

# Leishmaniasis

# Introduction

- Protozoan flagellate of the genus *Leishmania* (order Kinetoplastida, family Trypanosomatidae)
- Infect numerous mammalian species, including humans
- Transmission: phlebotomine sandfly
- Threaten 350 million in 88 countries (tropics and subtropics)
- 1.5-2 million new cases per year with 12 million at any time
- Identification is made microscopically or by isoenzyme electrophoresis

# Epidemiology

## ■ Vector: phlebotomine sandfly



- 2 – 4 mm (small)
- rest in dark areas during the day, and are very active at dusk and throughout the night
- only the females require blood meals so as to be able to lay eggs
- breeding sites remain unknown, affecting means of sandfly control
- geographical distribution: tropics and subtropics worldwide

# Morphology

## ■ Dimorphic parasite: Amastigote and Promastigote

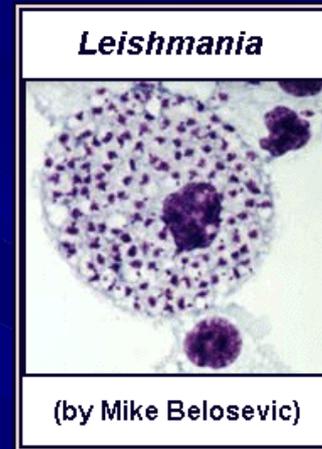
### Amastigote: round or oval body

- intracellular, within mononuclear phagocytic system of mammalian host
- contains nucleus, kinetoplast & internal flagellum
- multiply asexually within the vacuoles of macrophages

### Promastigote: long, slender body

- extracellular, within the intestinal tract of the insect vector
- central nucleus, kinetoplast, and long free anterior flagellum
- multiply asexually within intestinal tract of the sandfly

Amastigote



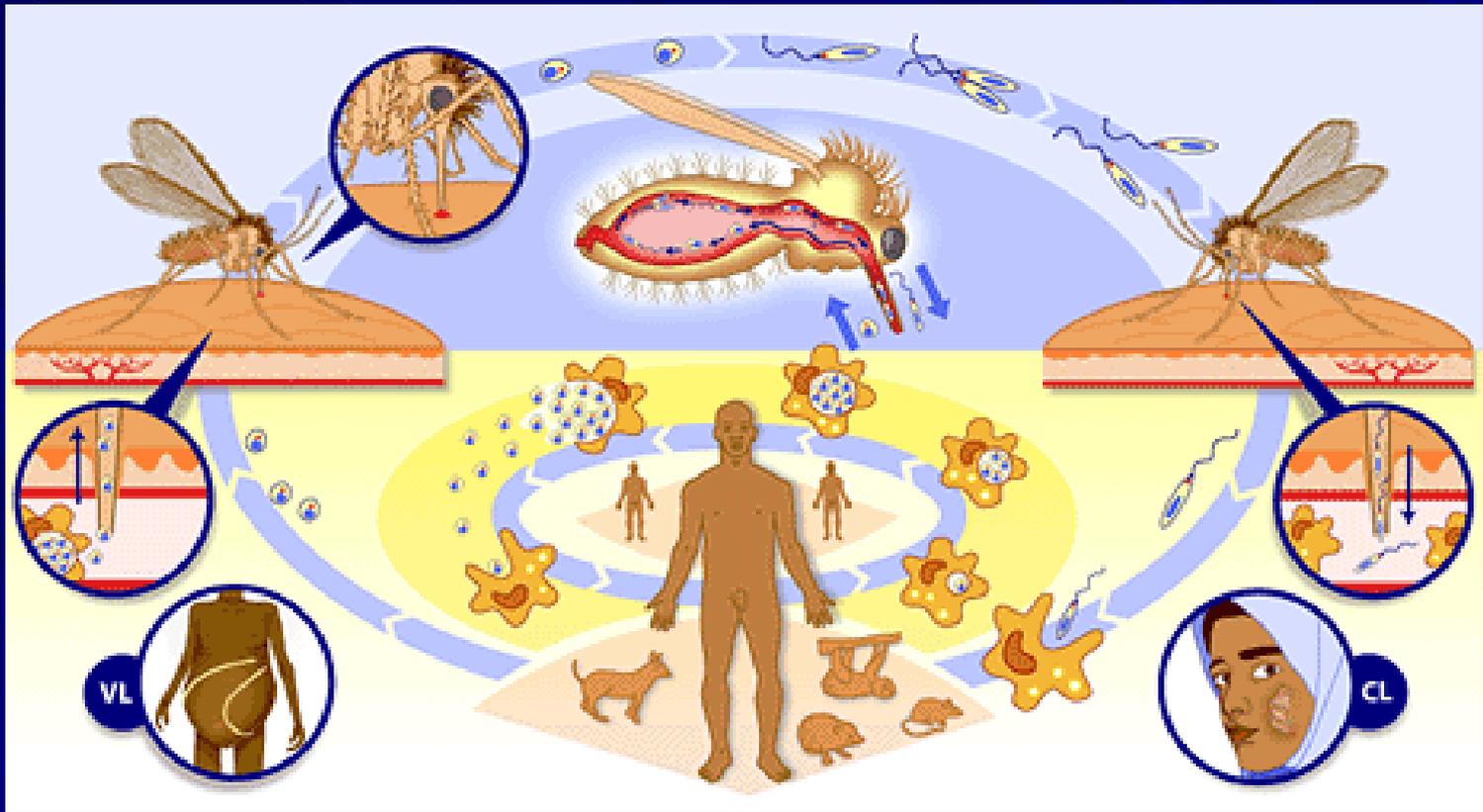
Promastigote



# Life Cycle

Alternatively hosted: insect and mammals (humans, dogs, rodents)

- Female sandfly takes a blood meal from infected mammal
- Intracellular amastigotes are ingested by the insect
- Amastigotes transform into promastigotes
  - trichomonads (free swimming) & hemomonads (attached)
- These promastigotes escape through the peritrophic membrane enveloping the blood meal and into the intestinal tract
- Intense multiplication
- Migrate to the anterior part of the midgut and become metacyclic promastigotes (infective form)
- The sandfly then bites and infects the vertebrate host
- Promastigotes are phagocytized by mononuclear phagocytes in tissue
- Promastigotes then change to amastigotes and multiply by mitosis inside the macrophages



# Other Transmissions: rare

- Congenital
- Syringes
- Sexual contact
- Contact of infected lesion with an open site

# Pathology

- Sandfly saliva enhances infectivity
  - causes vasodilation and immunosuppression of infected site
- Surface components lipophosphoglycan and glycoprotein enable *Leishmania* to escape complement activation
- Cutaneous Leishmaniasis
  - amastigote development remains localized at the inoculation site
  - cytokines released – results in swelling and lesions
- Visceral Leishmaniasis
  - amastigotes spread to the organs of the mononuclear phagocytic system (spleen, lymph nodes, liver, etc.)

# Clinical Features

# Visceral Leishmaniasis

- *L. donovani* & *L. infantum*
- Incubation period: 2-6 months, but can range from 10 days to 10 yrs
- Onset can be gradual or sudden
- Sudden: fever, immune system takes care of it
- Gradual:
  - protuberant abdomen
  - muscle wasting limbs
  - anaemia, weight loss
  - splenomegaly, hepatomegaly

# Visceral Leishmaniasis



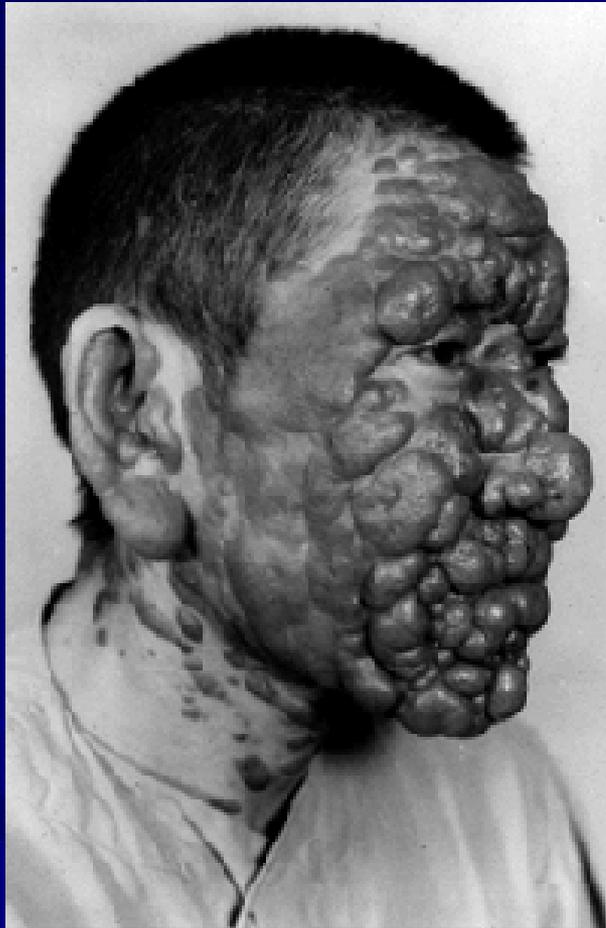
# Cutaneous Leishmaniasis

- *L. tropica*, *L. mexicana*, *L. braziliense*
- Localized skin lesions
- Starts as erythematous papule
  - ulcerative lesion (volcano appearance)
- Diffuse Cutaneous Leishmaniasis
  - large patches of nodules disseminated throughout body
- Mucocutaneous Leishmaniasis
  - primary cutaneous lesion, latent period, secondary mucosal involvement
  - starts in nasal mucosa
  - inflammation, necrosis, airway obstruction, etc.
  - death

# Cutaneous Leishmaniasis



# Cutaneous Leishmaniasis



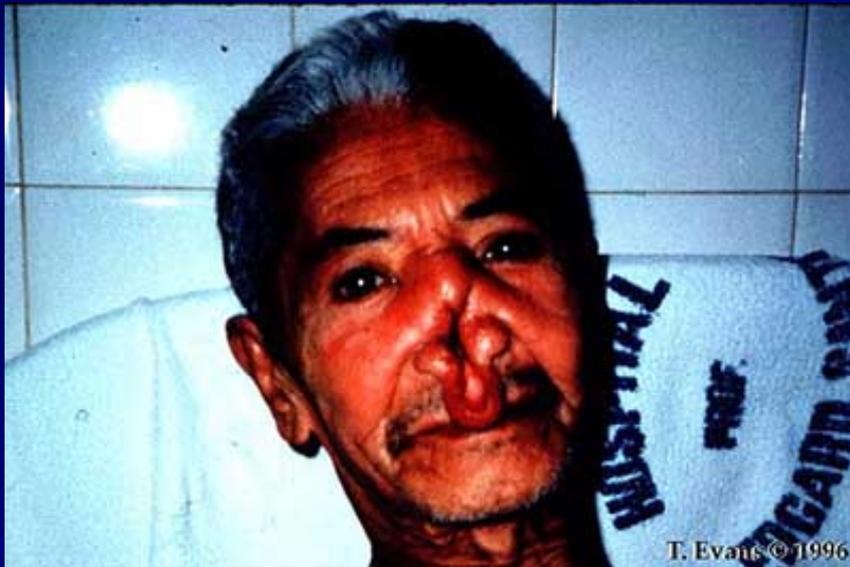
Cutaneous Leishmaniasis: I.  
T. Yamaguchi. Color Atlas  
of Clinical Parasitology, 1981.



# Cutaneous to Mucocutaneous Leishmaniasis



# Mucocutaneous Leishmaniasis



# Diagnosis & Treatment

- Detection of parasite: electrophoresis & microscopy
- Spleen, bone marrow and skin aspirations
- No significant treatments
  - becoming resistant to amphotericin B & antimonials
- Recently, miltefosine use is effective, but too new for long term prognosis
- Vaccine is being pursued

The End

# References

- Class Notes. Medical Microbiology Course.  
Weber State University, Dr. Harrington 2005
- Cook, Gordon C & Alimuddin Zumla. Manson's  
Tropical Diseases. Elsevier Science. 2004