

D III (H) Leishmania Donovani (Life cycle) contd.
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Paper III The life cycle of *L. Donovani* is digenetic, requiring two hosts. The primary host is the man and the secondary host is the sand fly (*Phlebotomus argentipes*)

Amastigote forms multiply by simple binary fission and promastigote form by longitudinal binary fission. Sexual reproduction is unknown.

In Man: *Leishmania donovani* (amastigote form) divides rapidly by binary fission in the reticulo endothelial cells (macrophages) usually in the liver

and spleen. Now, when a sand fly sucks the blood of an infected person, numerous amastigote forms enter into the alimentary canal.

In sand fly: In the gut of the sand fly, the

amastigote form changes into promastigote form. Now it rapidly multiply by longitudinal binary fission in the gut producing large number of promastigote forms. These migrate forward in the anterior part of alimentary canal (fore gut). Heavy infection of fore gut is found after 6-9 days of the infected blood meal. This is called anterior station development.

Transmission Now, when such infected sand fly bites a man, promastigote forms enter into the blood circulation and some of them enter inside the cells of the reticulo endothelial system while others are destroyed. Here they change into amastigote forms and undergo multiplication.

Pathogenesis: *L. donovani* causes Kala-azar or Visceral leishmaniasis in man. The characteristics of the disease include high fever, darkening skin, enlarged spleen and liver. The harmful effects are due to blockage of the reticuloendothelial system and anaemia resulting from infection of bone-marrow. If the patients are not treated, 75% to 95% die within 2 years.

Treatment: Two groups of drugs are used in the treatment of "Kala azar".

1. Pentavalent antimony compounds like sodium-antimony-glucosate, stibamine, aminostiburea etc.
2. Synthetic non-metallic compounds like pentamidine isethionate.

Fig: Life cycle of *L. donovani*

