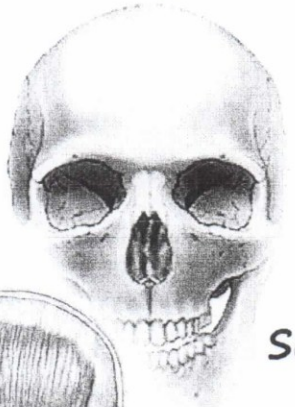
Lecture # :1

MSS

Musculoskeletal System

Medical Committee
The University of Jordan

[Microbiology]



Subject: Leishmania, Etc...

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Done by: Slides

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☒Slides ☐Sheet

☐Handout ☐Lab

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Trichinella spiralis :

Small worm 1.5 - 3.5 mm, adult rarely seen, larva has spear-shaped anterior end, there are no eggs laid.
Same animal for intermediate and definite host. Carnivores and omnivores e.g. human, pig, cat, rat, dog and foxes.
Transmitted by eating flesh containing viable encysted larvae.

Life cycle :
Encysted larva are eaten in raw meat, the larva is released in small intestine, burrows into mucosa, matures to adult male or female worms in 24 hours, female ready to lay larvae in 5 days, the male is expelled after fertilization into the lumen and passed out in the faeces.
The laid larvae (no eggs) reach lymphatics and then the blood stream (passes through the hepatic and pulmonary capillaries), and become disseminated to all parts of the body, but they can only develop further in striated muscle, in other tissues they soon disintegrate. They encyst in the muscles (in about 3 weeks) and eventually become calcified in 1/2 to 2 years (larvae remaining viable for years).
They may deposit in lungs, CNS, myocardium, where they may cause symptoms and then disintegrate.
Common in Europe and USA, from eating pig or exotic meat like bear meat. It is becoming less prevalent, due to better control over garbage fed to pigs, but occasionally a pig may eat an infected dead rat (cannibalistic rats perpetuate the infection). Hamburgers adulterated with pork may also cause infection. Absent in Moslems and Jews.

Symptoms :
G.I. diarrhoea, nausea, vomiting (due to adult worms).
Spread through blood may cause malaise, headache and fever (only Helminth to produce fever). There may be allergic manifestations e.g. skin rashes and periorbital oedema.
Myocarditis, pneumonia, meningism and encephalitis. Muscle aches and tenderness, symptoms are usually mild unless there is heavy infestation.
If the dose is massive death can ensue in 2-3 weeks.

Diagnosis:
Eosinophilia may be 40-80%, maximal at 4 weeks then declines. Eosinophils are important in immunity as they kill the larvae with the help of specific antibody IgE and IgG.
Specific IgE develops and useful in diagnosis (agglutination), seroconversion, or increase in titre, the IgE does not last more than one year (i.e. test is not +ve after 1 year) a strong +ve test indicates recent infection. Antibodies may give resistance against re-infection.
Muscle enzymes may be raised CPK, LDH.
The most definite diagnosis is muscle biopsy (in the 3rd to 4th week of infection), N.B. calcified cysts with no surrounding reaction may be old lesions not related to the present condition.
Treatment :
Mebendazole, thiabendazole. Steroids for myocarditis and CNS involvement. Bed rest and symptomatic therapy.

Avoid raw pork, sterilize hog feed, cannibalistic rats may perpetuate transmission (pigs eating dead rats).
Freezing meat to -21 degrees C. for 2-3 weeks kills the larvae.

Leishmania:

In the host, amastigote, 3 microns in diameter inside macrophages thriving inside the lysosomes.

The vector is the sandfly, **promastigote** in the fly (flagellum at one end, no undulating membrane) in the host it is an **amastigote** which infect endothelial cells and macrophages.
It is an intracellular parasite.
Wild reservoir includes dogs and rodents.
Access to phagocytes through complement C3b receptors.

Old world L. tropica, major and aethiopica.
L. tropica : also related to this are L. major and L.aethiopica, causes cutaneous disease (cannot grow at body core temperature) , nodule at site of bite which eventually ulcerates, it heals after about a year leaving a disfiguring depigmented scar. Immunity is solid (no recurrence).

New world : L. mexicana and L. braziliensis
L. braziliensis : causes mucocutaneous disease which may be fatal.
Initial lesion is similar to cutaneous leishmaniasis, it heals, but after months or years the lesions reappear involving the nasopharynx, destructive lesions and mutilation.
Nasal obstruction and bleeding.

L. donovani : also related are L. infantum and L. chagasi, causes visceral disease (kala azar).
Clinical disease tends to be associated with malnutrition.
A primary lesion may rarely be observed, followed 2-3 months later by fever, weight loss, enlarged spleen, liver and lymph nodes. Hyperpigmentation (black sickness).
If not treated 90% die after 2 years, the onset is insidious.

Diagnosis : tissue biopsy. Demonstration of parasite or culture. Antibodies are present in all forms but most useful in visceral disease where skin lesions are not apparent.
Bone marrow and node biopsy in visceral disease.

Treatment : pentavalent antimony compounds. Pentamidine.

Different Leishmania species distinguished by clinical and geographic difference otherwise they are identical, new methods of delineation are becoming available.
Transmitted from salivary glands of sandfly, it is a zoonosis e.g. rodents and canine animals dogs. Transmission directly from humans can occur.
After entry, complement is activated but at a distance from the membrane, C3b then helps the organism to enter the macrophage, flagellum is lost and becomes an obligate intracellular amastigote the Donovan body.

The clinical picture then depends on the response of the host and the species :

1)- Cutaneous : intense cell mediated reaction, IFN gamma, activation of macrophages and destruction of parasite.

2)- Diffuse cutaneous : antibody response but little cellular, similar to lepromatous leprosy. (L. aethiopica).

3)- Visceral (Kala-azar or black fever due to pigmentation of skin) :
spread of parasite to all RE system. This is probably to greater resistance of L. donovani to cidal effect of serum.

4)- Mucocutaneous : apparent cure of initial skin lesion is followed by disseminated mucocutaneous lesions at a later date.

TRICHINELLA SPIRALIS

TRYPANOSOMATIDAE

- TRYPANOSOMA
- LEISHMANIA

AMASTIGOTE

PROMASTIGOTE

L. TROPICA
L. MAJOR (OLD)
L. AETHIOPECA

L. MEXICANA (NEW)
L. BRAZILIENSIS

L. DONOVANI
L. CHAGASI
L. INFANTUM

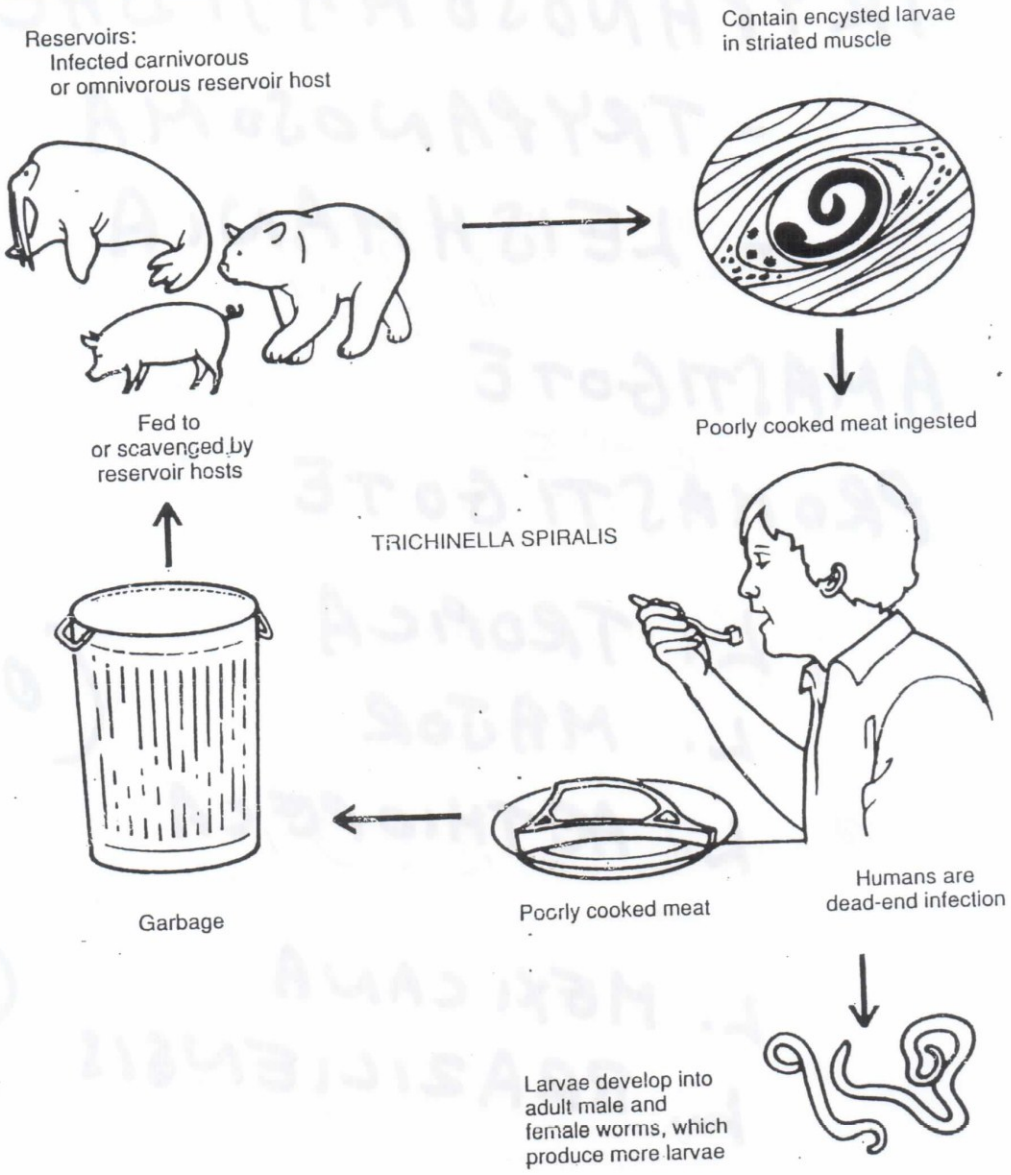

Figure 6–2. *Life cycle of Trichinella spiralis.*

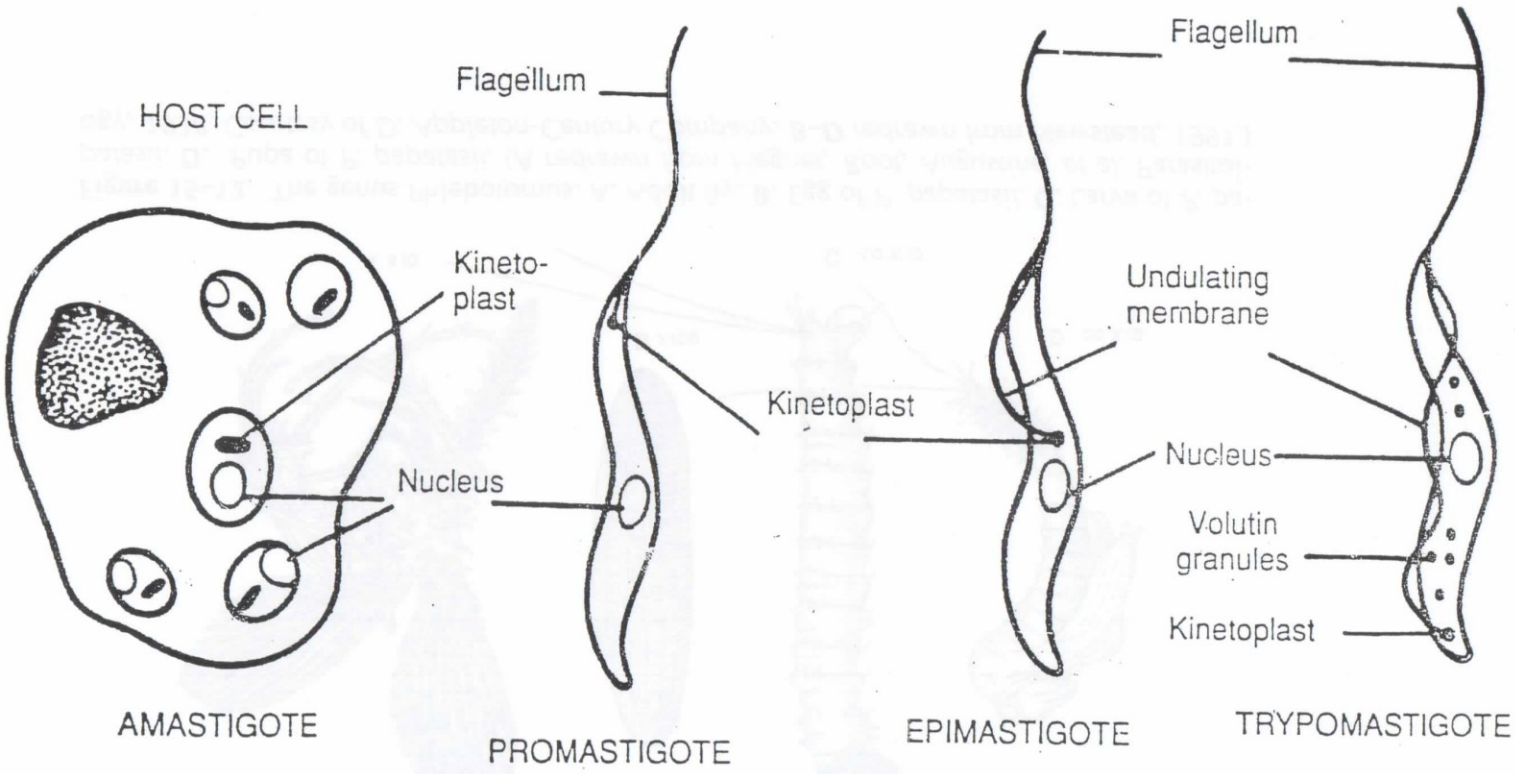

Figure 4–1. Developmental forms of Trypanosomatidae.

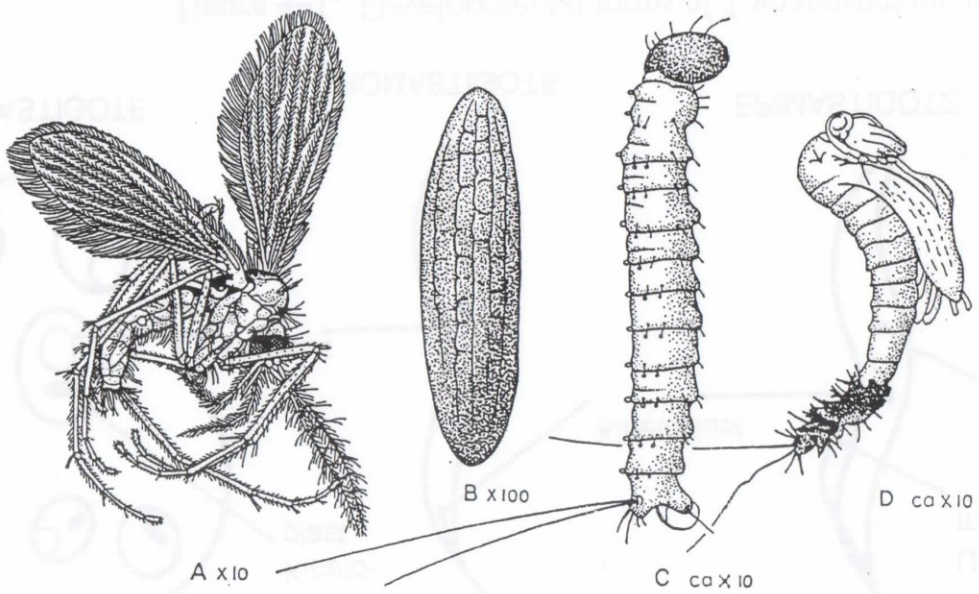

Figure 15–12. The genus Phlebotomus. **A.** Adult fly. **B.** Egg of *P. papatasii*. **C.** Larva of *P. papatasii*. **D.** Pupa of *P. papatasii*. (*A redrawn from Hegner, Root, Augustine, et al.* Parasitology, *1938. Courtesy of D. Appleton-Century Company. **B–D** redrawn from Newstead, 1991.*)

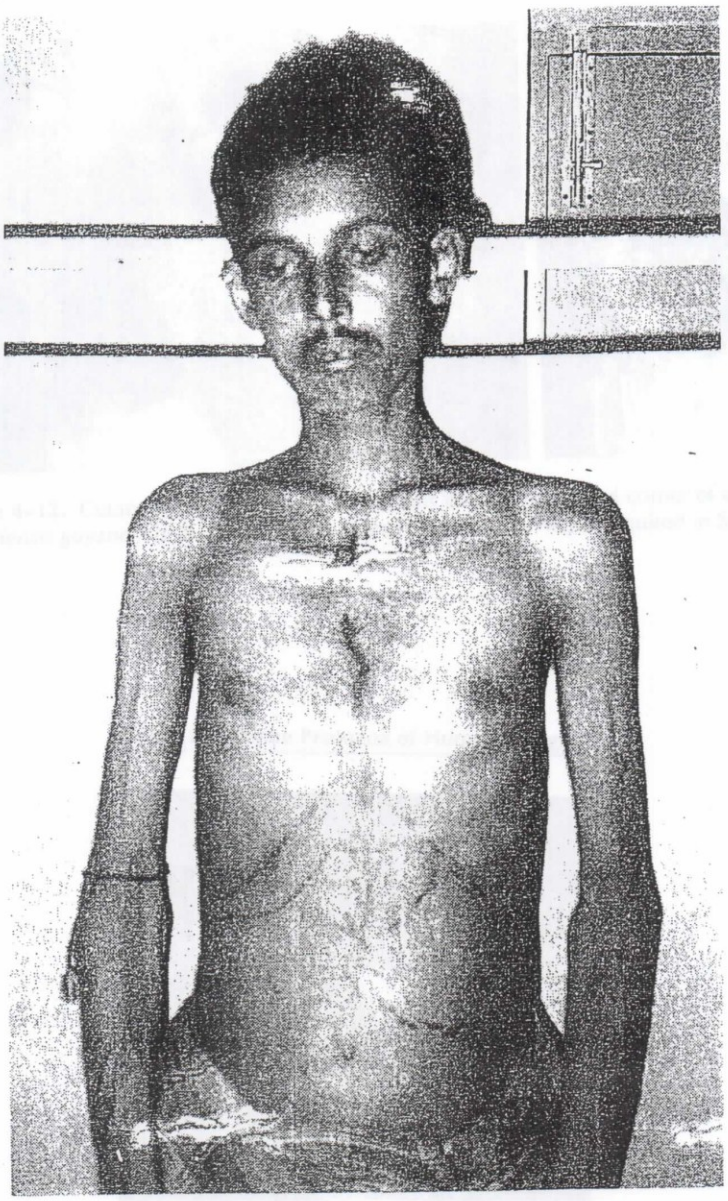

Figure 4–9. Indian patient with kala-azar. Enlarged liver and spleen are outlined. Note wasting of upper body.

Figure 4–12. Cutaneous leishmaniasis. A. Lesions of outer nose and corner of eye due to *L. braziliensis guyanensis*. B. "Wet" lesion of arm caused by *L. major* acquired in Senegal.

Figure 4–13. Mucocutaneous leishmaniasis in a Honduran female, probably due to *L. b. braziliensis* or *L. b. panamensis*.