Parasitic infections/ infestations of the musculoskeletal and integumentary system

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Learning objectives

By the end of this session students will be able to:

- Explain the causative agents, mode of transmission, life cycle, clinical manifestations, pathogenesis , laboratory diagnosis, treatment, prevention and control of medically important parasites of Musculoskeletal and integumentary system

Outline

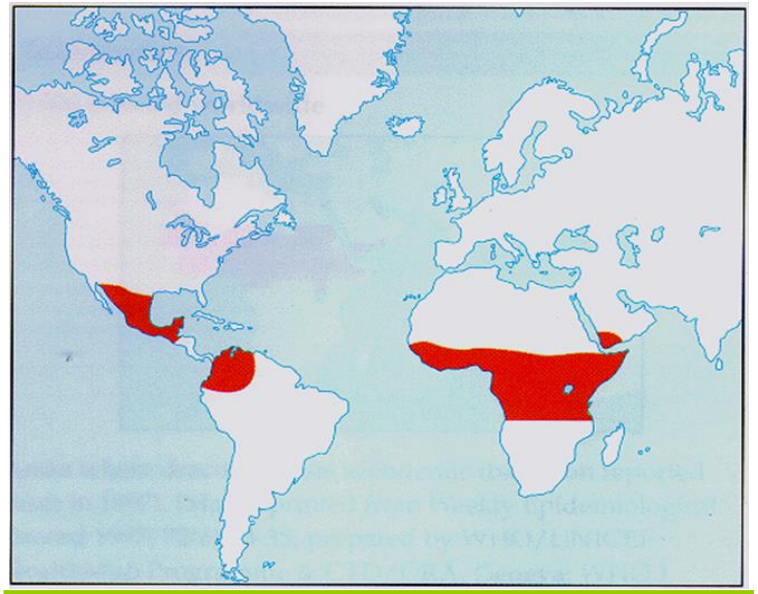
- ***Onchocerca volvolus:*** **Onchocerciasis**
- ***Dracunculus medinensis:*** **Dracunculosis**
- **Leishmaniasis (different *Leishmania* spps)**
  - **CL, DCL, MCL,PKDL**
- ***Sarcoptes scabiei*****: Scabies**
- Reading Assignement

Onchocerciasis

- Is a **filarial** disease caused by O. Volvulus
- Commonly known as **river blindness**
- The world's **second** leading infectious cause of **blindness**
- WHO estimates the global prevalence is 17.7 million, of whom about 270,000 are blind

Distribution:

- Occurs most widely along the courses of **fast running rivers** in the forests & Savannah areas of west and central Africa
- Also occurs in the Yemen, Arab Republic, Central and South America



Onchocerca volvulus

- Habitat:
  - Adult:
    - Subcutaneous nodules under the skin
    - Adults can live ~ 8 – 10 years in nodules
  - Microfilariae: live ~ 2 years
    - Skin, eye and other organs of the body
  - Infective larvae in:
    - Gut, mouth parts and muscles of **black fly**

Onchocerca volvulus

- ❖ Morphology
- ➢ Microfilariae:
  - 220 to 360 µm length
  - **No sheath**
  - **Anterior nuclei** are positioned side by side
  - **No nuclei** in the **end** of the tail
  - Tail is long and pointed

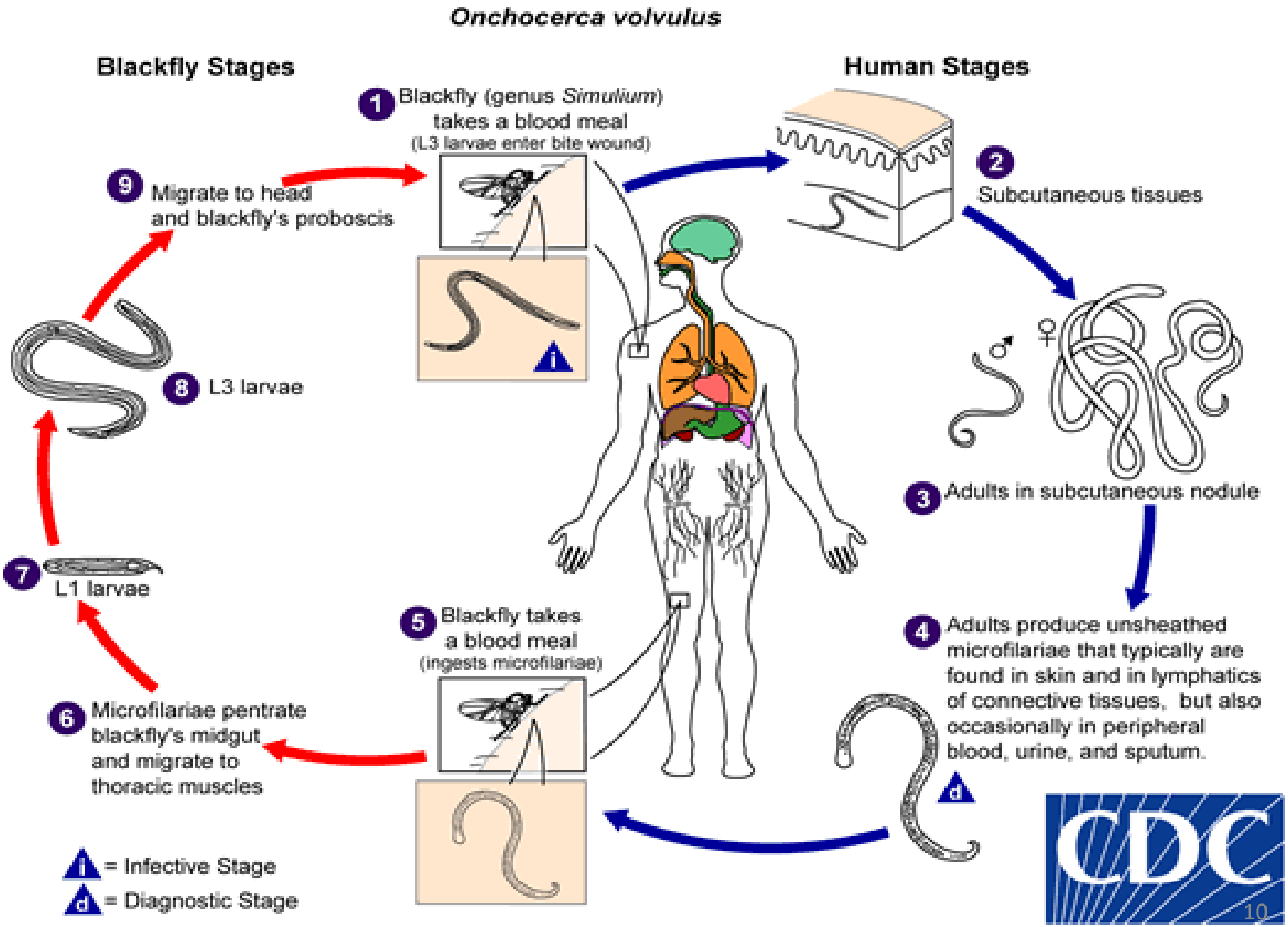
Onchocerca volvulus

- Adult:
  - Females - 33 to 50 cm in length
    - **Viviparous**: relase 1000-2000 Microfilaria (L1) per day
  - Males - 19 to 42 mm

Transmission

- Onchocerca is transmitted by the bite of infected **black flies (Simulium spec., especially S. damnosum)**
- **Only** the females bite
- Larvae and pupae are aquatic filter feeders, **living in fast flowing oxygen rich waters**

Life cycle of Onchocerca volvulus



Pathogenesis

- **Microfilariae**, **Adult worms** and ***Wolbachia*** bacteria of *O. volvulus* contribute to the pathogenesis, all through consequences **of host immune response.**
- **Adult worms** are the **least** pathogenic, usually causing **no** symptoms at all. At **worst**, stimulate the development of noticeable subcutaneous nodules called **Onchocercomas**.
- These nodules are usually situated over bony prominences.
- The presence of nodules **doesn't** cause **pain** or **ill** health to patients but does cause some disfigurement to the body.
- The formation of nodules is sometimes followed by **elephantiasis** (enlargement of body parts)

Pathogenesis…

The site of nodules vary according to geographical areas

- In most parts of Africa – commonly found in lower part of the body (coccyx, sacrum/iliac crest, hips)
- In Central America : nodules found in the upper part of the body (head, neck, shoulder)

Onchocercal nodule on the scalp of a Mexican patient.

Pathogenesis…

- **Microfilaria** migrate through the connective tissue especially the dermis of the skin and cause **most of the pathology**
- **Living** migrating microfilaria seem to cause **little** or **no inflammation**
- **Dead** microfilaria however stimulate potent inflammatory reactions
- **Treatment** can have therefore severe side effects, (mild with Ivermectin but can be pronounced with DEC **???** **)**

Pathogenesis…

- **Dead** juveniles in the skin result in a **severe dermatitis**.
  - The dermatitis is the result of inflammation due to the release of *Wolbachia* bacteria from dead juveniles.
- The inflammatory reaction causes progressive pathological changes of the skin
- The **first** symptoms of dermatitis are itching, bacterial infection, and abnormal pigmentation(skin rash) but **later** on followed by the loss of pigmentation (**leopard skin**), thickening and creation of skin cracks (lichenification or **lizard skin**).
- The last stage of dermatitis is exhibited by the **loss** of the **skin's elasticity**, which gives the patient the appearance of premature **aging**

Skin change due to onchocerciasis

Onchodermatitis.

leopard skin

lizard skin

Pathogenesis…

- Microfilariae can cause inflammation of regional glands: **Onchocercal lymphadenitis**
- Lymph glands become enlarged
- Surrounded by skin **lost elastic tissue**
- Lead to protruding lymph gland enfolded in pocket of skin
- Common in lymph gland around scrotum
- A condition known as **hanging groin**

Pathogenesis…

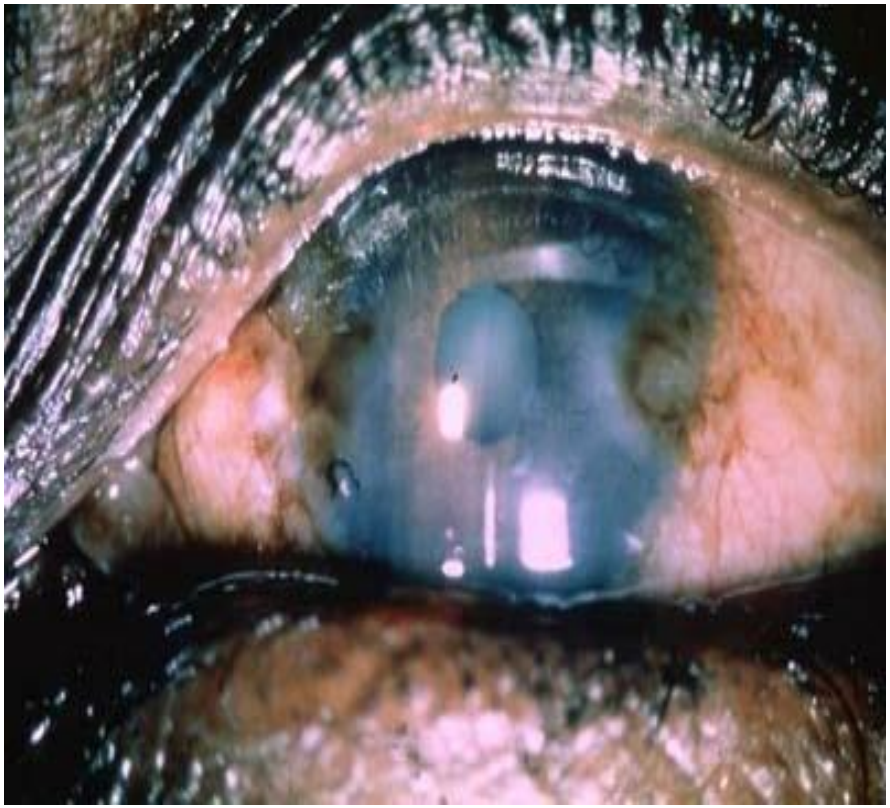
- Microfilariae cause **ocular lesion** can be due to both:
  - the trauma caused by migrating microfilariae
  - the host immune responses to **dead** microfilariae
- **Dead mf** induces the infiltration of: Eosinophils, Neutrophils
- Eosinophils and neutrophils would cover the dead worms
- Degranulation Eosinophils and Neutrophils, as reaction to worm antigens, interrupt fibril arrangement in cornea
- Result in the development of keratitis
- The most significant cause of blindness is sclerosing (scarring) keratitis characterized by the hardening inflammation of the cornea (**opacification**).

Pathogenesis…

The pathogenicity of the microfilariae:

- may differ according to the strain of the parasite. Example
  - a forest strain in Africa has low ocular pathogenicity
  - a humid savannah zone strain causes moderate disease
  - a dry savannah zone strain causes high pathogenesis and a high rate of blinds

Ocular lesion



Wolbachia bacteria

- Intracellular bacterium
- Endosymbiotic bacterium found in:
  - Lateral chords of female and male worms
  - Reproductive apparatus of females
  - Nematode larvae in the vector
- Wolbachia bacteria transmit transovarial (maternal transmission)
- **Wolbachia determines the fertility, viability and development of the filarial parasites**
  - antibiotic treatment affected worm growth, fertility
- Wolbachia facilitate metabolic activities of the worm
- **Protects** the filarial from hydrogen peroxide mediated damage
- Wolbachia associated molecules (WSP,LPS) induce: immune response

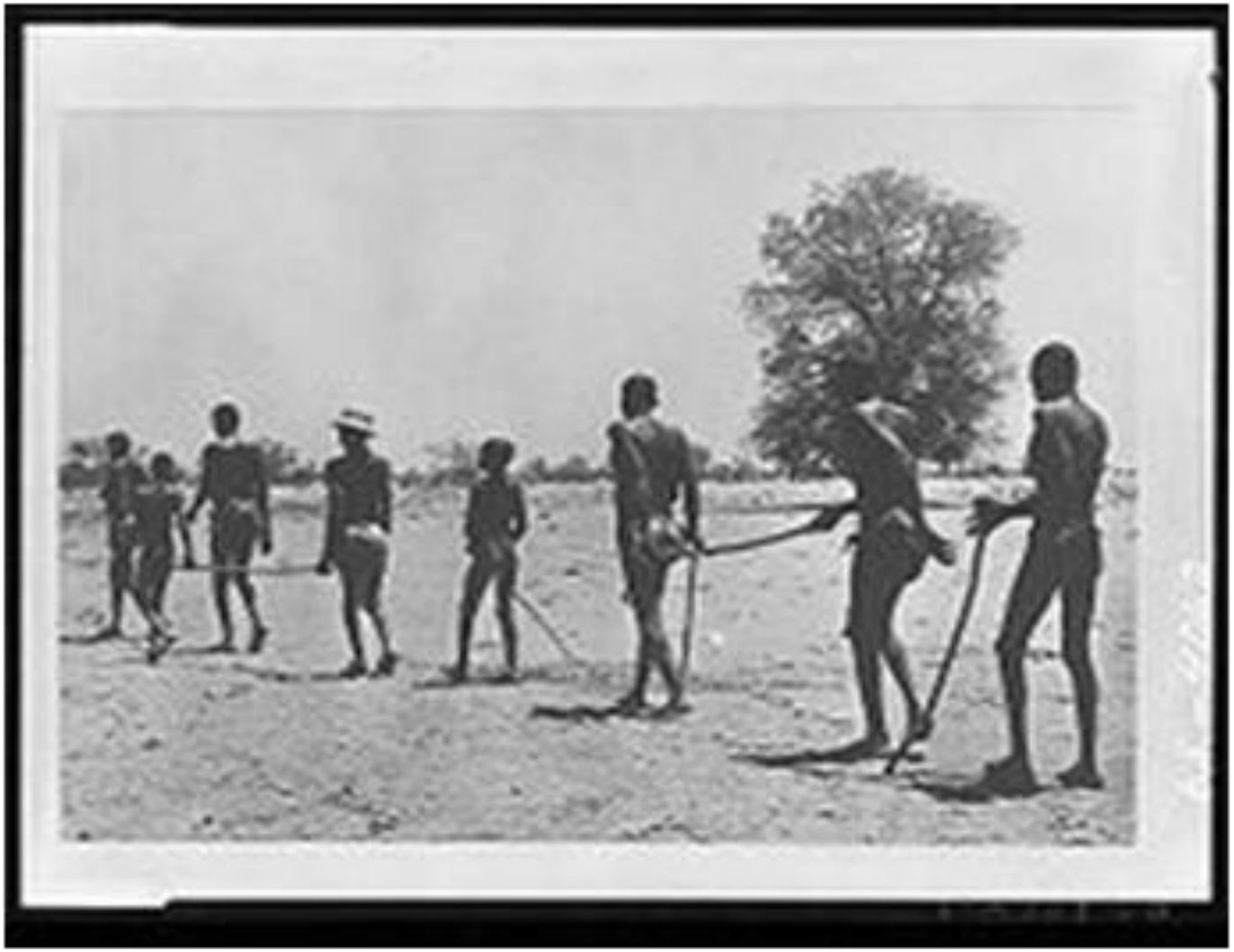
Signs and symptoms

- Skin involvement typically consists of intense itching, swelling, and inflammation.A grading system has developed to categorize the degree of skin involvement:
- Acute papular onchodermatitis - scattered pruritic papules;
- Chronic papular onchodermatitis - larger papule, resulting in hyperpigmentation;
- Lichenified onchodermatitis - hyperpigmented papules and plaques, with edema, lymphadenopathy, pruritus and common secondary bacterial infections;
- Skin atrophy - loss of elasticity, skin resembles tissue paper, 'lizard skin' appearance;
- Depigmentation - 'leopard skin' appearance, usually on anterior lower leg.

Signs and symptoms cont'd

- Ocular involvement provides the common name associated with onchocerciasis, river blindness and may involve any part of the eye from conjunctiva and cornea to uvea and posterior segment including retina and optic nerve.
- Punctate keratitis occurs in the infected area. This clears up as the inflammation subsides.
- However, if the infection is chronic, sclerosing keratitis can occur, making the affected area become opaque. Over time the entire cornea may become opaque, thus leading to blindness.

The burden of onchocerciasis: children leading blind adults in Africa.



CLASSIFICATION

Onchocerciasis may be divided into the following phases or types:

- Erisipela de la costa ;

An acute phase characterized by swelling of the face with erythema and itching. Onchocerciasis causes different kinds of skin changes and these changes vary in different geographic regions. This skin change, erisípela de la costa, of acute onchocerciasis is most commonly seen among victims in Central and South America.

- Mal morando ;

A cutaneous condition characterized by inflammation that is accompanied by hyperpigmentation.

- Sowda ;

A cutaneous condition, a localized type of onchocerciasis

CLASSIFICATION CONT'D

Additionally, the various skin changes associated with onchocerciasis may be described as follows:

- Leopard skin ;

A term referring to the spotted depigmentation of the skin that may occur with onchocerciasis.

- Elephant skin ;

A term used to describe the thickening of human skin that may be associated with onchocerciasis.

- Lizard skin ;

A term used to describe the thickened, wrinkled skin changes that may result with onchocerciasis.

Clinical feature

Onchocerciasis

- Acute onchocerciasis:
  - Itchy (pruritic)
  - Erythematous
  - Papular rash with thickening of the skin

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Clinical feature

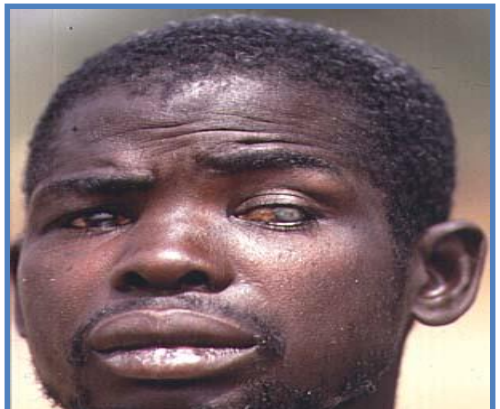
Chronic onchocerciasis:

– Elephant or lizard skin

Hanging groin

– Leopard skin

River blindness



Diagnosis

1. Demonstration of adult worm (Nodulectomy)
2. Skin snips
3. Slit lamp examination
4. Provocative test(Mazzotti test): single DEC (50mg)**???**
5. Serological
6. PCR

Diagnosis…

- **Skin snip**: demonstration of microfilaria in the cutis. A small piece of skin is cut and placed into saline. Microfilaria emerging from the sample can be observed microscopically
- Giemsa staining for differentiation from other species
- Mf in urine, blood & most body fluids (in heavy infection)

- Skin biopsy -Wet mount preparation -Giemsa staining

Diagnosis…

- **Nodulectomy**: adult worms can be removed surgically to reduce microfilarial load to alleviate symptoms
  - Demonstrate inside the excised nodule: females or males

- The nodules contain adult worms coiled together like a ball of string and are encapsulated by host-fibrous-tissue reaction.
- Typically, a nodule will contain **two** female worms and **one** male worm.

Treatment

- Ivermectin:
  - Paralysis of worms
  - Reduces the microfilarial load (does not kill macrofiliaria)
- Doxycycline: for wolbachia (four to six weeks)
- Surgical Care:
  - Nodulectomy: Removes adult worms

Prevention and control

- Destruction of Simulium (larvicides on target rivers or insecticide)
- Avoiding Simulium bites (Using insect replants)
- Treatment of communities (APOC)
  - MDA

Dracunculus medinenis
"Guinea worm, "

Dracunculiasis - Guinea worm disease
Dracunculus medinensis parasite

Dracunculosis

- Synonyms: Dracontiasis, Dracunculosis, Dracunculiasis
- Causative agent
  - Scientific name: **Dracunculus medinensis**
  - Common name: Medina worm or Guinea worm

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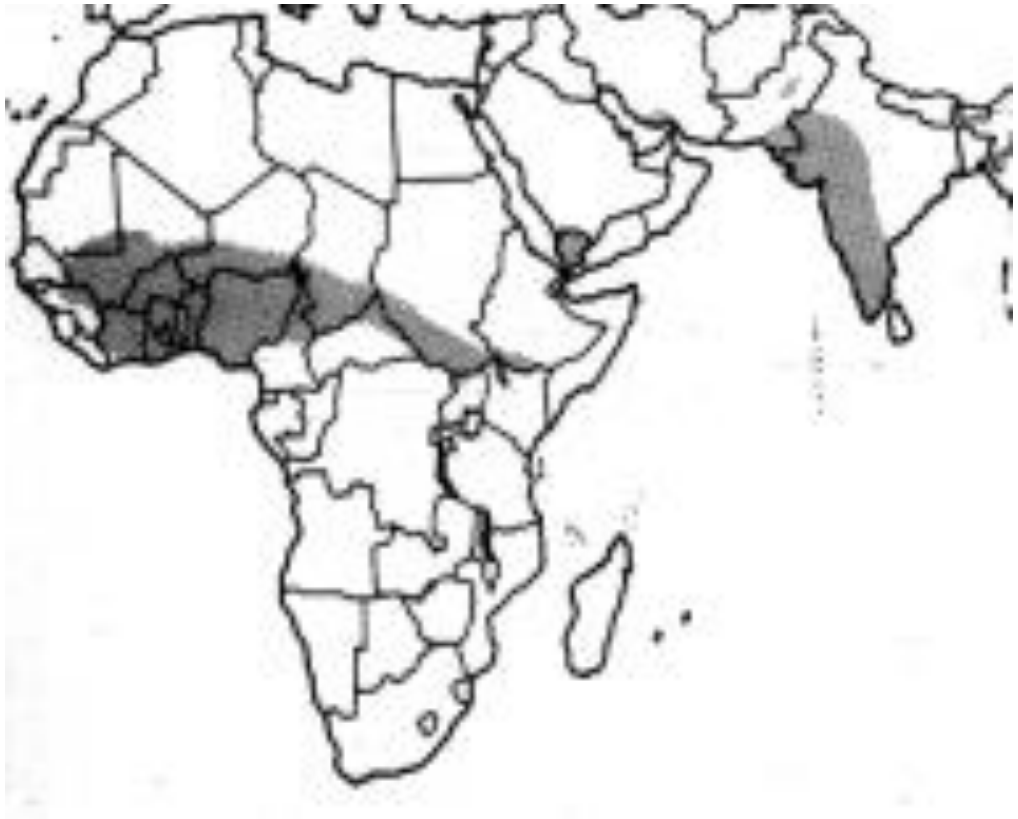
Epidemiology

- Most common in areas of limited water supply where individuals acquire water by **physically** entering water sources.
  - Walk-in **wells**
  - Water holes in parts of Africa

Distribution of *Dracunculus medinensis*



Global: Nile valley, India and areas where **wells** are used for water supply

Dracunculosis

- Habitat:
  - Adults in subcutaneous tissues of man/reservoir animals

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Morphology

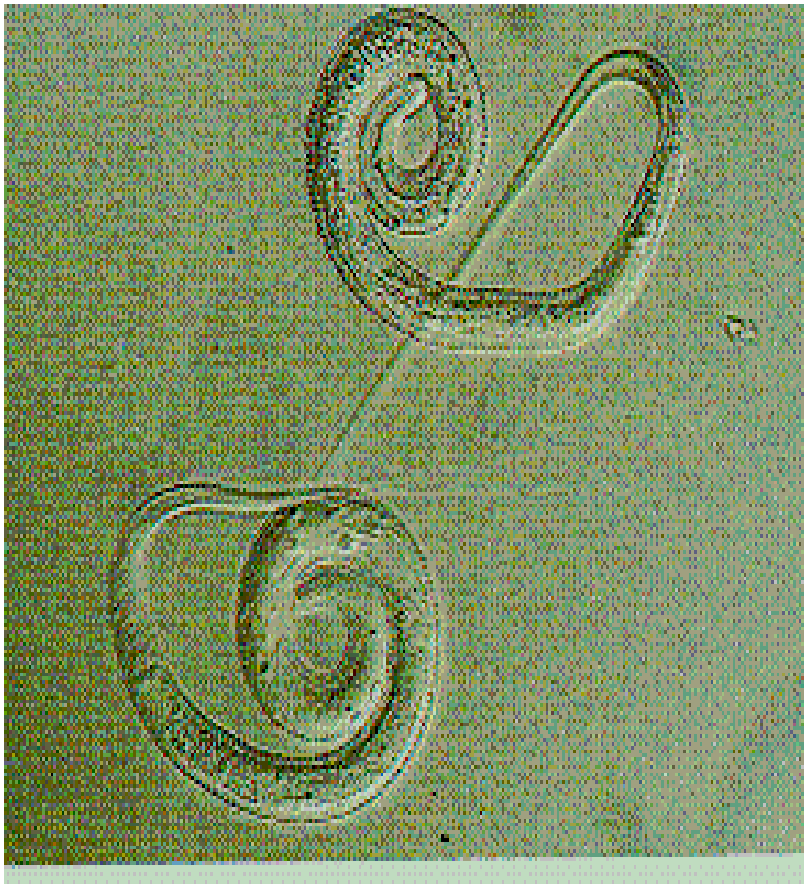
I. Adult: thread like, cylinderical oesophagus
II. Male: About 3 cm in length
    - Posterior end coiled
    - 2 unequal spicules

I. Female: About 30 to 100 cm in length
    - Swollen anterior end
    - Hooked posterior end
    - Inconspicuous vulva near anterior end

D. medinensis

2. Larva (or embryo):

- 600 x 20 μm
- Anterior end rounded
- Tapering and long tail (1/3 body)



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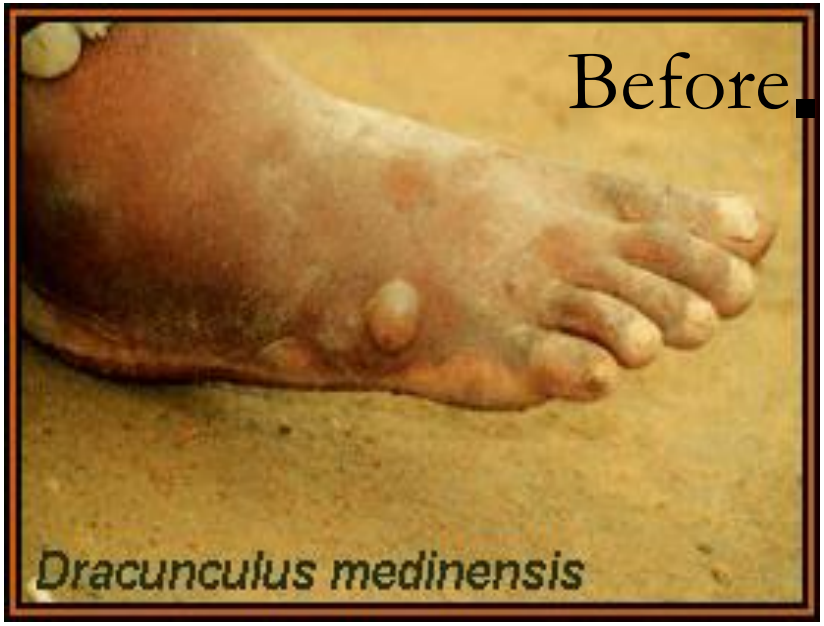
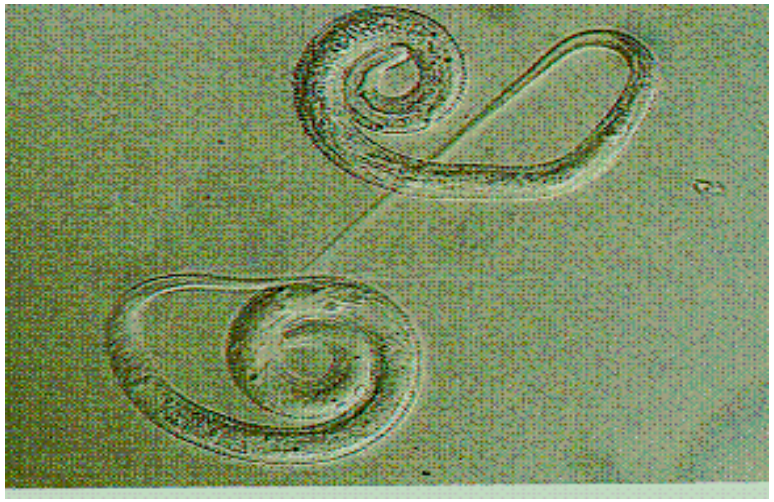
Life Cycle: *D medinensis*

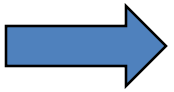
- A blister is formed from the female worm's production of embryos released under the skin, due to a **burning** pain that comes with this, the victims often immerses their legs in water for relief.
- With the **sudden drop** in temperature that follows, the blisters usually rupture, **releasing** the worms.
- These worms may release **thousands** of infective juveniles at this time, which enter the water.

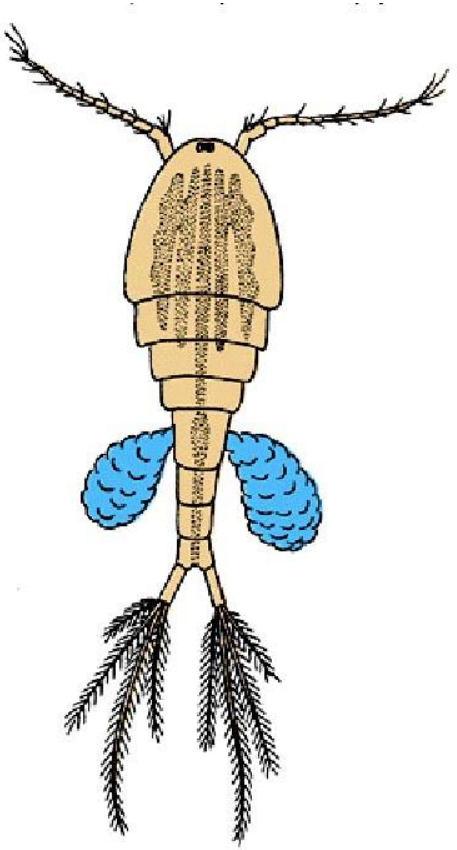


The cephalic end of the fertilized female pressing on the skin, produces a papule that becomes a blister and then ruptures forming an ulcer

Life Cycle of *Dracunculus medinensis*







Infective larvae(L1)

In water, larvae must be eaten by **Copepod** (Crustacean), the IH,

Life cycle...

- Man is infected on drinking water containing cyclops
- In the small intestine, the cyclops is digested , larvae liberated and penetrate through the duodenal wall and migrate to the subcutaneous tissues probably via lymphatics.
- At this point the females are fertilized by the males, and the males die. The females then migrate to the skin, reach sexual maturity, and produce juveniles.

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Life cycle…

- They tend to go to parts most likely to come in contact with water as the lower extremities
- Several months (9 or more) elapse between infection and appearance of the gravid female at the skin surface

Life cycle...

- The cephalic end of the fertilized female pressing on the skin, produces a papule that becomes a blister and then ruptures forming an ulcer
- When the ulcer contacts with water, a loop of the uterus prolapses through a rupture in the anterior end of the worm and larvae are discharged.
- larvae penetrate the intestine and settle in the body cavity to become infective in about 3 weeks

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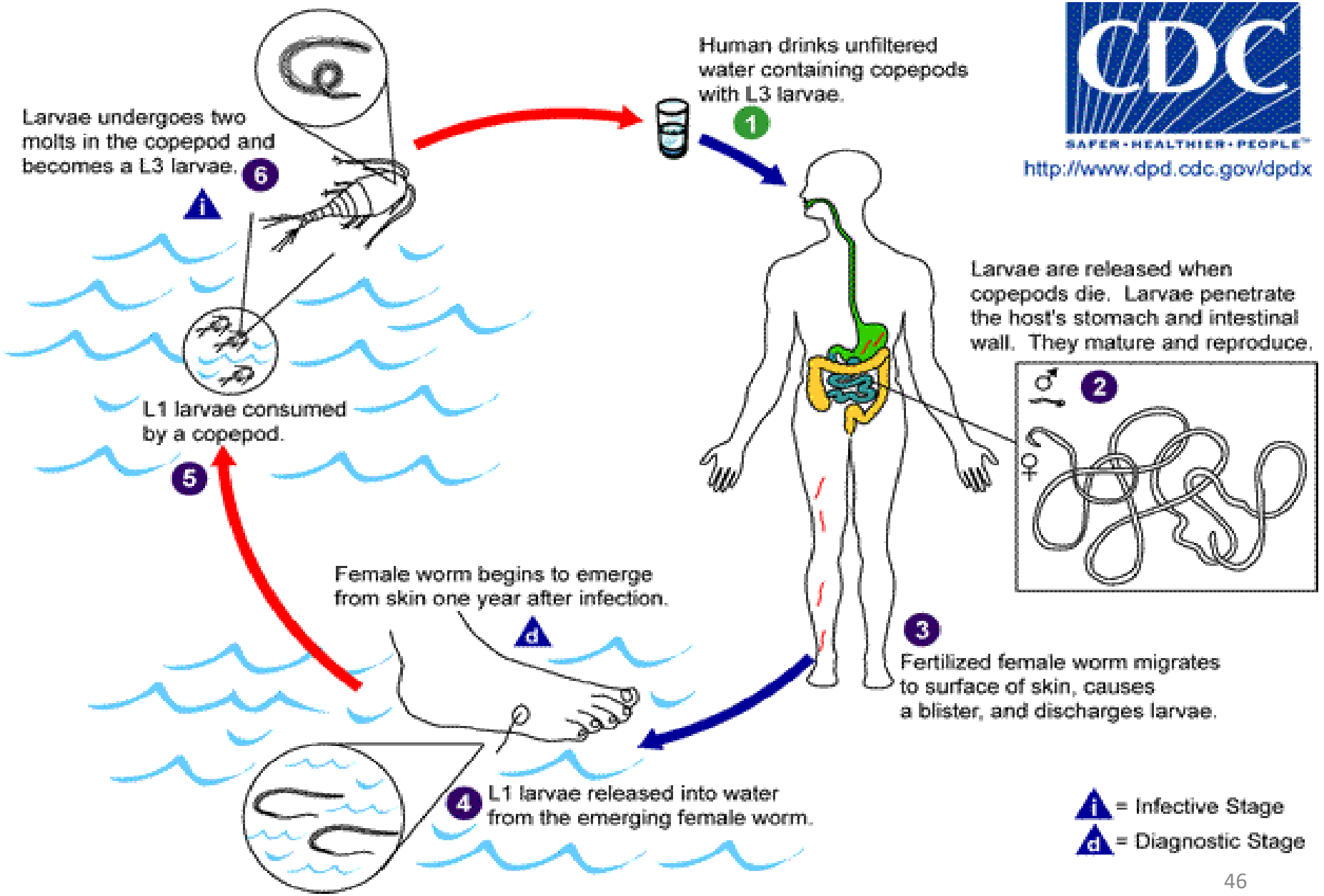
Life Cycle of D. medinensis



Pathogencity

- Early manifestatiosn when the female worm **approaches the skin**. It liberates a **toxic** substance that results in local erythema, tenderness and pain.
- Formation of a blister that turns into a vesicle & ultimately ulcerates------due to **irritant** chemicals
- Local or systemic symptoms as urticaria, pruritus, pain, dyspnoea, nausea and vomiting, which subside with rupture of the blister
- The ulcer may be secondarily infected producing **cellulitis** and **induration**
- **Eosinophilia ???**

D. medinensis

Blister containing the worm

A

Ruptured blister with filamentous worm

B

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Adult worm of D. medinensis

Diagnosis of D. medinensis

- Laboratory tests to investigate dracunculiasis are limited because the larvae are normally washed into water
- A diagnosis is usually made when the blister has ruptured and the **anterior** end of the female worm can be seen

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Diagnosis of D. medinensis

- Laboratory confirmation of the diagnosis can be made as follows:

1. Place a few drops of water(cold) on the ulcer to encourage discharge of the larvae
2. After a few minutes collect the water in a plastic bulb pipette or pasteur pipette
3. Transfer the water to a slide and examine microscopically using 10x objective – **motile larvae** will be observed

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Prevention & Treatment

- People with an open Guinea worm wound should **not** enter ponds or wells used for drinking water.
- Water can be boiled, filtered through tightly woven nylon cloth, or treated with a larvae-killing chemical.
- **No** medication is available to end or prevent infection.

Prevention & Treatment

- The only treatment is to remove the worm over many weeks by **winding** it around a small **stick** and pulling it out a **tiny** bit at a time.
- Sometimes the worm can be pulled out completely within a few days, but the process usually takes weeks or months.
- The worm can be surgically removed before the wound begins to swell.
- **Antihistamines** and antibiotics can reduce swelling and ease removal of the worm.

Removing of D. Medinensis by stick

Leishmania species

- Causative agent of **Leishmaniasis**
- **Obligate intracellular** protozoa of the genus ***Leishmania***
- Named after Leishman, who first described it in London in May 1903
- Human infection is caused by about 21 of 30 species that infect mammals.
- The species are morphologically indistinguishable, but they can be differentiated on the basis of on their
  - ✓ clinical features, geographical distribution, serologic tests

- Leishmaniasis can easily classified clinically as

  - **Visceral leishmaniasis**
  - **Cutaneous leishmaniasis**
  - **Mucocutaneous leishmaniasis**
  - **Diffuse cutaneous leishmaniasis**

- These different forms of the disease is caused by the different species of *Leishmania*

- **Cutaneous leishmaniasis(CL)**
  - *L. tropica*
  - *L. major*
  - *L. aethiopica*
  - *L. panamensis*
  - *L. guyanensis*
  - *L. peruviana*

- **Visceral leishmaniasis(VL)**
  - *L. donovani* -----**PKDL**
  - *L. infantum*------***old world CL***
  - *L. chagasi*

- **Mucocutaneous leishmaniasis(MCL)**
  - *L. panamensis*
  - *L. guyanensis*
  - *L. Brazilliensis*

- **Diffuse cutaneous leishmaniasis(DCL)**
  - Cutaneous infection with non- ulcerating nodules resembling **lepromatous** leprosy
    - ✓*L. amzonensis*
    - ✓*L. aethiopica*

Epidemiology

- 350 million people are at risk in 88 countries around the world
  - ✓72 of which are developing countries

- An estimated 12 million cases world wide ;1.5 to 2 million new cases occur every year
  - ✓CL form representing 50 to 75% of all new cases

- Geographical distribution of leishmaniasis is **limited** by:
  - The distribution of the sand fly,
  - Its tendency to take blood from humans or animals only, and
  - Its capacity to support the internal development of specific species of *leishmania*

- The incidence of leishmaniasis is increasing, mainly because of:
  - Man-made environmental changes
  - Poverty and malnutrition
  - Movement of **susceptible** populations into endemic areas

- **In Ethiopia**
  - Four species of *Leishmania is found*, namely,
    - ✓*L. aethiopica*,
    - ✓*L. major*
    - ✓*L. tropica*
    - ✓*L. donovani-----?*

- **Cutaneous leishmaniasis**

  In Ethiopia:

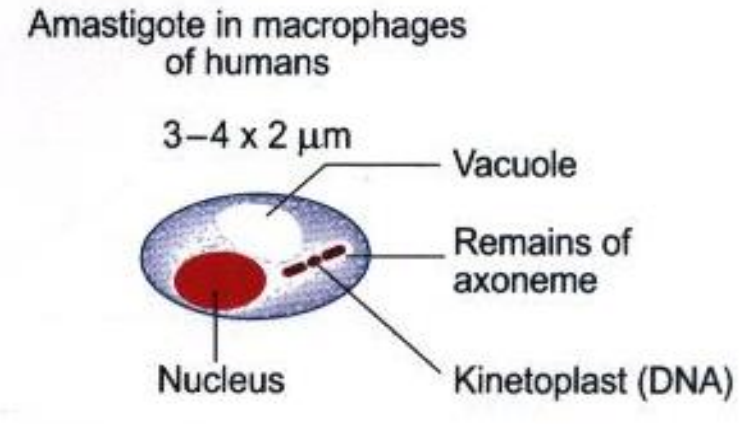
  – Endemic at altitudes between 1400 and 2700 m in most administrative regions

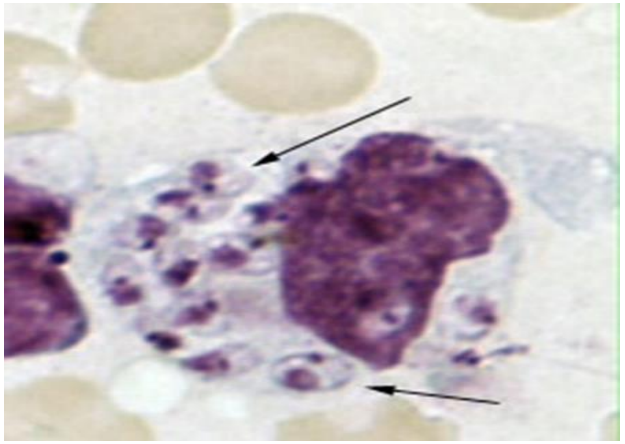
  – Prevalence rates of 5.5 – 40% were reported from villages in Shewa , Wello and G.Gofa with the highest rate in Ocholo village in G. Gofa

  – **rock hyraxes** (*Procavia habessinica*) and **tree hyraxes**(*Heterhyrax brucei*) serving as reservoir host for *L. aethiopica*

Morphology and habitat

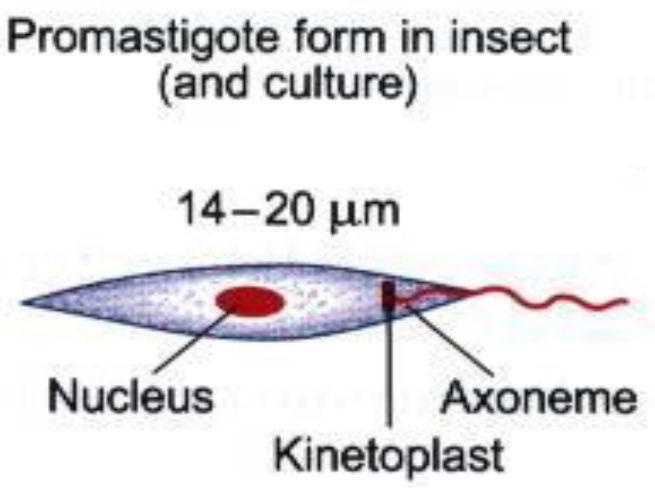
- Amastigote (Leishmanial form)
  - Rounded body, central nucleus and eccentric kinetoplast visible
  - no free flagellum,
  - The intracellular forms of all leishmania species





Morphology…

- **Promastigote (Leptomonad) form**
  - Elongated body, central nucleus, **anterior** kinetoplast
  - Single anterior flagellum arises from kinetoplast
  - found in the **invertebrate host**, and in **culture media** (of all *Leishmania* species)



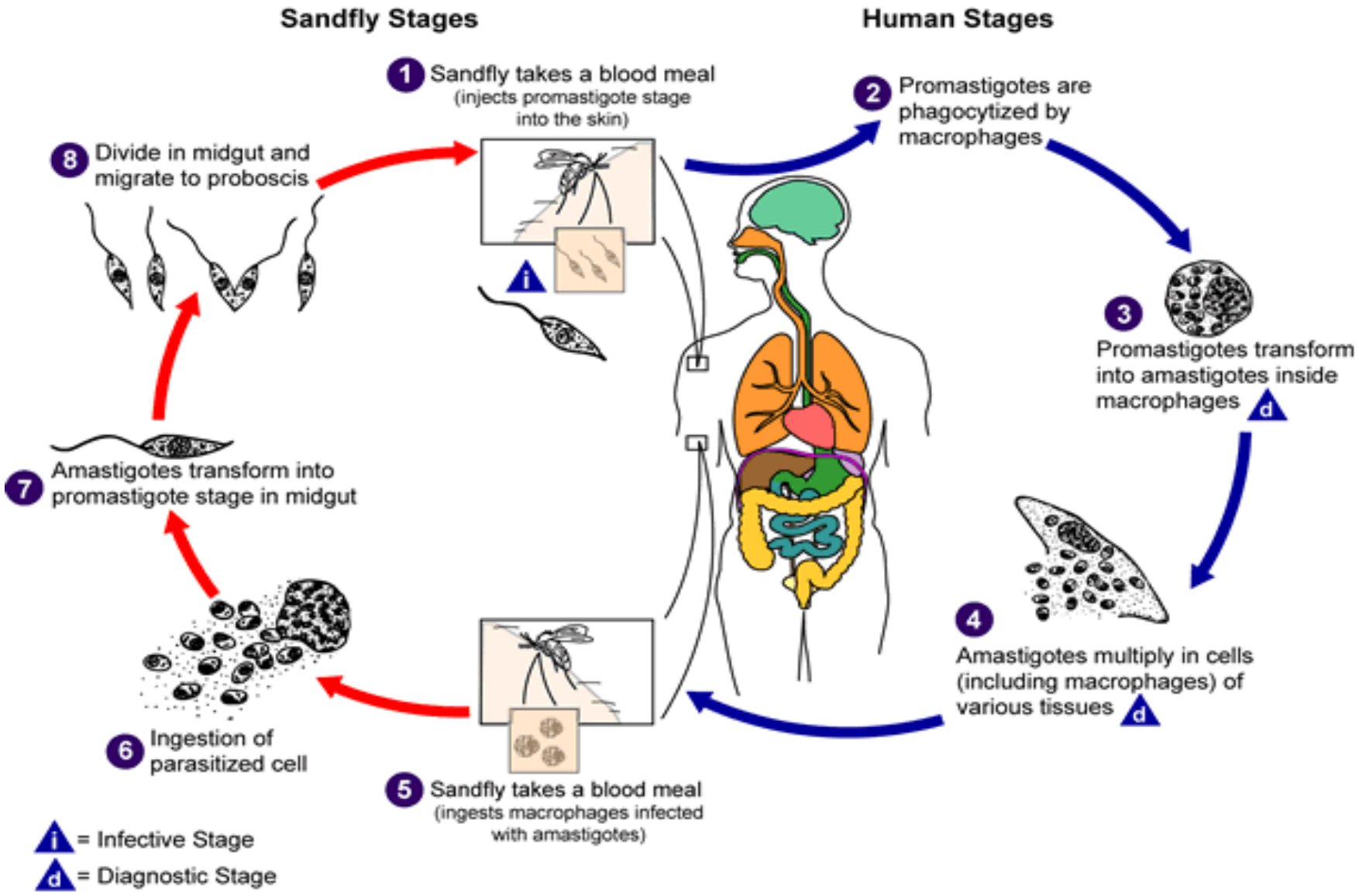
Transmission

- **Common** mode of transmission:
  - **Bite** of **sandflies**
    - ✓Genera *Phlebotomus in* Old world
    - ✓*Lutzomyia in* New world
- **Uncommon** modes of transmission:
  - Congenital transmission,
  - Blood transfusion,
  - Rarely, inoculation of cultures

Mammalian R. Hosts

- **Rodents**
- **Gerbils**
- **Hyraxes**
- **Bats**
- **Porcupines**
- **Opossums**
- **Sloths**
- **Primates**
- **Dogs**
- **Foxes**
- **Anteaters**
- **. . . . .**

Life cycle of *Leishmania* species



Pathogenesis

- Entrance into the host and establishment of infection by Leishmania is enhanced by **saliva** from the vector

- Two substances were involved
  - ✓**maxadilin,** or maximum dilation molecule: keeps the capillary bed open
  - ✓ **SIP** or **salivary immunosuppressive protein** : restrains the immune system's early efforts to eliminate the parasites

Pathogenesis…

- Infective promastigotes entering the blood of the vertebrate are covered by two key molecules:
  - The **protein gp 63** and **lipophosphoglycan (LPG):**
    - Both mediate the uptake of promastigotes by **macrophages**

- The promastigotes are engulfed & form **phagosome**

Pathogenesis…

- Phagosome fuse with the lysosome to form a **phagolysosome**
  - ✓ As the **promastigotes** transform into **amastigotes**, which produce compounds that counter lysosomal enzymes
    - ✓ The **gp 63** molecule **inactivates** proteolytic enzymes
    - ✓ **LPG** protects against other enzymes
- Leishmanial organisms are able to survive the highly acidic environment of lysosmes by regulating their internal P^H
- In addition, the parasite **shuts down** the generation of reactive oxygen intermediates by the macrophage.
  - E.g catalase, superoxide dismutase

Pathogenesis..

- **Intracellular** parasites are **difficult** to kill. This has important implication for the host immune response:
  - Antibody is not effective at killing leishmania
  - The most effective immune response is **killing parasites within the infected cells.**
  - The infection site has to be **walled off** by a ring of immune cells to prevent the spread of infection.
- The most important cells to protect against leishmania infection are:
  - **Macrophages** and **CD4+Th1 cells**

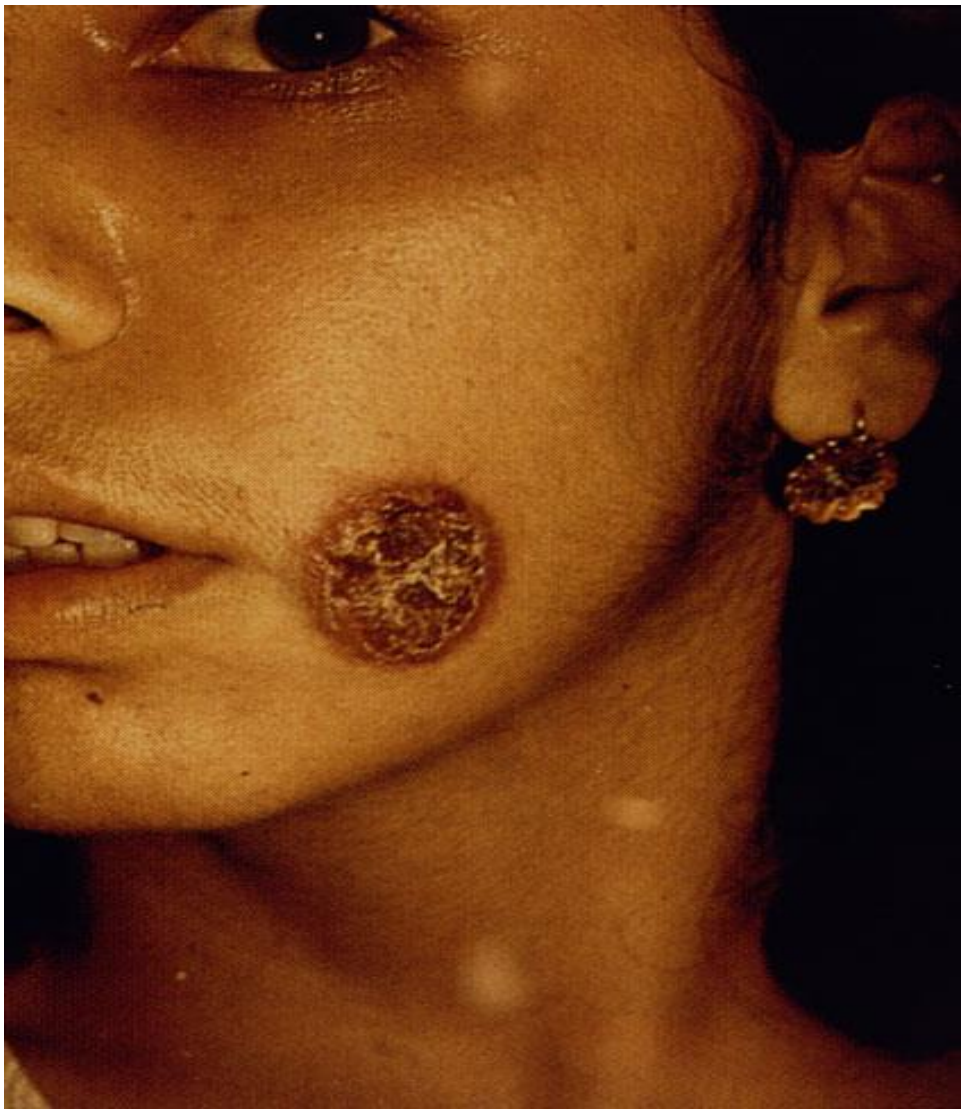
Clinical features and pathology

Cutaneous Leishmaniasis

- Causative agents
  - ✓ *Leishmania tropica*
  - ✓ *Leishmania major*
  - ✓ *Leishmania aethiopica*
  - ✓ *Leishmania mexicana*
  - ✓ *Leishmania peruriana*
  - ✓ *Leishmania panamensis*
  - ✓ *Leishmania guyanensis*

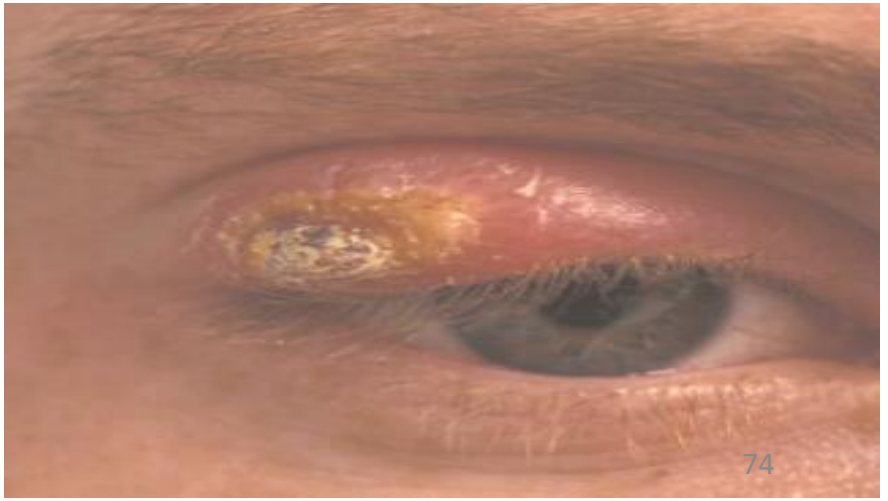
- most common form,
- Relatively benign self healing skin lesions (localized or simple CL)

Old World CL

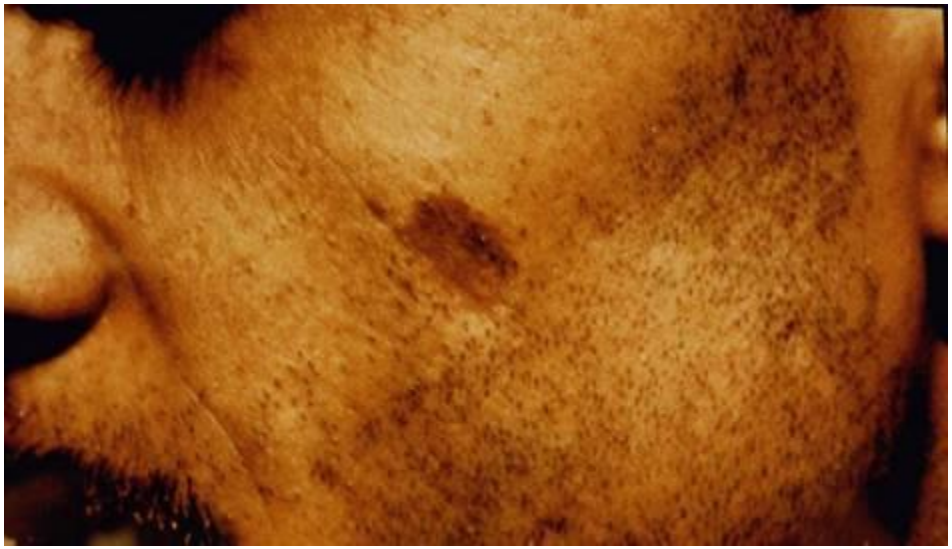


L. Tropica

- SW Asia, N.Africa
- **Anthroponotic** or dog reservoir
- dry ,urban ,chronic, old world oriental sore
- **'dry painless lesion'**
  - ✓ 25-70mm diameter



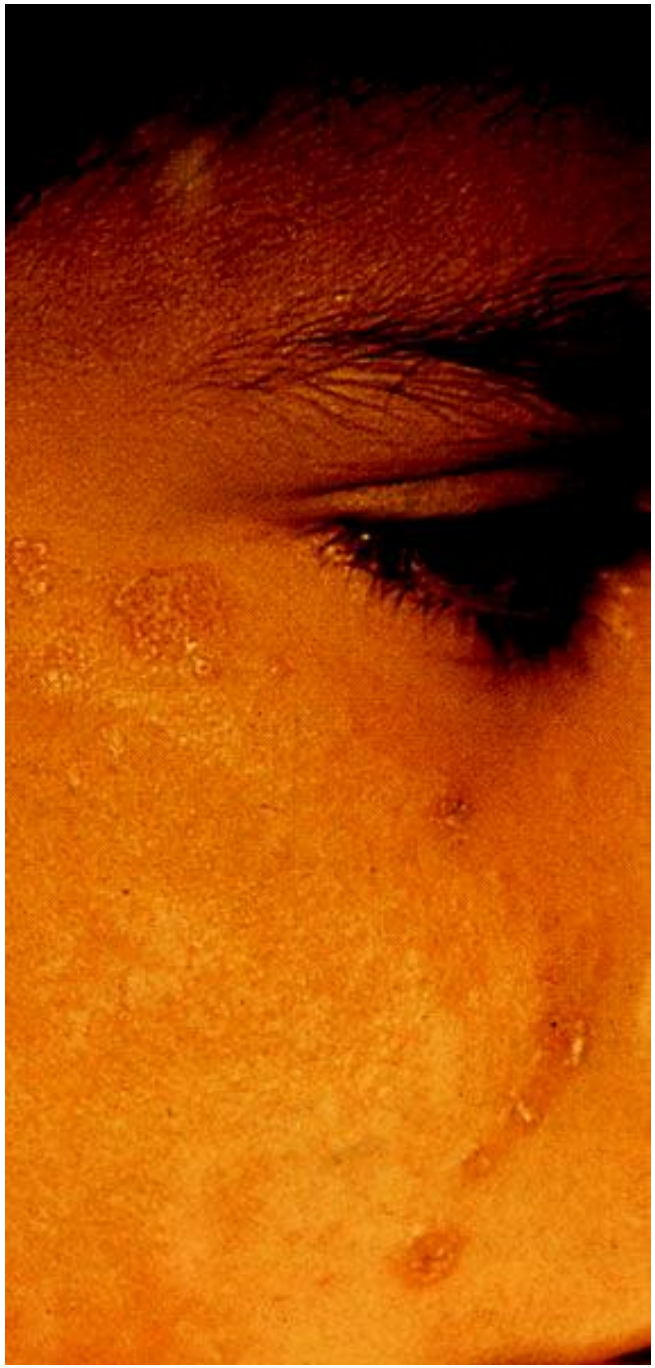
Old World CL….



hyper-pigmentation of scar

L. tropica

- Are self-healing,1-2yrs
- Often **leave** disfiguring scars
- Immune to re-infection
- Rarely develop multiple un-healing lesion known as leishmaniasis recidivans (LR)



Old World CL….

Leishmaniasis Recidivans (LR)

- multiple un healing lesions, often on the face
- **Relapsing** leishmaniasis
- Often due to inadequate treatment or allergic state
- Nodular lesions or rash around central healing
- Can last for many years and difficult to treat
- Untreated LR is destructive and disfiguring

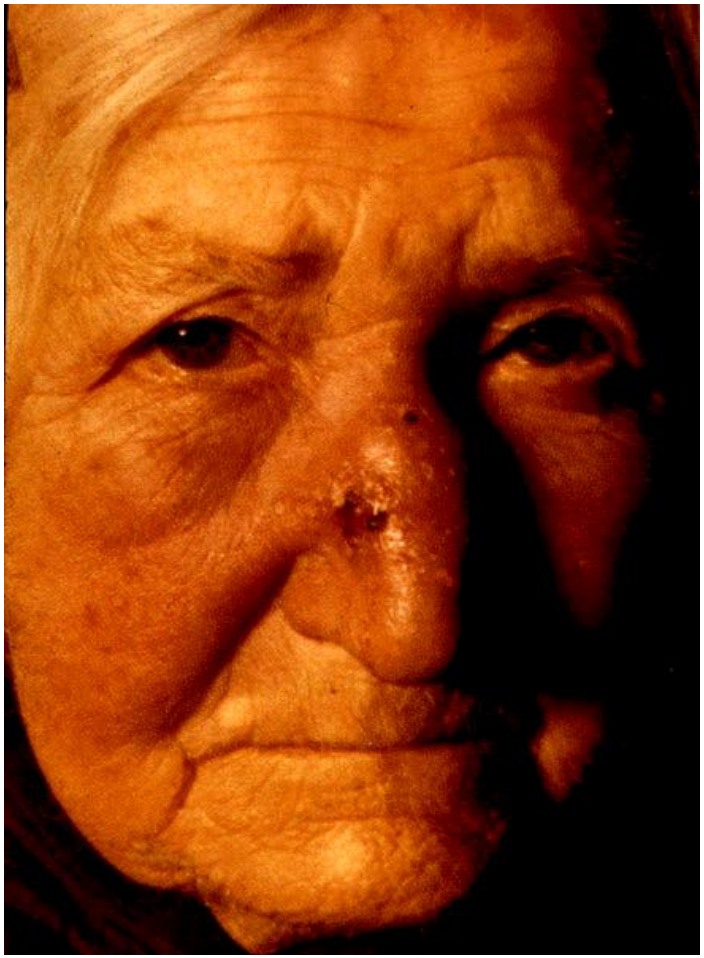
Old World CL….

ulcers are moist or open with seropurulent exudate

L. major

- central Asia, middle East, Africa
- rural (rodent reservoir)
- wet oriental sore
- Early papules is inflamed (5-10mm)
- Develop to large uneven ulcer
- Self-healing (3-6mths)
- Protect against reinfection & also with *L. tropica*

Old World CL…



L. infantum

- ✓ Mediterranea, Europe
- ✓ **dermotrophic** strains recently recognized

L. aethiopica

- ✓ highlands of Kenya and Ethiopia
- ✓ Similar to oreintal sore
- ✓ Self-heal 1-3 yrs
- ✓ Can cause DCL

New World CL

L. mexicana

- ✓ Initially, the lesion is a small, red papule up to 2 cm in diameter
- ✓ Change in size and appearance over time
- ✓ Chiclero Ulcer

New World CL…

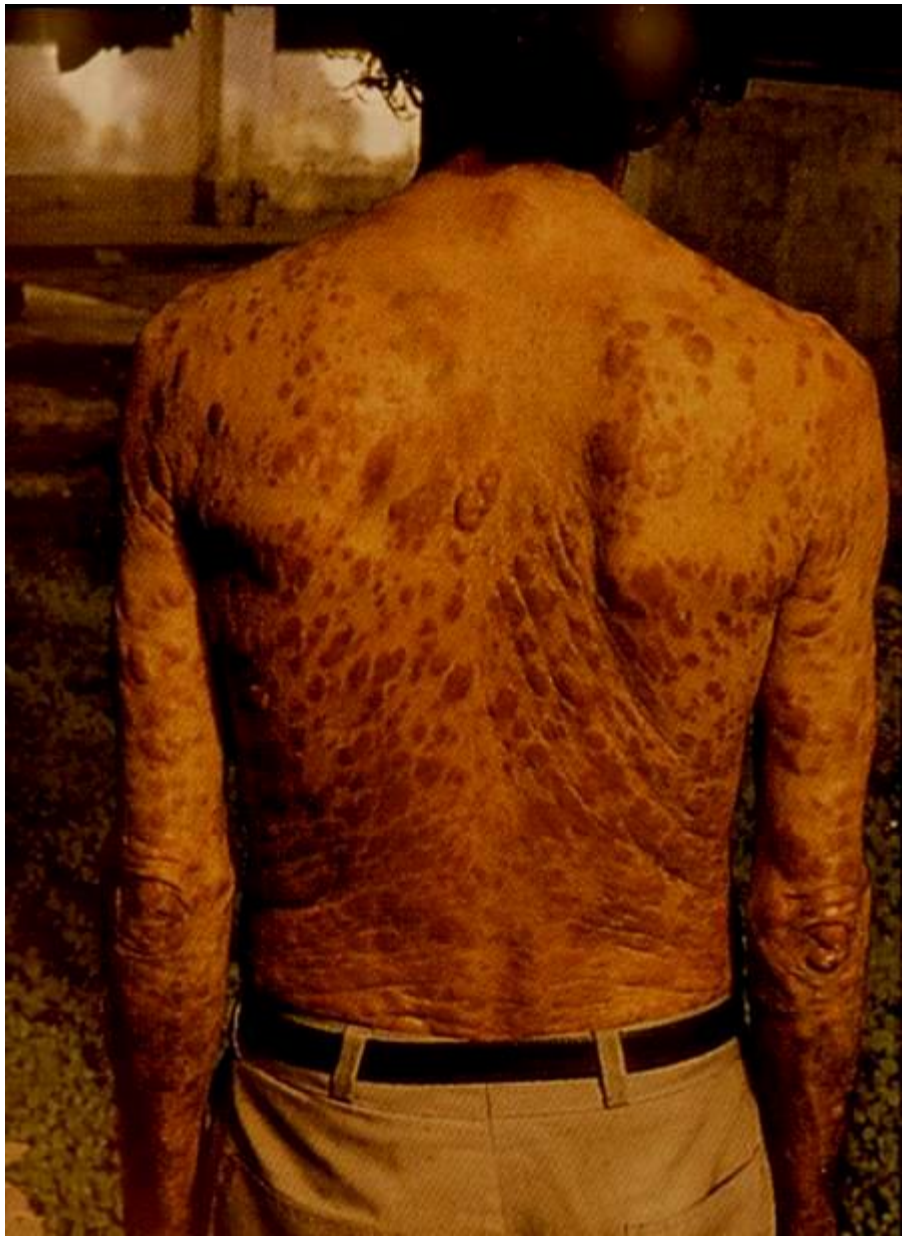
L. mexicana

- chronic ulcerated, papular, or nodular lesion
- lesion is painless, non-tender, non-pruritic

Lesions of the body tend to self-healing but those on the ear may last up to 30 years and entirely destroy the pina of the ear

Diffuse Cutaneous Leishmaniasis

- Caused by ***L. aethiopica*** **and** ***L. amazonensis***
- Skin lesion develop over large areas of the body
- Scaly, not ulcerated, nodules
- Chronic and painless
- Numerous parasites in lesions
- Seldom heal despite treatment



Mucocutaneous Leishmaniasis

- simple skin lesions that metastasize to mucosa especially nose and mouth region

- Primarily *L. braziliensis* : known as **espudia**
- Two stages
  - ✓ simple skin lesion
  - ✓ 2º mucosal involvement
- Metastasis via blood or lymphatic systems
- Can occur after primary lesion (up to 16 years)
- Frequently in naso-pharyngeal mucosae
  - ✓ Junction of skin and mucosa

Mucocutaneous Leishmaniasis

- Variable types and sizes of lesions
  - ✓ chronic and painless
- Non-ulcerative type
  - ✓ local edema (upper lip)
- Ulcerative type
  - ✓ rapid and extensive mutilation

Mucocutaneous Leishmaniasis

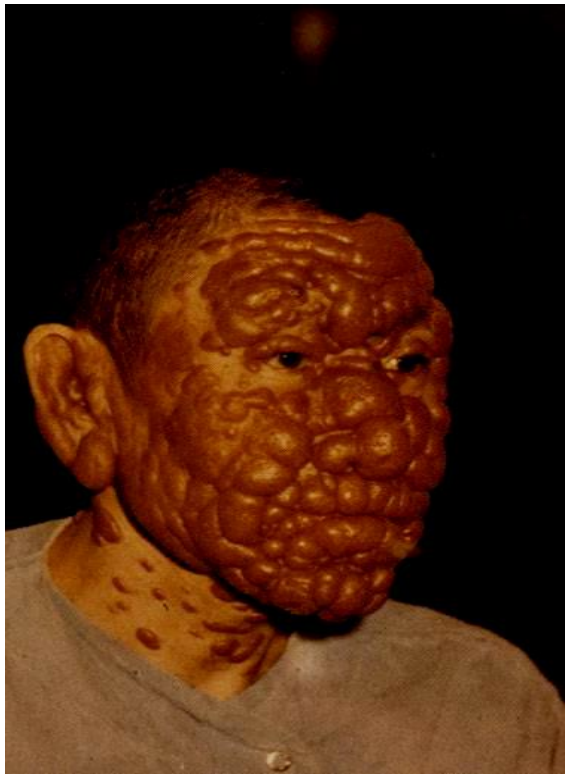
- Disfiguration is often extreme with complete destruction of the
  - ✓ nasal septum, perforation of the palate and damage of the tissues of the lips and larynx
  - ✓ **'tapir'** nose

Post Kala Azar Dermal leishmaniasis (PKDL)

- ✓ Cutaneouse form of leishmaniasis, which can occur after resolution (after treatment and recovery) of VL
- ✓ It require expensive and prolonged treatment
- ✓ characterized by hypo pigmented and raised erythematous patches on the face, trunk of the body and limbs
- ✓ May develop in to nodules and resembles those of lepromatous leprosy, fungi infections or other skin disorders

PKDL

- ✓ Occasionally there is ulceration of lips and tongue
- ✓ occurs in 1-3% of Indian and 50% of Sudanese VL patients



Laboratory Diagnosis of CL, MCL, DCL

- Suspected because of:
  - ✓ geographical presence of parasite
  - ✓ history of sandfly bite
  - ✓ positive skin lesion:
    - chronic, painless, 'clean' ulcer
    - nasopharyngeal lesions
    - nodular lesions

Laboratory diagnosis:

1. Demonstration of parasite amastigotes (scrapings, biopsy, aspirates)
2. Culture from ulcer material
3. Leishmainin test
4. serology?

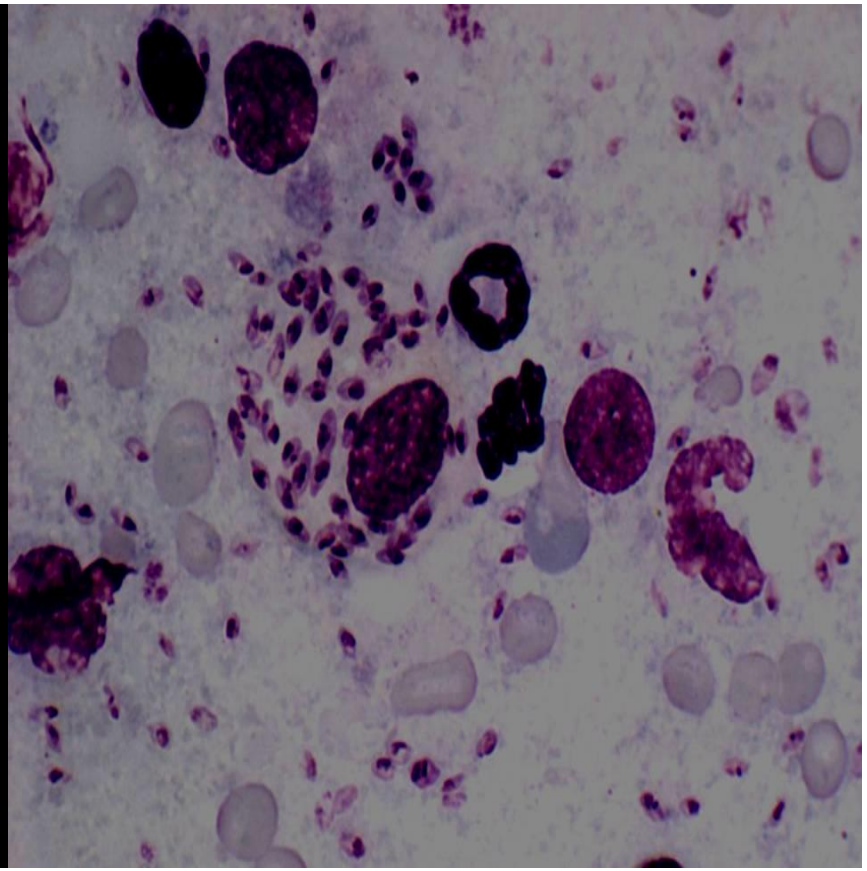
1. Collection and examination of slit skin smears for amastigotes

- Should be taken from the inflamed raised swollen **edge** of an ulcer or nodule **not** from its base or centre which usually contains only necrotic tissue

- If bacterial infection is present, examination for Leishmania amastigotes is best delayed until antimicrobial treatment has been completed and the bacterial infection has cleared.

Make incision in active part of lesion

Prepare Giemsa-stained smear

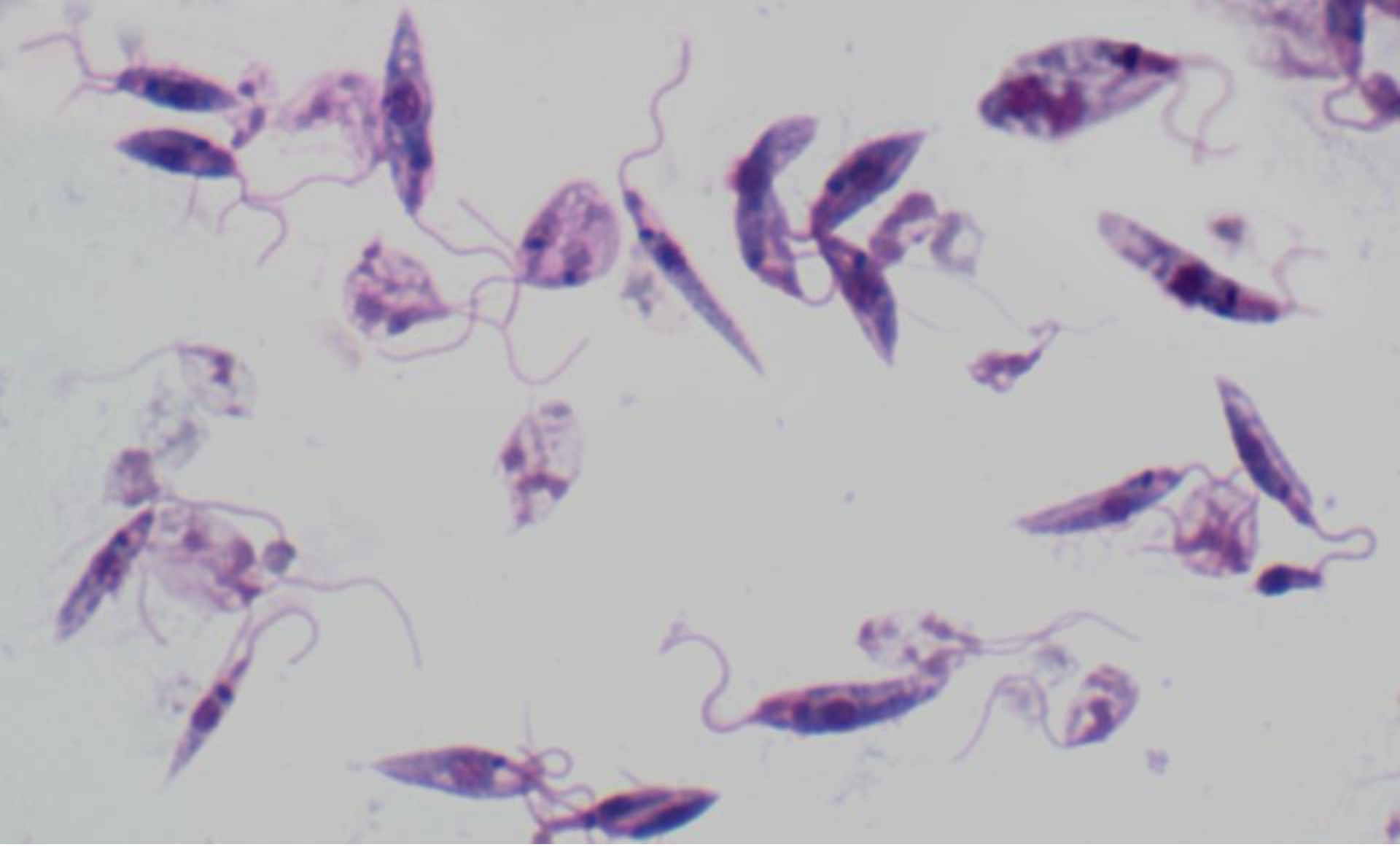


2.Culture of ulcer material

- When cutaneous leishmaniasis is suspected and parasites cannot be found in smears

- Material for culture is best obtained by injecting and then aspirating a small quantity of sterile physiological saline in and out of the hardened margin of the ulcer

- A few drops of the final aspirate is used to inoculate the culture medium
  - ✓Novy –Nicolle-MacNeal(NNN), M199, or Grace's and Tobies medium



promastigotes following in vitro culture

3.Leishmainin or Montenegro test

- Delayed Hypersensitivity Skin Test
- intradermal inoculation of leishmanin
  - ✓ suspension of whole or killed promastigotes
  - ✓ preferably from local area
  - ✓ include negative control
- *Positive reaction:* when the area of indurations ± erythema of 5mm in diameter or more indurations in 48-72 hours

4.Serology

- Because of the poor antibody response in CL, serological tests are of little value in diagnosis

Treatment

- Sodium stibogluconate (Pentostam)
- Pentamidine isethionate
- Amphotericin B
- Cryotherapy and thermotherapy

Prevent and control

1. Personal protection from sand fly bites by:
   - Using insect replants
   - Avoiding endemic areas especially at times when sand flies are most active
   - Use of pyrethroid impregnated bed nets and curtains

2. Vector control by the use of light traps, sticky paper traps, or residual insecticide spraying of houses

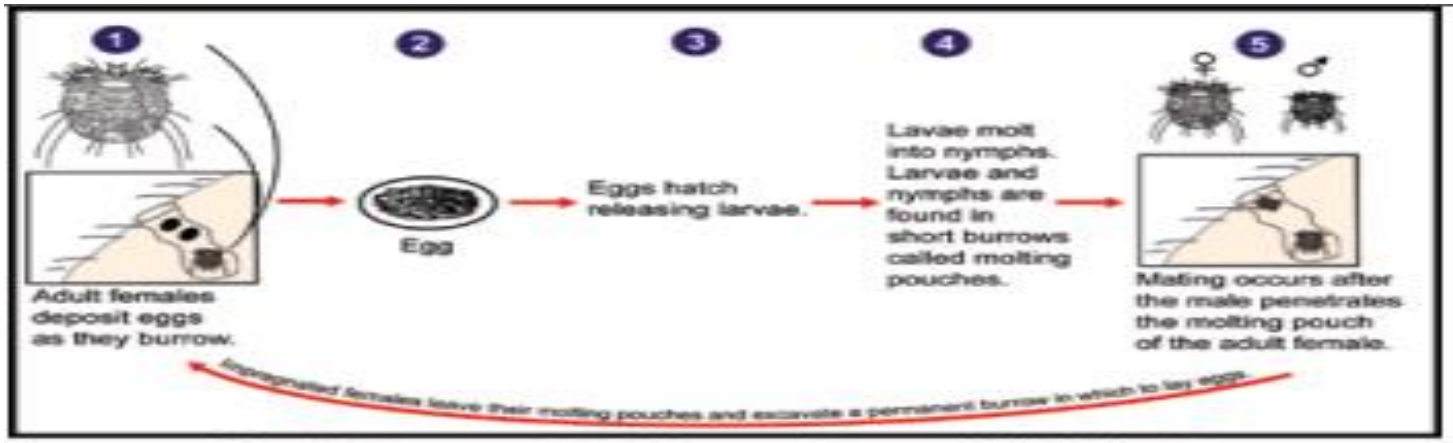
Prevent and control…

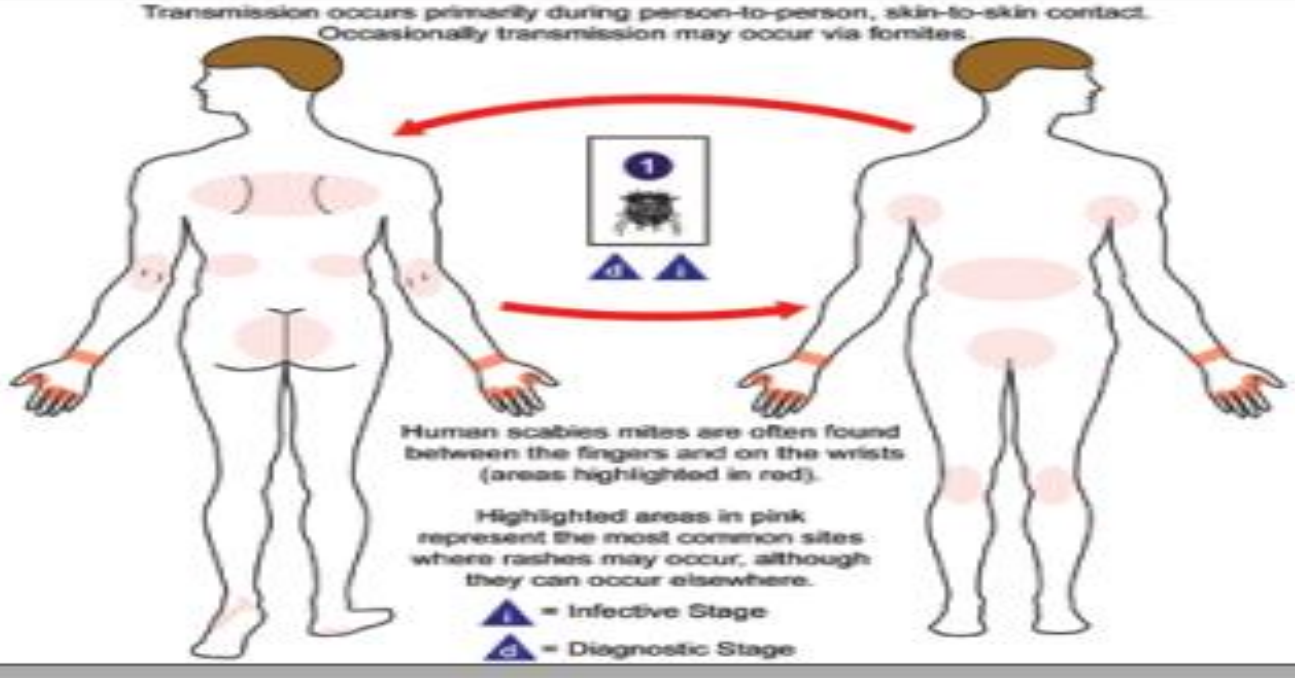
3. Destruction of stray dogs and infected domestic dogs
4. Elimination and control of rodents
5. Sitting human dwellings away from the habitats of animal reservoir hosts where sand flies are known to breed

Sarcoptes scabiei

- *Sarcoptes scabiei* var. *hominis* is the human itch mite causing scabies
- **Transmission**
  - ✓ spread by direct, prolonged, skin-to-skin contact with a person who has scabies
  - ✓ Spread of the infection to other areas of the body → by scratching
  - ✓ spread rapidly under crowded conditions where close body contact is frequent
    - nursing homes, extended-care facilities, and prisons
- **Distribution**: World wide

Life cycle


1
2
3
4
5
Egg
Eggs hatch releasing larvae.
Larvae molt into nymphs. Larvae and nymphs are found in short burrows called molting pouches.
Adult females deposit eggs as they burrow.
Mating occurs after the male penetrates the molting pouch of the adult female.
Impregnated females leave their molting pouches and excavate a permanent burrow in which to lay eggs.


Transmission occurs primarily during person-to-person, skin-to-skin contact. Occasionally transmission may occur via fomites.
1
Human scabies mites are often found between the fingers and on the wrists (areas highlighted in red).
Highlighted areas in pink represent the most common sites where rashes may occur, although they can occur elsewhere.
= Infective Stage
= Diagnostic Stage

Pathogenesis and Clinical feature

- Adult mites enter the skin, creating serpiginous burrows in the upper layers of the epidermis.
- The female mite lays her eggs in the skin burrows, and the larval and nymph stages that develop also burrow in the skin
- The female mites live and deposit eggs and feces in epidermal burrows for up to 2 months
- The presence of the mites and their secretions cause intense itching of the involved areas
- The mite is an obligate parasite and can perpetuate itself in a single host indefinitely

Pathogenesis and Clinical feature

The most common symptoms of scabies are

- itching and a skin rash caused by **sensitization** (a type of "allergic" reaction) to the proteins and feces of the parasite
- Severe itching (pruritus), especially at night, is the earliest and most common symptom of scabies
- A **pimple-like** (papular) itchy (pruritic) "scabies rash" is also common
- The intense itching of scabies leads to scratching that can lead to skin sores
  - The sores sometimes become infected with bacteria

- **Diagnosis**
  - ✓Usually clinical presentation
  - ✓identifying the mite or mite eggs by carefully removing the mite using needle or skin scraping
- **Treatment**
  - ✓Scabicide lotion or cream
- **Prevention and control**
  - ✓Avoiding direct skin contact with an infested person or with items such as clothes
  - ✓Wash clothes with hot water and clean home
  - ✓Treated infected person and family members
  - ✓Health education

Reading assignment

- ***Trichinella larva:*** Migration of larvae to muscle causes muscle damage.
- ***Entamoeba histolytica***: cutaneous amoebiasis...perianal ulcers, urogenital ulcers (e. g, labia, vagina, penis)
- **Schistosmoa cercaria:** Penetration of skin by cercaria causes transient dermatitis (swimmers itch)
- **Hook worm larvae**: At the site of skin penetration cutaneous invasion of the infective larvae causes local reaction called **ground itch** characterized by erythematous, papular rash
- **Trypanosoma bruci complex**: multiply in the tissue at initial bite site—local inflammation-----Trypanosoma chancer
- **Strongyloides Larva**: Large number of larva produce itching and erythema at the site of infection within 24 hours of invasion. At the site of penetration of skin the infective filariform larvae cause itchy dermatitis (ground itch), rash and epidermal atrophy with hypo-pigmentation.
- **Myiasis**: infestation of the body by the larvae of flies (usually through a wound)