course possible.

# Contents

**ABBREVIATIONS**

| | |
|---|---|
| AIDS | Acquired Immunodeficiency Syndrome |
| CCF | Congestive cardiac failure |
| CSF | Cerebral spinal fluid |
| CNS | Central nervous system |
| DDT | Dichloroethyltrichloroethane(Decophane) |
| DPHO | District public health officer |
| HIV | Human immunodeficiency Virus |
| IV | Intravenous |
| LBRF | Louse-borne relapsing fever |
| LLITNS | Long lasting insecticide treated net |
| OV | Onchorcerca Vulvulus |
| TB | Tuberculosis |
| Tbb | *Trypanosoma brucei brucei* |
| Tbg | *Trypanosoma brucei gambiense* |
| Tbr | *Trypanosoma brucei rhodensiense.* |
| TBRF | Tick-borne relapsing fever |
| VBD | Vector-borne diseases |
| WHO | Would Health Organization |

# INTRODUCTION

Welcome to Unit 8 of your course on communicable diseases. In this unit we will cover the various diseases that are transmitted through vectors. In your practice you have probably come across various vectors such as insects, rodents or other disease transmitting agents that pose great danger to our communities. These diseases sometimes come in epidemics that cause very high levels of fatalities.

Vector borne diseases are of great economic importance to Africa, especially in the sub-Saharan Region. This is mainly because they are associated with high levels of mortality and morbidity. Some of the diseases we shall look into in this unit are:

- Relapsing fever,
- Bancroft's filariasis,
- Onchocerciasis,
- Yellow fever,
- Trypanosomiasis,
- Plague,
- Schistosomiasis,
- Dracunculosis,
- Leishmaniasis.

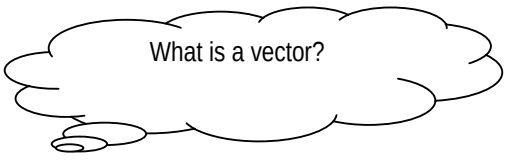
Malaria is a common and the most significant vector born disease in Africa. However, because of its importance, it will be featured as a unit on its own.
In each of this diseases, we shall look at the cause of the disease, the clinical features, diagnosis, treatment, disease distribution and control and prevention.
At the end of every disease there are some self evaluation questions. These are to help you know how well you have understood the section.

## *Specific objectives*

By the end of this unit you should be able to:

- Identify common vector borne diseases within your working environments;
- Discuss the principles of vector control.
- Describe the spread of vector borne diseases;
- Diagnose and describe the management of vector borne diseases;

# Section 1: Introduction To Vector Borne Diseases




A vector is an organism that transmits or carries a disease from one human to another or from one animal to another and in some cases from an animal to a human being. Therefore a vector simply is a carrier of disease.

The organism that causes the disease is carried within the vector, without causing any harm to it. These disease-causing organisms may undergo some physiological development of their life cycle while they are within the vector. The developmental changes that the organism undergoes while within the host are called *extrinsic incubation period*.

Therefore, for an organism to qualify to be a vector, it must have a disease-causing organism within its body system and that disease-causing organism must undergo some physiological changes.

Some insects do the physical transportation of the disease-causing organism, without necessarily hosting these organisms in their own system. This does not qualify such insects to be a vector. For example, a housefly transports bacteria and amoebic cysts to contaminate food. This does not qualify the housefly to be a vector.

Most vectors are usually insects that would usually acquire the disease-causing organism by sucking blood from an infected host and later transport it to an uninfected host. However, there may be other ways of disease transmission. Infections may be transmitted trough skin breaks and abrasions either from the foecal mater of an infected insect or from body fluids of an insect when it is crashed. In addition to insects, flukes, worms and rodents have also acted as carriers to certain diseases.

## *Distribution Patterns of Vector Borne Diseases*

Vector borne diseases are mainly concentrated in areas where there are suitable conditions for the vectors to thrive. Most of the vectors breed, feed and thrive well in certain environmental conditions and, therefore, most vector borne diseases are common in certain environmental and ecological conditions.

In Africa many vector borne diseases such as malaria, schistosomiasis, filariasis and trypanosomiasis are *endemic.* This means they are present all the time in certain geographical areas. However in most areas there are seasonal variations in the number of infections. These variations are mainly due to seasonal climatic changes that favour the breeding of the vectors.

In other instances the diseases have occurred in sudden onsets that cause high levels of infections, mainly called *epidemics*. Epidemics of vector borne diseases may appear in areas where they have occurred before or in areas where those diseases have never before occurred. In Africa, for example, some very serious epidemics of yellow fever have been experienced in Nigeria, Sudan, Ethiopia and Ghana.

Write down at least three factors that may affect the distribution of vector borne diseases and then compare your answer with the factors given below.

Some of the factors that affect the distribution of vector borne diseases are:

- **Meteorological factors**: certain climatic conditions such as rainfall or high temperatures may favour the breeding of certain vectors.

- **Human factors**: Certain human behaviours have predisposed them to high infection rates thus causing epidemics, for example nomadic migrations to areas of high vector concentrations.
- **Socio economic factors**: poor living conditions favour the presence of certain vectors such as *Pediculosis* which is a vector for relapsing fever.

- **Vulnerability factors**: change in vulnerability levels, such as malnutrition and HIV/AIDS, have shown a sudden increase in vector borne diseases in the affected populations.

### *Effects of Global Warming and Climate Change to Vector Borne Diseases*

As mentioned earlier in the introduction, the distribution of vector borne diseases is largely determined by the geographical distribution of the vectors themselves.

The greenhouse effect of carbon emissions has been a global warming that has already caused a climate change in most parts of the world. This rise in global temperatures has opened up new habitats for vectors in areas where they were previously absent, for example, mosquitoes which were mainly common in the warmer and low lying areas of the tropics are now found in cooler and higher areas. This has led to high rates of exposure to highland malaria to previously non-immune highland populations in Kenya, leading to fatal epidemics.

It is not only malaria but other vector borne diseases, previously quite uncommon outside the tropics that can now be found there more and more often.

Now that we have learnt about the distribution patterns of vector-borne diseases, let us find out how we can control those vectors so as to prevent infection in the communities. Section 2 will focus on the principles of vector control.

## Section 2: Principles Of Vector Control

Vector-borne diseases (VBD) are of great socio-economic importance in developing countries as they contribute significantly to morbidity and mortality in this region. Vector control measures have been put in place in many parts of the world and have been quite successful, yet in Sub-Saharan Africa, their prevalence still remains high.

The easiest way of preventing VBD is through vector control. In order to eradicate the vector-borne diseases, we need to eradicate the vectors that harbour them. For this we need a good understanding of their life cycle so as to attack them before they enter the human body and cause disease. Vector-borne diseases have an extrinsic cycle where the organism exists outside the human being. This offers a good opportunity to control the disease without necessarily interfering with the behaviour of human beings. However, a good knowledge of vector behaviour is vital. Therefore,

before embarking on any vector control exercise we need to have a good understanding of the vector's:

- Breeding places,
- Nesting places,
- Feeding patterns
- Flying distances.

Different vectors have different behaviours. Some bite during the day while others attack during the night. Therefore, in certain areas more than one vector control measures have to be put in place at one particular time. The main principles of vector control are:

- Reducing the reservoir host,
- Controlling the vector population,
- Protecting the susceptible host.

*Make a list of four different ways of vector control, then compare your list with the one given below.*

There are four main ways of vector control:

- Targeting the adult vector using Insecticides;
- Targeting the larvae stage using larvicides;
- Preventing the breeding of the vector through environmental interventions;
- Protecting the susceptible hosts.

We shall now look into each one of these methods.

## *Use of Insecticides*

Insecticides normally target to kill the adult vectors. Insecticides are either residual or non residual. Residual insecticides are mainly organic chemicals that when applied on surfaces will remain effective for several months. These include DDT, Dieldrin, Lidane, HCH and organophosphates. DDT is not easily broken down and easily contaminates the environment therefore most countries do not advocate for its use.

## *Use of Larvicides*

Larvicides mainly target the larvae. They are mainly used in the breeding sites. However, most larvicides are easily diluted by water. Oils applied on water surfaces, over which the larviciides are sprayed, have proved effective to some extent. Polystyrene powder, applied on pit latrines can also be used.

## *Environmental Interventions*

Prevention of breeding of vectors is normally a cheap and effective way of disease control. Some of the environmental control measures include:

- Clearing of bushes and grass along water bodies:
- Draining and collecting all containers that may hold water;
- Covering water containers and tanks with lids;
- Draining waterholes, ditches and any unwanted water bodies around the villages;
- Disturbing snail habitat by changing the water levels, filling or draining the water body and use of molluscides.

## *Protecting Susceptible Individuals*

These interventions are meant to prevent the vector from coming into contact with a susceptible individual. The most common ways through which this is achieved are:

- Use of insecticide treated bed-nets, such as the long lasting insecticide treated nets (LLITNS);
- Use of house screens;
- Use of insect repellents;
- Use of protective clothing;
- Avoid staying outdoors at night.

***Figure 1: A woman demonstrating use of LLITNS. The front flap will be dropped at night to cover her and the baby.***

## *Community Involvement in Vector Control*

To improve programme success in vector control, involvement in vector control measures of the community members, community leaders and the local governments is vital. This not only improves the outcome of such programmes but also ensures their sustainability. It is of vital importance to involve the community in:

- Bush clearing and habitat control;
- Mosquito net distribution;
- Indoor residual spraying;

- Mass treatment campaigns;
- Pool drainage and removal of containers.

***Figure 2: Community directed treatment of Onchorcerca Vulvulus in Nigeria***

Before we proceed any further, let us first look at the various vectors and causative organisms that cause some of the most common diseases in Africa and more particularly, in our region of East Africa.

Now study carefully Table 1 below. It summarises for you the most prevalent vector borne diseases and their causative organisms

Table 1: Vector borne diseases and their causes

| **Vector** | **Vector species** | **Causative organism** | **Disease** |
|---|---|---|---|
| Mosquitoes | *Anopheles funestus* | *Plasmodium malariae* | Malaria |
| | *Anopheles gambie* | *Plasmodium malariae* | Malaria |
| | *Culex and anopheles* | *Wochereria banchrofti* | Filariasis banchrofti |
| | *Aedes spp* | | Yellow fever and dengue fever |
| Buffalo gnats | *Simulium damnosum* | | Onchocercosis |
| Black fly | *Simulium naevae* | *Onchocerca vulvulus.* | Onchocercosis |
| Biting flies | *Glossina palpalis Glossina morsitans* | *Trypanosoma brucei gambiense Trypanosoma* | Trypanosomiasis |

| | | | |
|---|---|---|---|
| | | *brucei rhodesiense* | |
| Cyclopes | *Dranculosis medinensis* | *Dracunculus medinensis* | Dranculosis(Guinea worm) |
| Lice | *Pediculosis corporis* *Pediculosis capitis* | *Borrelia recurrentis* | Relapsing fever Epidemic typhus |
| Fleas | Xenophyslla cheopis | *Arbovirus* | Plague |
| Soft ticks | Orthodorus moubata | *Borrelia recurrentis* | Relapsing fever |
| Hard ticks | Rhipicephalus spp | | African tick borne typhus |

We shall now turn our attention to each one of these diseases, their vectors and causative agents and study them in detail.

# Section 3: Banchroft's Filariasis

Bancroft's filariasis or Bancroftian filariasis is a filarial disease that is caused by the filarial worm *Wochereria banchrofti* , a nematode worm. It is commonly known as Elephantiasis.

In Africa filariasis is commonly found along the tropical coastal belts where the temperatures are high and also along the low lying lake regions. The disease is transmitted by culex and anopheles mosquitoes and therefore it is uncommon in highlands and cooler areas. The disease is more disfiguring rather than dangerous. In some hot costal belts its prevalence is as high as 30% among adult males.

*Figure 3: A view of Wochereria Filarial worms*

## *The Life Cycle of Wochereria Microfilaria*

Banchroftian filariasis is caused by *Wochereria Banchrofti*, a nematode worm. It is transmitted by two species of mosquitoes: Culex and *anopheles*. When a mosquito sucks blood form an infected person, it picks the microfilaria into its system. While in the mosquito, the microfilaria undergoes some changes and after ten days the microfilaria larvae can be transmitted to another person. The mosquito then deposits the microfilaria larvae in the subcutaneous tissue of a healthy person during a blood meal. The larvae find its way into the lymphatic system where they mature in the lymphatic vessels. It takes about one year for the larvae to mature into an adult filarial worm. The filarial worm is normally 4-8 cm long and 1.2 cm in diameter when

fully mature. The microfilaria appears in the peripheral blood of the victims mainly at night when the mosquitoes suck blood.

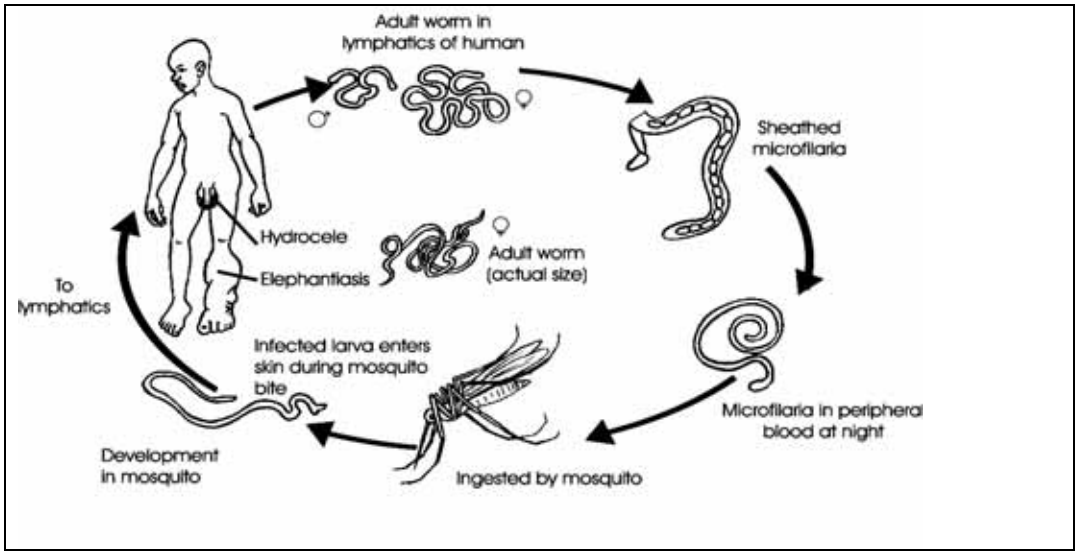



*Figure 4: The life cycle of Wochereria Microfilaria*

## *Clinical Presentation*

The microfilaria causes a reaction due to the presence of foreign proteins within the lymphatic system. After the microfilaria worms die, they release proteins in the system which cause even stronger immune reaction by the host body. The clinical picture of the disease can be classified into three categories:

- Acute phase,
- Sub-acute phase,
- Chronic phase.

### Acute Phase

The acute phase begins a few months after the infection and can continue for up to one year. This stage is mainly due to the hypersensitivity reactions to the presence of the larvae. It is characterised by the following symptoms:

- Fever,
- Lymphadenopathy,
- Eosinophilia.

At this stage the microfilaria are not yet present in the blood stream and the worms are not yet mature.

### Sub-acute Stage

The sub-acute stage occurs mainly one year after the infection has taken place and it is mainly due to immune reactions to the presence of adult microfilaria. This stage is characterized with fever, lymphadenopathy, funiculitis and epididymitis. The symptoms may occur, disappear after some time and then recur. There are lungs symptoms including dry cough and pulmonary oesinophilia.

### Chronic Stage

After years of repeated attacks the lymph gland and lymph vessels become obstructed. This results into lymphoedema, the oedema mainly affecting the legs and the scrotum (elephantiasis). It may also involve some other body parts such as the vulva, breasts and arms. The skin also becomes irritating, thick and keratotic.

## *Diagnosis*

The diagnosis is done mainly through the clinical presentation. If a blood smear is taken, the microfilaria will be found to be present in the peripheral blood. Since the microfilaria only migrate to the peripheral blood stream at night, the blood sample should be taken at night between 10.00 p.m. and 2.00 a.m. Fluids from the oedematous areas can also be taken for examination.

## *Management*

The drug of choice is Diethylcarbamazine (Hetrazan or Benocide). It kills microfilaria and also has some effect on the adult worms. However, it should not be used during the acute stage as it is known to cause severe allergic reactions. The treatment is normally combined with anti histamines to minimize the effects of allergic reactions. In chronic stages chemotherapy does not yield much benefits and surgery is normally recommended.

## *Prevention and Control*

The principle of control is based on vector control measures. Educate the community to destroying the vector breeding areas by ensuring that any stagnant water is drained and by covering pit latrines. Preventing mosquito bites by use of mosquito nets and insect repellents is equally important. The human reservoir can be reduced by mass treatment campaigns these should continue for at least ten years.

# Section 4: Onchocerciasis

Onchocerciasis is a disease caused by a filarial worm nematode *Onchocerca volvulus.* The disease mainly manifests in the form of skin lesions and nodules that mainly affect the bony surfaces and later causes eye lesions that easily result into blindness.

*Take Note*

*Onchocerciasis is a major cause of blindness in areas of Africa where there is repeated infection with the microfilaria.*

The disease is transmitted by a small black fly called Simulium. The most important species is *Simulium damnosium.* The Simulium breed in well oxygenated water

hence the proximity of the disease to water, and most frequently fast flowing or “white water.” The average flying distance of the simulium fly is 40 kms, therefore, the infections are common within this radius around fresh water bodies.

## *Life Cycle of Simulium Damnosium*

River blindness is caused by *Onchorcerca.Vulvulus* (OV). The adult worm is about 35 cm long and about 15 mm in diameter. It can live up to 15 years with the females producing thousands of microfilaria that can live up to five years.

*What is the life cycle of Simulium damnosium?*
*Try to draw your own sketch of the life cycle, then compare it to Figure 5 below.*

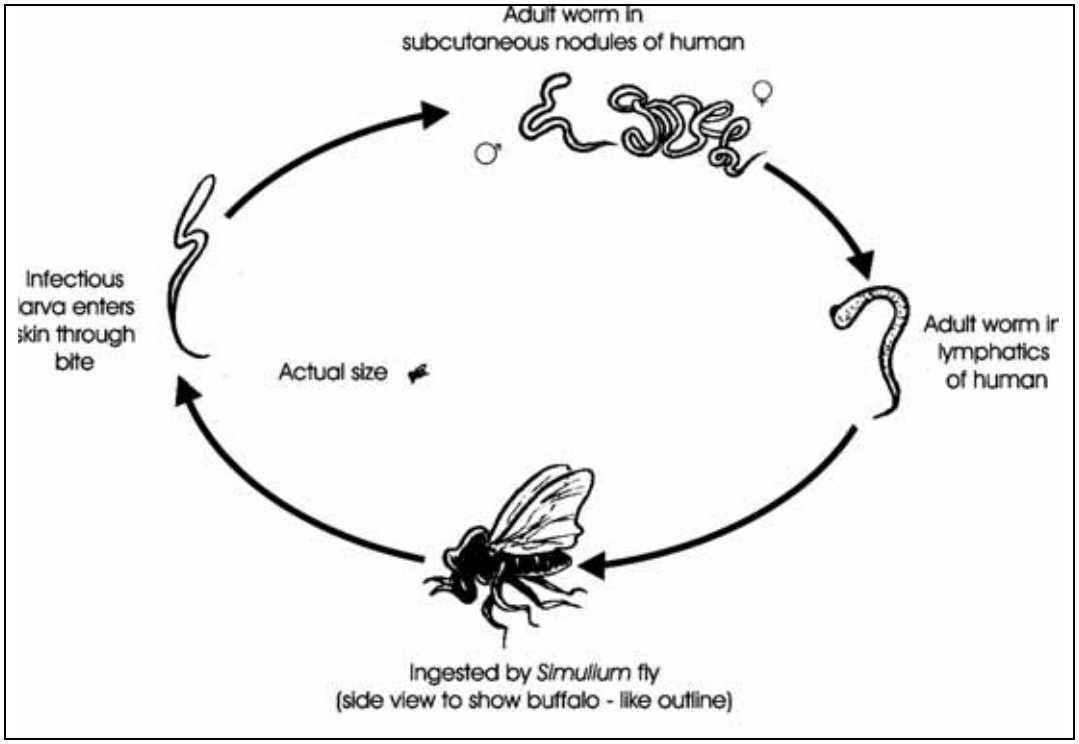



*Figure5: The Life cycle of Simulium Damnosium*

The vector Simulium Domnosium picks up the microfilaria when it sucks blood from an infected person. While within the simulium, the microfilaria develops into an infective larva that is passed on to a new host when the simulium bites again.
The simulium fly mainly attacks people outdoors mostly during the day but not in very bright light, therefore, the bites mainly happen in the morning and evening.

*Figure 6: The simulium breeding ground is mostly fresh flowing waters*

## *Clinical Presentation*

The larvae injected in the subcutaneous tissue develop into adult worms that collect in small colonies under the skin. These colonies form hard palpable nodules that are mainly found on bony skin surfaces e.g. elbow, shoulder, scapular, skull, ribs, and iliac fosa. The microfilaria moves to various body parts including the eye, the skin and sometimes the brain.

## *Onchocerciasis and the Eye*

If the microfilaria invades the eye it provokes inflammation in all parts of the eye. Different structures in the anterior segment of the eye may be involved, including the cornea or the iris. At first there is corneal oedema then cornea spots will appear. Finally cataracts, iritis and glaucoma develop that cause blindness.

*Figure 7: Onchorcarciasis of the eye*

## *Onchocerciasis and the Skin*

Inflammation of the skin produces early reversible inflammatory changes which if untreated, can then become permanent. The early effects are severe itching which leads to scratching, minor trauma which becomes infected giving oedematous skin and involvement of lymph nodes.

*Figure 8: An elderly man suffering from OV. Note the leopards skin on the lower limbs.*

## *Diagnosis*

Diagnosis is mainly through clinical presentation and physical examination. The microfilaria are normally not found in the blood, therefore a blood test may not reveal their presence. Skin snips examined under microscope reveal microfilaria while the fluids from the nodules reveal the adult worms

## *Management*

Onchocerciasis is not a fatal disease. However, the complications are mostly not reversible. Most of the treatment modalities prevent the disease from progressing. Two treatment modalities have been on use. These are Diethylcarbamazine (Hetrazan) and of late Ivermectin (Mectizan).

Hetrazan has been traditionally used for the treatment of Onchocerciasis at a dose of 2mg/ kg body weight, three times daily for three weeks. However, it does not kill the adult worms.

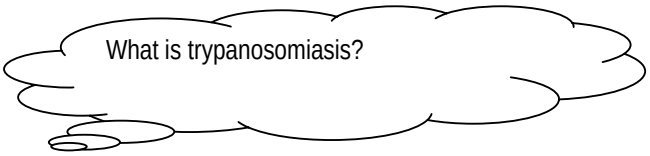
The treatment of choice since 1987 has been Ivermectin, a micro-filaricide (which kills microfilaria) but unfortunately does not kill the adult worm, so the treatment needs to be given for around 20 years. This treatment has the advantage that it has to be taken only once a year at a stat dose of one to four tablets depending on the body weight. The dose can be calculated by weight or height

Ivermectin is one of the safest drugs on the market but care must be taken in areas where there is co-endemicity between OV and Loa-Loa.

### *Prevention and Control of Onchorcerca Vulvulus (OV)*

Recent trials have shown that multiple treatments with Evermectin to all communities in the endemic areas are the best way of controlling OV. Mass and community based treatment campaigns have been developed in Nigeria, Ghana, Kenya and southern Sudan. These campaigns have advocated for community directed treatment with Evermectin. Of equal importance is destruction of the simulium habitats and breeding sites. The use of insecticides on the rivers and other simulium breeding sites has some effect. Barrier methods such as insect repellents may seve to prevent bites by the simulium fly.

## Section 5: TRYPANOSOMIASIS




Trypanosomiasis is a disease caused by a protozoan transmitted by the glossina fly, also known as Tse tse fly. It does not only affect humans but also cattle and wild animals. There are various species of trypanosomes but only two affect human beings. These are *Trypanosoma brucei gambiense* (Tbg) and *Trypanosoma brucei rhodesiense* (Tbr). The disease is often fatal and is characterized by chronic fever, general malaise, cerebral involvement and often death.

Tbg causes a slow progressive illness that is commonly called sleeping sickness, while Tbr causes an acute and rapidly developing illness that easily causes death. The disease is common in most of the tropical Africa and is of great economic importance, especially among the pastoralists population.

### *Life Cycle of the Tripanosomes*

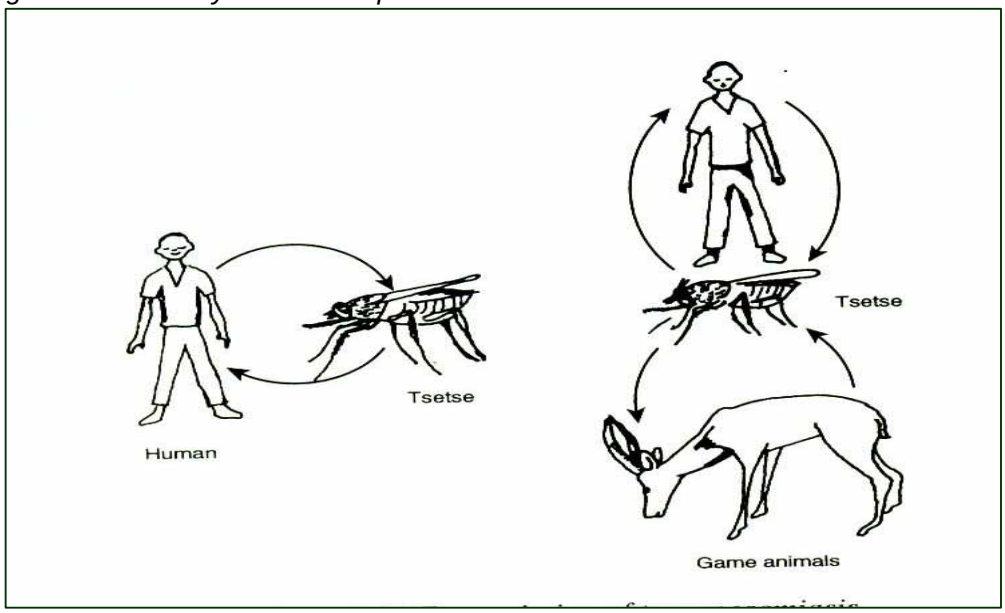
The disease is transmitted by the tse tse fly. There are two types of tse tse flies of importance in Kenya. The first one is the riverine (*Glossina Palpalis*) type that breeds along the rivers and lakes and is the main vector for *T. b. gambiense.* Infection occurs mainly along the rivers and the woodlands near water bodies. The second type is the woodland type *G. morsatans* that lives in lightly wooded areas and prefers to bite cattle and wild animals but may also bite humans.

In the dry season the tse tse flies are fewer and they recede to the areas with permanent water bodies where they breed. Tse tse flies bite on sight and are attracted by dark moving objects such as cattle and buffalo herds.

Once they bite an infected human or animal, they suck in the protozoa into their own system. The trypanosomes undergo some physiological changes once in the

Glossina which enable the fly to transmit them to an uninfected host. Contacts between the glossina and humans are not very common and the disease occurs sporadically among the communities which live in highly infested areas.

*Figure 9: The life cycle of the Tripanosomes*




## *Clinical Presentation*

The disease manifests in three stages: the primary or the chancre stage, the systemic or the blood stage and the cerebral or the sleeping stage.
The disease pattern in both Tbr and Tbg is the same but what differs is the speed at which the disease develops. In Tbr the disease is rapid and more severe while in Tbg it is slow and less severe.

### Primary Chancre) Stage

This stage is characterized with a painful undulating swelling at the site of the bite. It may last for one to two weeks and later resolve by itself. However, this stage is more often noticeable among the light skinned races.

### Systemic Illness (Blood) Stage

This stage is characterized with dissemination of the trypanosomes in the blood system and also into the lymphatic system. It is more rapid in Tbr than in Tbg.
It is characterized with the following symptoms:

- Fever,
- Lymphadenopathy,
- Pruritic rash,
- Hepatosplenomegally,
- Anorexia,
- Impotence and sometimes menstrual irregularities,
- Cardiac failure, mainly in Tbr.

**Cerebral (Sleeping) Stage**

This is the terminal stage of trypanosomiasis and it is characterized with Central nervous system (CNS) involvement after the trypanosomes have invaded it. In Tbg this stage takes about two years after the initial infection. However, in Tbr the stage occurs within a few months and is often rapid progressing and fatal. The stage is characterized with the following symptoms.

- Drowsiness and mental deterioration,
- Convulsions,
- Restlessness,
- Body weakness,
- Slow motor activities,
- Localized signs such as hemiplagia,
- Facial palsy,
- The patients progress to coma and death.

## *Diagnosis*

In endemic areas where there are no laboratory services the clinical picture and physical examination are instrumental in diagnosis. In areas where laboratory services are available, microscopy of the chancre fluids reveal the organisms. Other laboratory tests include blood smears, serological tests and cerebral spinal fluid (CSF) analysis.

## *Management*

Patients with trypanosomiasis should always be treated in the hospital under strict medical observation. This is so because the drugs used are highly toxic. Treatment modality depends on the type of trypanosomiasis and also on the stage the disease has progressed to. CSF should always be drawn to determine whether there is CNS involvement.

Table 2: Treatment schedule for Trypanosomiiasis

| | T.b,rhodensiense. | T.b.gambiense |
|---|---|---|
| CSF normal | Give a course of Suramine. | Give Suramine or Pentamidine. |
| If CSF is involved | Combine Suramine and IV Melarsoprol. | Give Melarsoprol combined with either Suramine or Pentamidine. |

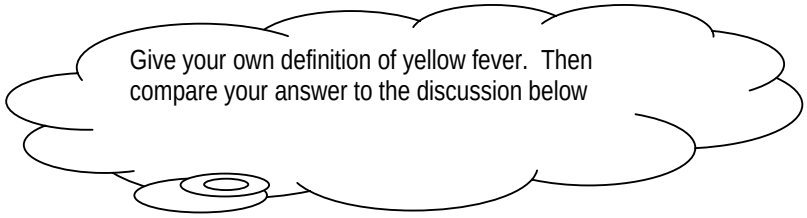
Dosages for these drugs should be followed strictly, especially in children because they have very high incidences of toxicity.

## *Prevention and Control*

Prevention and control of trypanosomiasis is based on early detection and treatment as well as vector control. Vector habitat destruction through bush clearing is an effective method. However, an environmental effect of harming the environment exists. Spraying of habitats with insecticides is also a method used to control the disease. Baited fly traps have been used with some degree of effectiveness in some

communities. However, there are no effective drugs that are used to prevent the development of the disease.

## Section 6: Yellow Fever




Yellow fever is an acute infectious disease caused by an arbovirus, transmitted to humans by the Aedes mosquito which acquires the infecton either from humans (urban type) or from animals (jungle type).

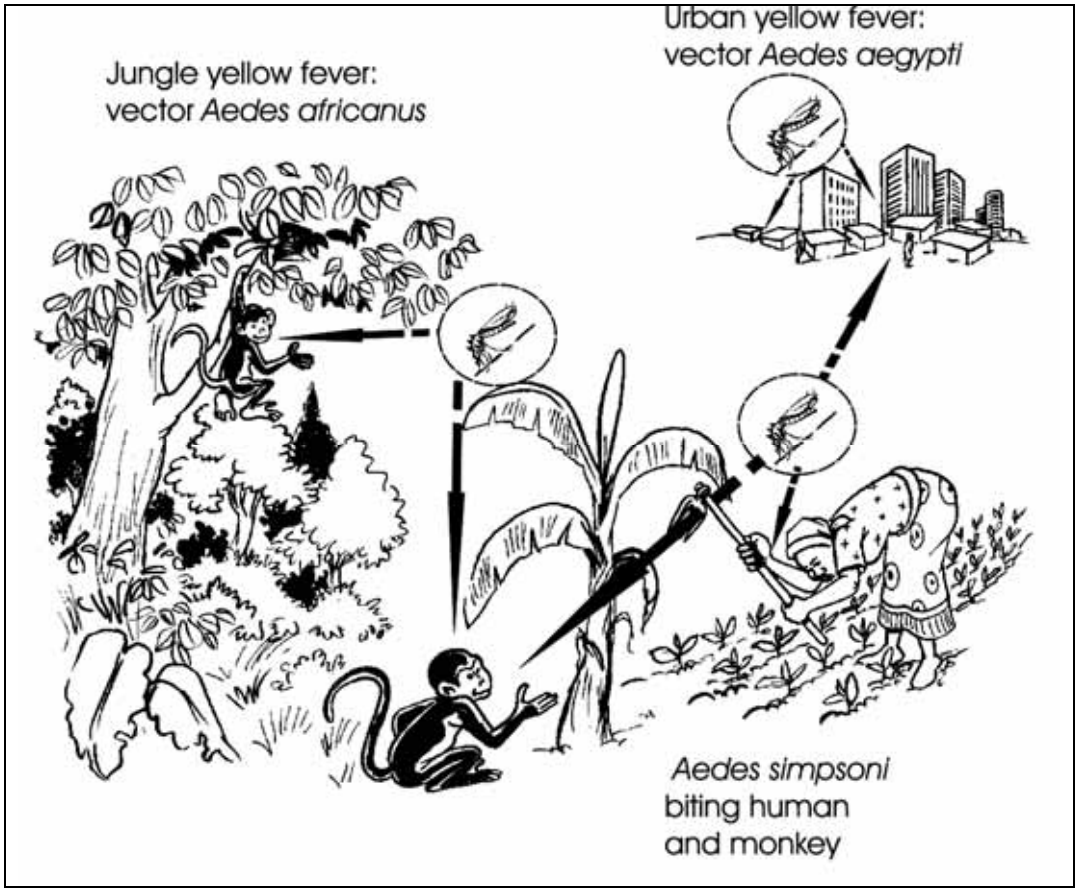



*Figure 10: Types of yellow fever and their vectors*

Yellow fever is characterized by a sudden onset of fever, rigors, headache, nausea, vomiting, jaundice and sometimes there are signs of kidney failure.

This disease is very common in the tropical African areas of Southern Sudan, Uganda and Kenya. However, it is not present in Tanzania. It spreads rapidly and quite often occurs in epidemics that have very high levels of fatalities.

## *Life Cycle*

Yellow fever is zoonotic and is transmitted to humans from highland monkeys and sometimes other forms of primates. The humans are often infected when a mosquito carrying the virus bites them during a blood meal.

## *Clinical Presentation*

The onset of yellow fever is often sudden with fever, headache, backache, and nausea and vomiting. The patients then experience bleeding mainly from the gums or nose. Sometimes this is accompanied by haematemesis (vomiting of blood) and melena (dark stools stained with blood).

Symptoms of liver infection set in after a few days leading to jaundice, liver failure and death easily results. Involvement of the kidneys is common, leading to low or no urine output, (albuminuria).

Patients who survive the first week of the infection usually recover and get active immunity from the infections.

## *Diagnosis*

Yellow fever is mainly diagnosed through high clinical suspicion in areas of high endemicity.

Laboratory investigation through serology is done as confirmatory tests.

### Differential Diagnosis

Most often the disease is confused with symptoms of:

- Hepatitis,
- Malaria,
- Reptospirosis,
- Relapsing fever,
- Hemorrhagic fevers such as Marburg and Lassa fevers.

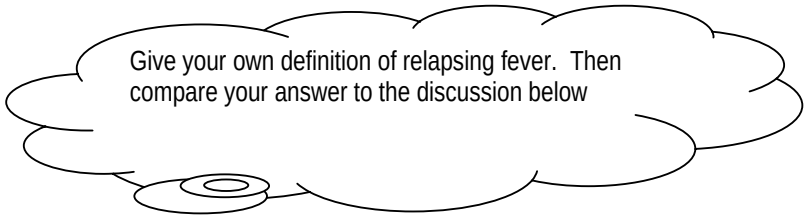
## *Management*

There is no specific drug for yellow fever, supportive care is advocated for.
Patients should be nursed in a well screened and ventilated room.
Avoid infections to other patients by the use of mosquito net.

### *Prevention and Control*

The most effective way of prevention yellow fever is immunization. In highly endemic areas children and adults should be immunized against yellow fever. Travellers moving from and to highly endemic areas should be immunized against the disease. Insect control in endemic zones by the use of insecticide treated bed nets must be advocated for. Residual indoor spraying for the houses within the endemic zones is also advocated for. Applying larvicides in areas where the mosquitoes breed is another method of preventing yellow fever.

## Section 7: RELAPSING FEVER




Relapsing fever is an acute systemic infection that is caused by spirochaeta of the genus Borrelia. It is characterized by alternating febrile episodes. It is also referred to as the recurrent fever, or tick fever. There are two types of relapsing fevers.

### *Louse-borne Relapsing Fever*

The louse-borne relapsing fever (LBRF) is transmitted by two types of lice. The most common is the body lice *Pediculus humanus corporis* and also the head louse *Pediculus humanus capitis*.

*P. corporis* are present in clothes that are worn continuously without adequate cleaning. They are common in overcrowded areas where the living conditions are poor. It is common in the slums, the prisons and the refugee camps.

The disease mainly occurs in epidemics in the above living conditions. Outbreaks have been reported in Rwanda, Sudan, Somalia and Uganda.

### *Tick-borne Relapsing Fever*

The tick-borne relapsing fever (TBRF) is transmitted by soft ticks (ornithodorous *moubata*) that normally live in the cracks and the crevices of the floors or on rodents and other animals. The disease is common where the housing conditions are poor. Adults in endemic areas are semi- immune, while the disease mainly affects the newcomers and children.

### *Life Cycle of the Borrelia*

As we said earlier on, recurrent fevers are caused by spirochetes of the genus Borrelia. Louse borne relapsing fever is caused by *Borrelia recurrentis*. It is

transmitted by body lice, for which man is the reservoir. When a louse sucks the blood of an infected person, the spirochaeta multiply in the body of the louse and as it bites another person it transmits the disease. However, the spirochaeta are not present in the salivary fluids of the louse. Therefore, they cannot be transmitted directly when the louse bites another person. Instead, they are transmitted when the louse is crushed near the bite wound, releasing its body fluids on the bite site**.**

For Tick-borne relapsing fever, the tick picks the bacteria when it sucks blood from an infected person or rodent. The spirochaeta will multiply in the tick's body and within a week will be present in the saliva of the tick and ready for transmission to a new host during a blood meal.

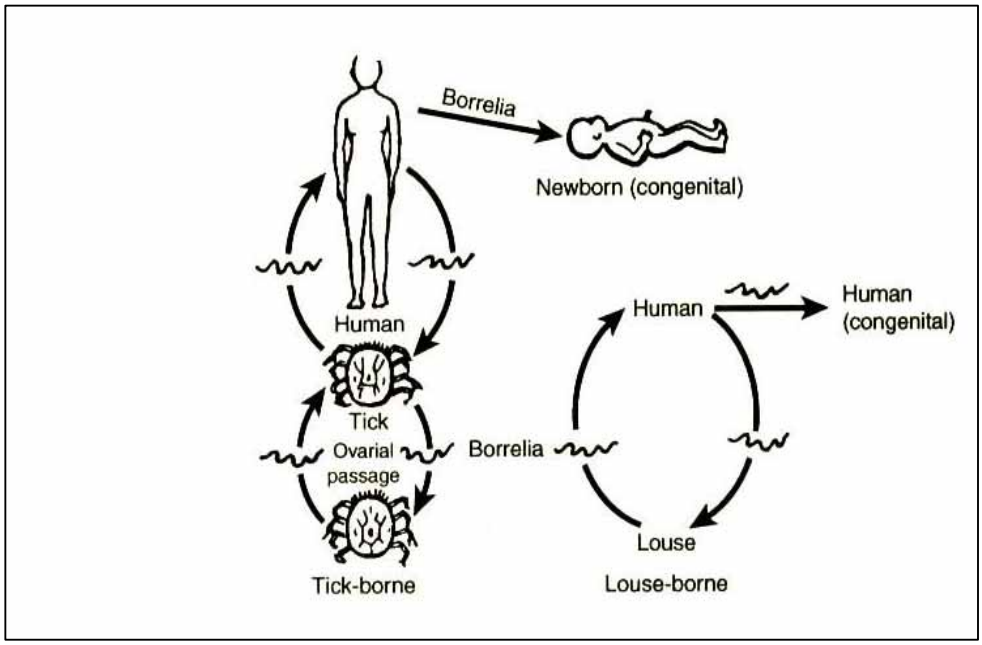



*Figure11: Life cycle of the spirochaeta Borrelia*

An interesting feature in tick-borne relapsing fever is that the spirochaeta are transmitted to the ovary of an infected tick, and therefore, an infected tick spreads it to its newborn.

## *Clinical Presentation*

Once an individual is infected, the spirochaeta multiply within the body fluids and produce end toxins that affect the liver, the spleen and the endothelial cells of the blood vessels.

The person mainly presents with a high grade fever 2-10 days after the initial contact. The fever is also accompanied by chills, headache, joint pains, vomiting and jaundice.

Five to ten days after the onset of fever, the temperature comes down. In two thirds of the patients the fever relapses 5 -7 days after the initial attack and will again subside after a few days. A second relapse may occur in a quarter of the patients. Relapses may be as many as ten times in untreated persons and are more common in the tick-borne variety then the louse-borne.

Most adults may develop immunity after a few relapses and the disease may resolve. However, the disease is usually severe on children, pregnant women and people with immunity disorders. In such cases complications easily occur, leading to death.

*Take Note*

In pregnant women the spirochaeta cross the placenta and may result in miscarriage, stillbirths and congenital infection of the newborn.

## *Diagnosis*

The clinical picture may be similar to that of malaria and yellow fever. However, diagnosis can be reached through microscopy of a blood film and other laboratory investigations.

## *Management*

The drug of choice has been Tetracycline 250 mg taken orally four times a day. Other drugs that have been used are Potentate antibiotics E.G Augmentin. Penicillins have also been widely used, given as injections for severe cases.

Take Note

Severe reactions (Jarisch Herxheimer reaction) to medication have been observed resulting to sudden deaths. This reaction comes after the antibiotics kill large numbers of spirochaeta, resulting into release of toxins into the circulation.

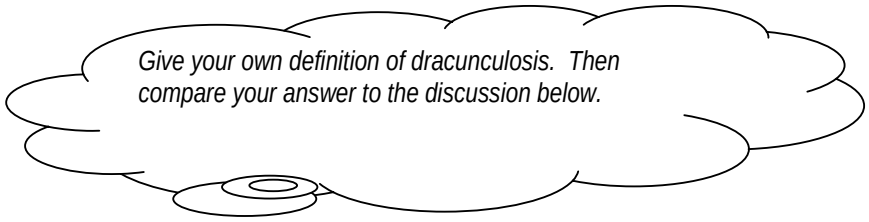
The reaction is characterized by rapid breathing, chills and a cardiovascular collapse. Such patients should be nursed flat and given adequate fluids.

## *Prevention and Control*

Educate the community to implement the following measures for the prevention and control of relapsing fever:

- Improve housing conditions and keep houses clean (especially for TBRF)
- Use insecticides to disinfect crowded areas
- Wash regularly and keep clean clothes (especially for LBRF)
- Reduce overcrowding in prisons, refugee camps and slums.

# Section 8: Dracunculosis (Guinea Worm)




Dracunculus is a tropical worm commonly known as Guinea worm common in Tropical Africa where the water and sanitation status are poor. It is transmitted by a small crustacean called Cyclops. Dracunculosis occurs in Uganda, Sudan and in West Africa (Ghana, Cameroon and Nigeria, especially). It is often seen in localized tropical areas with poor water supply. For example, in Uganda it is mostly found in villages in the North of the country.

## *Life Cycle of the Dracunculus*

Humans become infected by drinking water containing *Cyclops* crustaceans which are infected with *dracunculus medinensis.* The *Dracunculus medinensis* larvae get into the subcutaneous, inter-muscular and connective tissues of humans where they slowly develop. The whole cycle takes about one year. After mating the male dies and the female, about 1 meter in length, moves to the surface of the body and on contact with water discharges new larvae usually through an ulcer on the leg. As they enter the water, the newly hatched larvae will infect the Cyclops again. After two weeks, the larvae which have developed in the Cyclops, will require a temperature of about 25$^{o}$C and will be discharged in the water from where they will again get into the human system.

In some areas, transmission may be greater in the dry season when many people visit a few pools, while in other areas it may be greatest at the start of the rains.

## *Clinical Presentation*

An itching and painful blister develops on the site where the adult female worm emerges, thus making an ulcer. Ulcers can occur anywhere, but they are most common in body areas which are most likely to come into contact with water such as feet, legs and genitals. The thread like warm can be seen in the ulcer.

The complications mainly include dermatitis, continuous wounds that don't heal easily and tetanus due to the wounds. Most of the times there are multiple infections with more than one worm growing.

## *Diagnosis*

Diagnosis of dracunculosis is mainly through the clinical presentation of the patients in the endemic zones. Most of the patients will come with the ulcers as the adult female worms try to lay the eggs.

### *Management*

The worm is normally wound around a stick and teased to come out, but the worm can break causing a severe reaction. Drugs such as Nitronidazole, Metronidazole and Thiabendazole have been tried, but they probably only help to reduce inflammation.

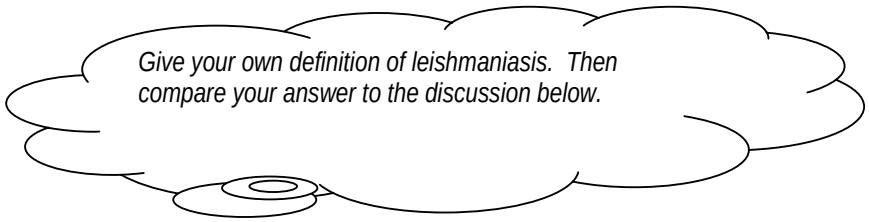
### *Prevention and Control*

This is a condition that is easily eradicated by improving the water and sanitation state of the community. Train the community to Avoid drinking surface water from pools, swamps and dams. Drilling of boreholes and community education is really helpful.

In Uganda, one of the more effective measures was the drilling of boreholes and the provision of pumps so that people did not have to use small open pools. However, in countries where there is war, civil unrest and population movements, the condition increases in frequency and may even produce epidemics because there are no organized control programmes.

Further action can be to boil water or filter drinking water through a cloth. By far the most important measures are to educate the population, improve water supplies and stop people with ulcers from standing in public water sources.

## Section 9: Leishmaniasis




Leishmaniais is an infection caused by a parasite mainly affecting rodents and canines. It is mainly transmitted from animals by sand flies or the Phlebotomus fly. It is common in Africa where it manifests in two forms: visceral and cutaneous.

### *Visceral Leishmaniasis*

Visceral leishmaniasis is also known as kala-azar. Kala-azar is a disease found in many parts of the world. In Africa, it occurs on the Mediterranean coast and in a belt across the arid and semi-arid areas from Lake Chad in the west to Somalia in the east, excluding the highlands of Ethiopia. The two main foci of the disease are Sudan and Eastern Africa. The main endemic area is in eastern Kenya along the Tana and Athi rivers. In the Sudan, the disease is found in the flood plains of the Nile and its tributaries. In the early 1990s it caused a large epidemic and thousands of deaths. Without treatment the disease is usually fatal because of intercurrent infections and general wasting.

## Life Cycle of Visceral Leishmaniasis

Visceral leishmaniasis is a zoonosis which circulates among wild and peri-domestic animals and sand flies and only occasionally affects humans. The animals most involved are dogs and rodents such as rats. The parasite which causes visceral leishmaniasis is called *L. donovani.* It is spread by sandflies of the species *Phlebotomus orientalis* in the Sudan and *P. martini* in Kenya. In the Sudan, the rat is the zoonotic host, while dogs, rodents and lizards may form the reservoir of the disease in Kenya. The disease mainly affects male teenagers and may affect special groups such as soldiers and hunters who enter endemic areas.

## Clinical Presentation of Visceral Leishmaniasis

The incubation period of visceral leishmaniasis is 4-10 months or longer. Often, before the definitive symptoms begin, a papular skin lesion known as a *leishmanioma* appears. This may precede the onset of the disease by *4-6* months.

The disease may set in gradually or suddenly. If the onset is sudden, it presents with intermittent or remittent fever which lasts for *2-6* weeks before becoming persistent and low grade. If the onset is gradual, it starts with discomfort in the left hypochondria as the spleen enlarges. During this time the patient may present with acute intercurrent infections such as dysentery or pneumonia. The disease then runs a chronic course lasting 1-2 years and is associated with chronic fever, hepatosplenomegaly, lymphadenopathy, anaemia and emaciation. In *96* per cent of cases, the patient dies of intercurrent infection, if not treated.

## Diagnosis of Visceral Leishmaniasis

Diagnosis is by finding the parasites in stained smears from splenic puncture or by immunological tests such as the formal gel test, leishmanin test and complement fixation test. In endemic areas, exclude kala-azar in patients who present with wasting, fever, anaemia and enlarged spleen and liver. Transfer to hospital for definitive diagnosis and treatment.

## Management of Visceral Leishmaniasis

The drug of choice is Sodium Stibogluconate intravenously or intramuscularly, in doses of 0.1-0.2 ml/kg (1 ml = 100 mg) daily for 6-30 days. Alternately, Pentamidine Isothianate may be given intramuscularly in doses of 0.1 ml/kg (1 ml = 40 mg) three times weekly until ten doses have been administered. Amphotericin B is reserved for cases resistant to other drugs. It is given by slow intravenous infusion in 5% dextrose in doses of 1 mg/kg on alternate days until a total of 2 g has been given.

Exacerbation of kala azar by HIV infection and AIDS is now causing some concern. The two diseases also look alike. Supportive treatment is to ensure bed rest, provide good nursing, supply adequate nutrition, correct anaemia and treat intercurrent infections.

## Prevention and Control of Visceral Leishmaniasis

Although sandflies are susceptible to insecticides, control of the vector is difficult because the flies breed away from homes. However, in Baringo District of Kenya some measure of control has been achieved with the use of fine insecticide-treated

curtains in homes. Destruction of infected dogs and rodents will control the reservoir.

Early diagnosis and treatment can also control the spread of the disease from human to human. Educate the population about the disease and the need for early treatment.

## *Cutaneous Leishmaniasis*

Cutaneous leishmaniasis is also known as Oriental sore. This is a zoonotic infection characterized by a single or several chronic cutaneous sores which usually heal spontaneously. The disease affects parts of the Sahelian region of West Africa where it is caused by *Leishmania tropica major.* Human cases are uncommon and sporadic. The zoonotic reservoirs are desert gerbils and dogs and the vectors are *Phlebotomus papatsi* and *P. sergenti.* In eastern Africa, the Ethiopian and Kenyan highlands are affected by the disease caused by *L.aethiopia.* The zoonotic reservoir is the rock hyrax and the vectors are the high-altitude sandflies *P. longipes* and *P. pedifer.*

### Life cycle of Cutaneous Leishmaniasis

Cutaneous leishmaniasis is a rare disease of which there are three main forms caused by different species and trains of Leishmania. The rural form of the disease affects humans in uninhabited areas and in villages situated on the edge of the desert. In these cases, the disease is caused by *L. tropica major.* The desert gerbil is the main reservoir and is transmitted by *P. papatsi.* The urban form of cutaneous leishmaniasis is caused by *L. tropica minor* and is an infection of humans and dogs in big cities and towns. It is transmitted by *P. sergenti.* The third form of the disease is that found in the Ethiopian and Kenyan highlands. It is an infection of humans and rock hyraxes caused by *L. aethiopia.* The vector *P. longipes* bites people in their houses at night.

### Clinical Presentation of Cutaneous lLishmaniasis

The incubation period is 2-8 weeks. The disease starts as a small itchy papule, usually on the face, which expands and grows over several weeks to form a single indolent ulcer or multiple ulcers. The ulcer(s) may resemble skin tuberculosis of the face (lupus vulgaris). There may be local lymphadenopathy. Rarely, when the lesion infiltrates the skin diffusely, it may resemble lepromatous leprosy. Spontaneous healing starts 2-12 months afterwards. The disease does not spread to involve the viscera.

### Diagnosis of Cutaneous Leishmaniasis

Diagnosis is mainly based on clinical presentation and lab analysis of the fluids from the skin eruptions.

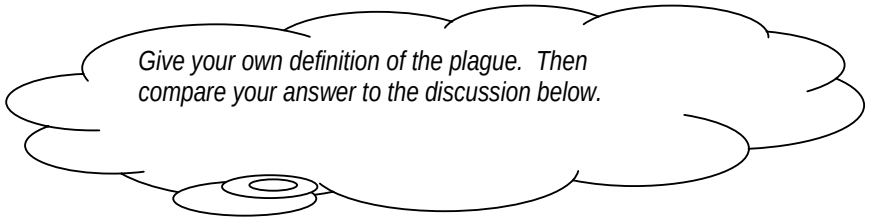
### Management of Cutaneous Leishmaniasis

Treatment is usually not necessary as spontaneous healing occurs. If the lesions are diffuse or extensive, use Pentavalent Antimonials such as Sodium Stibo-Gluconate as in the treatment of visceral leishmaniasis. The response is, however, not as satisfactory. Surgical treatment may be considered in some cases.

### Prevention and control of Cutaneous Leishmaniasis

Prevention and control are as for visceral leishmaniasis. Exclude Tuberculosis and leprosy in patients with cutaneous leishmaniasis. Refer to hospital for diagnosis and treatment. Also institute control measures.

## Section 10: The Plague




The plague is a bacterial infection that is highly infectious. It was of great public heath concern in the mid-nineteenth century. It occurs in epidemics that may claim a large number of people within a short time. It is spread by fleas that live on rodents. It is uncommon this days but East Africa continues to be one of the few remaining foci of the plague in the world. Small outbreaks have occurred in Uganda, Kenya, Tanzania, the Democratic Republic of Congo (Zaire) and Namibia.

## *Life cycle of the Plague*

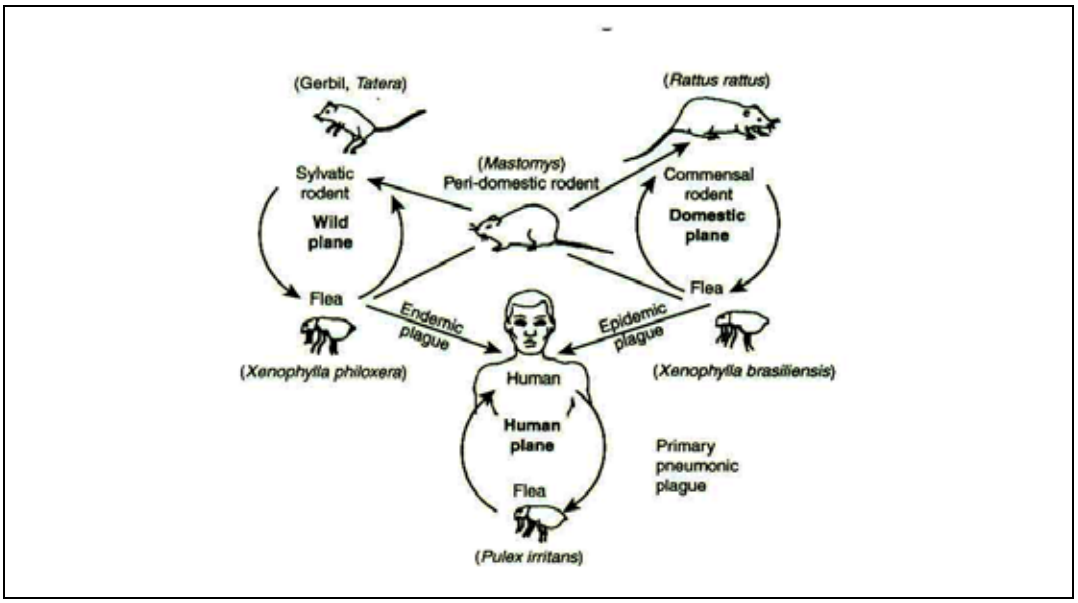



*Figure 12: The plague cycle*

The plague is caused by a Gram-negative bacillus called *Yesinia pestis* (formerly *pasteurella pestis*). The disease is endemic in wild rodents living especially in the highlands. It is spread by fleas from the rats. In the past, great epidemics of the plague were caused when wild rats died from the disease and their fleas looked for substitute domestic rat hosts. When the domestic rats also died, the fleas started to bite humans (Figure 9). When the first human is infected, the disease causes bubonic plague. By coughing infective plague bacilli, the person infects relatives and suddenly many people die from an undiagnosed disease.

## *Clinical Presentations*

There are three clinical presentations of the plague: Bubonic plague, Septicaemic plague and Pneumonic plague.

### Bubonic Plague

Bubonic plague is the commonest form of the plague. It is characterised by swelling of the lymph glands (buboes) which appear within 24 hours of the onset of the disease. The glands most affected are those of the groin, although the armpit and other places may also be affected. The buboes vary in size and may be tender or non-tender. Bubonic plague may be complicated by septicaemia.

### Septicaemic Plague

Septicaemic plague may be due to an initial infection with highly virulent organisms, or it may be a terminal complication of the bubonic plague. When it is an initial infection, glandular involvement is minimal. The illness is of sudden onset. The patient is prostrated, febrile, weak, pale and apathetic. Stupor, coma and death may follow on the first, second or third day, or later. In the septicemia stage the bacilli are everywhere in the organs, including the lungs. The patient may cough and spread the bacilli to attendants who then develop the pneumonic type of plague.

### Pneumonic Plague

Pneumonic plague usually follows or complicates the other two forms of plague. Attendants and visitors are at especially high risk of infection because of the numerous bacilli the patient coughs out. The illness is characterised by sudden onset of rigors, malaise, intense headache, body aches, fever, and severe prostration. This form of plague is rare in the tropics.

In the early stages it may be difficult to diagnose pneumonic plague, but the extreme illness of the patient in the absence of definite physical signs should raise the suspicion of this type of plague. At first the sputum is watery, but soon it becomes blood stained. Pleural effusion may form, and death usually occurs within 1-2 days. This is the most infectious and the most fatal form of the plague.

A few people who contract bubonic plague may not appear to be very sick. The fever comes down when the bubo bursts and starts discharging pus. The lesion can be differentiated from lympho-granuloma venereum by making a smear of the pus.

## *Diagnosis*

Diagnosis can be made from the presence of vast numbers of Gram-negative bacilli in sputum or pus from lymph nodes. The bacilli are short and thick with rounded ends; the extremities stain a deeper colour giving a bipolar appearance.

## *Management*

Use of antibiotics has drastically reduced the incidence of the plague as the bacilli are very sensitive to most antibiotics except Penicillin. Early treatment with Streptomycin 1 g 12-hourly for 3 days and then 1 g daily for 7 days, or Tetracycline or Chloramphenicol 500mg four times a day by mouth for 7 days are all effective. Cotrimoxazole is also effective in doses of 2 tablets twice a day for 7 days.

## *Prevention and Control*

Early diagnosis is most important. In cases of suspected plague, patients should not be referred to the hospital but the District Public Health Officer (DPHO) must be informed immediately.

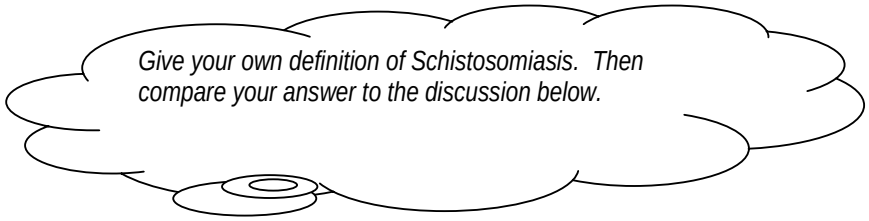
The plague is an international notifiable disease and should be reported to the World Health Organization (WHO).
Chemoprophylaxis of all contacts must be started forthwith (Tetracycline or Cotrimoxazole) if there is no plague pneumonia. The area where the disease occurs must be quarantined (isolated from all neighbouring places). The police can help with this. Cases must be isolated and contacts quarantined for 10 days.
Insecticides should be distributed to kill fleas. Encourage people to kill rats and deal with the rubbish which attracts them. Vaccination during an epidemic is not effective.

It is hoped that you will never see a case of the plague, but remember that most probably the initial recognition of the disease will be by health staff working in small units, so your quick action may save the lives of many people.

If you suspect a case of plague, do the following:

- Notify your District Medical Officer (DMO);
- Ask the officer to come immediately;
- Plan to inform the public and tell them of their role in controlling the outbreak;
- Treat the patient with antibiotics;
- Give prophylaxis to all contacts (including yourself);
- Request supplies of insecticide and rat poison such as Warfarin.

# Section 11: Schistosomiasis




Schistosomiasis is a chronic disease caused by trematodes of the genus *Schistosoma* which infect the large bowel or the urinary bladder, depending on the species. The clinical disease is caused by the body's reaction to the presence of the eggs of these worms and will therefore depend on the location of the adult worms. The disease is commonly known as bilharzia.
Schistosomiasis is the second most frequent disease, after malaria, in some countries. In Egypt, 37 per cent or more of the population may be infected. The infection, however, even though it generally causes symptoms such as haematuria, does not necessarily lead to a disabling clinical disease.

This depends on the worms load, duration of infection, immune state of the patient and the presence of other concurrent disease. Anaemia, which may be caused by schistosomiasis, certainly causes some morbidity in children and lack of productivity in adults. Although schistosomiasis can result in killing conditions, such as portal hypertension, uremia and heart failure, it is not known how often these occur. There is, therefore, no general agreement on the seriousness of the ill effects of schistosomiasis on the general health of the people.

The incidence of schistosomiasis is related to water use. Development and water projects for irrigation or electricity provide the habitat for the snail vectors. Such projects may cause epidemics schistosomiasis. The rise of economic levels with improved agricultural techniques has often been accompanied by an increased incidence of schistosomiasis.

## *Life cycle of Shistosoma*

Schistosomiasis is caused by tissue reaction against the eggs of the schistosome worm. Depending on the species, the eggs are excreted in urine or faeces. The main schistosomes which infect humans in Africa are Schistosoma mansoni haematobium.

When the eggs reach water, they are hatched and the miracidia emerge. Miracidia are free-swimming larvae which have to reach a snail host within 24 hours or die. In the snail, the maracidia develop and multiply into many cercariae. These are the infective agents of schistomisis. They are shed from the snail in 1-7 weeks. In this condition they can only live up to 48 hours unless they enter a human body. A human becomes infected when entering cercaries-infested water. This can occur while bathing, swimming, laundering, cultivating or fishing. The cercariea penetrate the skin and enter the blood stream where they are carried to the liver or the bladder where they develop into adult worms. See Figure 13 for an illustration of the life cycle.

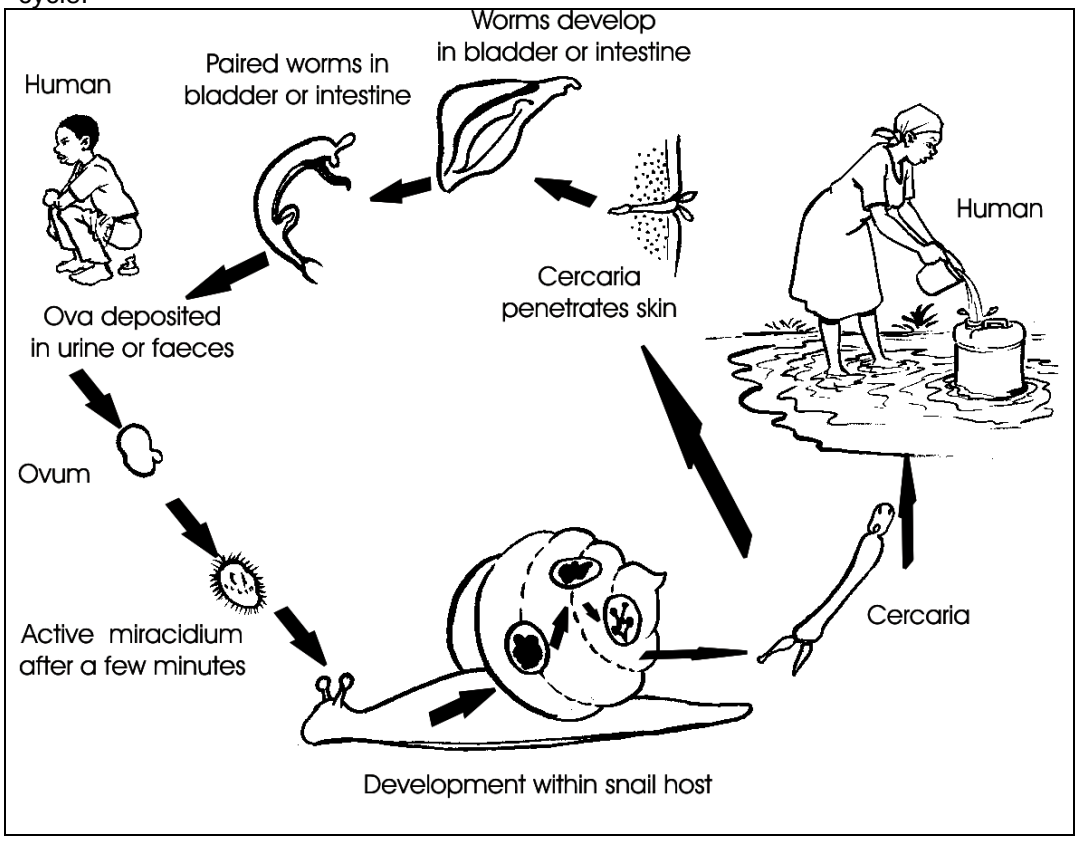



*Figure 13: The life cycle of schistosoma*

Two types of schistosomiasis are found in Africa, each with a different clinical picture. Each type of schistosomiasis has a different distribution depending on the type of snail vector and snail-host behavior. Schistosoma haematobium lives in the venous plexus of the urinary bladder. The eggs are excreted in the urine. The vector snails belong to the genus *Bulinus* which lives in temporary water bodies such as ponds, dams and paddy fields. It adapts to adverse conditions by inactivity (aestivation) during the dry season. *Schistosoma mansoni* lives in the mesenteric plexus of the large intestine. The eggs are excreted with feaces. The vector snails belong to the genus *Biomphalaria* which prefers permanent water in streams, irrigation schemes and lakes.

## *Clinical Presentation*

Schistosomiasis as a disease has several stages: Invasion, maturation, established infection and late stage. We shall discuss each one of theses stages in some detail.

### Invasion

At the invasion stage the *cercariae* penetrate the skin. This causes cercarial dermatitis with itching papules and local oedema. Very often, however, this is not noticed or not reported. The cercariae enter the circulation and reach the liver via the right side of the heart and the lungs by an unidentified mechanism.

### Maturation

The *schistosomes* mature in the liver. This stage is associated with fever, eosinophilia, abdominal pain and transient generalized urticaria. It is known as the ***Katayama syndrome***. After maturation, the adult worms descend to the portal vein. *S. mansoni* migrates to the mesenteric veins in the interstinal wall, and *S haematobium* finds its way to the bladder venous plexus. This stage often passes largely unnoticed.

### Established Infection

This stage is characterized mainly by the production of eggs by the mature females. The eggs penetrate tissues with the help of their spines but some fail to reach the lumen of the bowel or bladder, hence provoking an inflammatory reaction and the formation of granulomas. It is this inflammatory reaction that causes the early signs and symptoms of schistosomiasis: signs of colitis with bloody diarrhoea and cramps in *S. mansoni* infection: terminal haematuria and dysuria in *S. haematobium* infection.

### Late stage

This stage comes with such complications as:

- Obstruction to and dilation of the ureters (hydro-ureter) and kidney (hydronephrosis), possibly leading to kidney failure;
- Pyelonephritis;
- Calcification of the bladder which shows on X-ray or ultrasound investigation;
- Cancer of the bladder (seen in Egypt and Mozambique);
- Liver complications;
- Congestive cardiac failure (CCF).

## *Diagnosis*

The diagnosis of schistomiasis is confirmed by finding eggs in stools or urine. If these are repeatedly negative, a biopsy can be done (rectal snip, bladder biopsy) Concentration techniques can be used to improve the detection rate of the eggs in stool. Examination can sometimes be misleading.

## *Management*

Treatment of schistosomiasis has drastically changed with the use of drugs that are easy to use and are less toxic to humans. However, community involvement is vital in the management of the disease. Community based interventions should be adopted in the disease control.

When there is a high prevalence of schistosomiasis, treat all those with symptoms. If there are limited resources, give frequent health education on how the disease is spread, stress the importance of a safer water supply and emphasize the need for building latrines. Table 3 summarizes the drugs available for management of shisosomiasis.

Table 3: Drugs used in the treatment of schistosomiasis.

| Species | Drug | Dosage | Side effects | Contraindi-cations |
|---|---|---|---|---|
| S. *haematobium* | Metrifonate | 7.5 mg/kg body weight once every 2 weeks total of 3 doses | None | None |
| | praziquantal | 40mg/ kg body weight. Stat dose. | Mild drowsiness. | None |
| *S. mansoni* | Oxamniquine | 15-30 mg/ kg body weight | Drowsiness, Dizziness occasionally psychosis | Epilepsy |
| | Praziquantal | 40 mg/ kg body weight once | Mild dizziness | None |

## *Prevention and Control*

Use of pit latrines is an effective method of control. The use of pit latrines is very important not only in the prevention of schistomiasis but of other diseases as well.

Snails could also be brought under control by using Molluscicides. However, the method is sometimes expensive and toxic to the environment. Copper sulphate is the best chemical for this so far.

Environmental sanitation is another control measure. Draining or filling water bodies and clearing the vegetation in water bodies to deprive the snails of food and nesting places will have excellent results. Snail control through environmental sanitation or Molluscicides needs maintenance over a long period (at least 10years). It is almost impossible to continually use either of these methods in most of Africa because of prevailing economic conditions.

Community health education is the most effective way to protect humans from schistosomiasis. The community should be educated about the disease and given ample supply of safe water.

Water can be made safe by keeping it in a container for 48hours using the three-pot system. Within this period the cercaria will die.

**SUMMARY**

You have now come to the end of this Unit. I hope you enjoyed it well enough to learn all that it contains. Please go back to the objectives and see if you have achieved them.

If you have achieved the objectives, you are then ready to do your tutor marked assignment. Good luck! ☺

**DIRECTORATE OF LEARNING SYSTEMS**
**DISTANCE EDUCATION COURSES**

Student Number: _____

Name: _____

Address: _____
_____


# COMMUNICABLE DISEASES COURSE

## Tutor Marked Assignment

## Unit 8: Vector Borne Diseases

1) Define a reservoir host.

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2) What is a vector?

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3) State three factors that affect the geographical distribution of trypanosomiasis.----
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4) Match the vector species in Part A with the diseases in part B by writing the letter corresponding to the correct disease next to the vector species;

| Part A | | Part B |
|---|---|---|
| *I. Simulium narvae* | ---------- | a) Trypanosomiasis |
| *II. Aedes ssp.* | ---------- | b) Onchocarciasis |
| *iii. Glossina morsatans* | --------- | c) Relapsing fever. |
| *iv. Orthodurus moubata* | ---------- | d)Yellow fever. |

*5)* Using a sketch diagram briefly describe the life cycle of *Wochereria banchrofti.*

6) Mr. John Makuac lives in Upper Nile in South Sudan. Recently he noted an ulcer on his left ankle joint which has not healed despite the use of antibiotics. When he examined it carefully he noted a white thread protruding from the ulcer.

a) Which disease is John suffering from?----------
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b) What advice would you give Mr. Makuac?
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c) How can he prevent other members of his family from getting similar infections?
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7) The government of Kenya is embarking on a new rice growing scheme in the coastal part of the country. Apart from Schistosomiasis, list three other vector-borne diseases that the residents of the area would be exposed to.

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8) Suppose you are hired by the Ministry of Health to draw up a schistosomiasis control strategy project.

a) Explain the recommendations that you would give to be included in the project.-

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b) How would you ensure that the measures recommended above are sustainable?

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9) Explain three ways through which tse tse flies population can be reduced.

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10) State two diseases that can be transmitted by rats.

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11) Global warming is a universal problem causing global climate change. Explain how this phenomenon is affecting the geographical distribution of vector-borne diseases.

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