Medical Committee
The University of Jordan

*The Skin and*
# MUSCULOSKELETAL
*System*

# MICROBIOLOGY

SLIDES ☐
SHEET ☐
LECTURE #

DOCTOR: Dr. Hassan
DONE BY:

Handout for half of lecture 3 (read the rest from sheet 3)

BY MOHAMED F. ABU ALIA

**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.