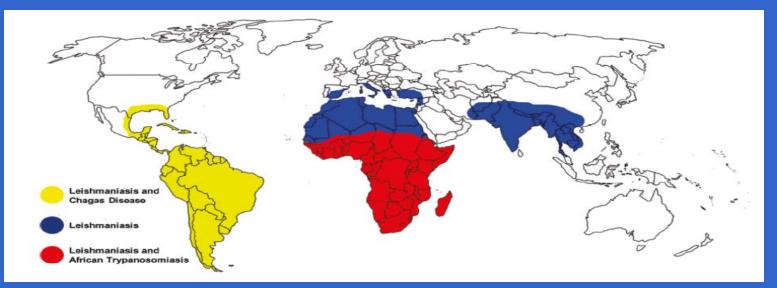
The neglected protozoan infections of tropical region: an update

Kallesh Danappa Jayappa, Ph.D student Infectious minds presentation 13th Oct 2011

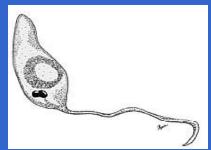
The neglected protozoan infections

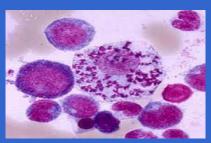


disease	global prevalence	population at risk	regions of highest prevalence	deaths	clinical manifestations and associated disabilities	primary drugs	weakness of current drugs
Chagas disease	8–9 million	25 million	Latin America and Caribbean	14000	cardiomyopathy, megacolon, megaesophagus	Nifurtimox (4), benznidazole (5)	poor efficacy and toxicity
human African trypanosomia- sis	300 000	60 million	sub-Saharan Africa	48 000	sleeping sickness	Suramin (32), pentamidine (33), melarsoprol (34), eflornithine (35)	drug toxicity and drug resistance
leishmaniasis	12 million	350 million	India, South Asia, sub-Saharan Africa, Latin America, Caribbean, and Mediterranean area	51 000	cutaneous and mucocutaneous disease, kala-azar	Antimonials (39, 40), amphotericin B (41), pentamidine (33), miltefosine (42), paromomycin (43)	drug toxicity and drug resistance

(Cavalli et al., 2009)

Leishmaniasis





(Leishmania *sp*)



Clinical presentation	Pathogen	Region	
	L.d. donovani	China, India, Iran, Sudan, Kenya, Ethiopia	
Visceral leishmaniasis (kala-azar or dumdum fever)	L.d. infantum	Mediterranean countries	
	L.d. chagasi	Brazil, Columbia, Venezuela, Argentina	
	L. tropica	Mediterranean countries, Afghanistan	
Cutaneous leishmaniasis (oriental sore or tropical sore,	L. major	Middle East, Western and Northern Africa, Kenya	
uta ulcer or chiclero ulcer or Aleppo boil)	L. aethiopica	Ethiopia	
	L. mexicana	Central America, Amazon regions	
Mucocutaneous leishmania- sis (espundia)	Lbraziliensis complex	Brazil, Peru, Ecuador, Columbia, Venezuela	

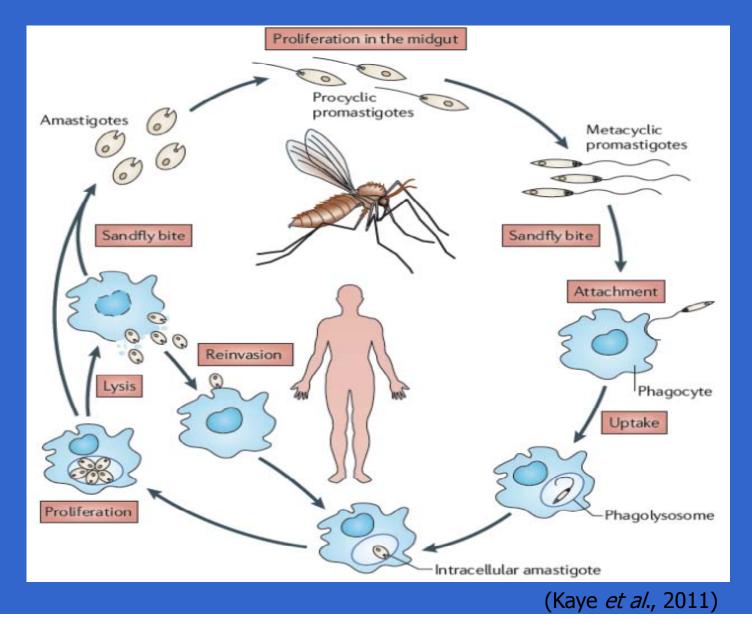




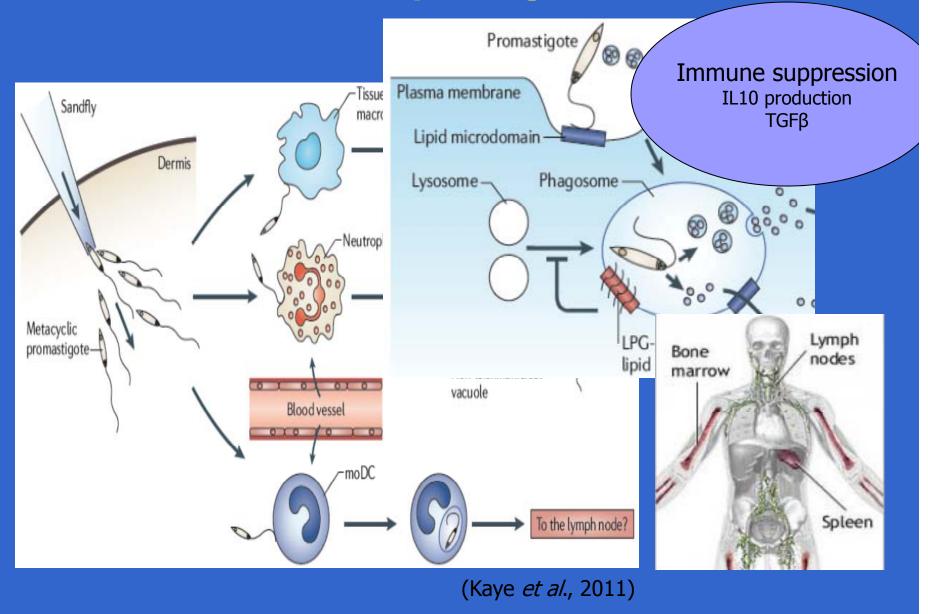


(Google images/Neuber.,2008)

Leishmania life cycle



Leishmaniasis pathogenesis



Post kala azar dermal leishmaniasis (PKADL)



(Ganguly *et al.*, 2010)



(Zijlstra *et al.,*2003)

- Post visceral leishmaniasis complication
- Mostly seen in India and Sudan
- India- generally seen after 2-3 years of VL
- Sudan- onset is much quicker (avr 6 months)
- Serve as a potential source for infection
- No standard markers for PKDL prediction

Leishmania diagnosis





Skin smear Bone marrow aspiration

• CL: Skin smear and tissue explant biopsy examination

• VL: Bone marrow punch biopsy examination and serological tests (ELISA, IFT)

Treatment and prevention

- VL: Pentavalent antimonials, Miltefosine and Paramomycin
- CL: Paramomycin oinment
- Sand fly control, mosquito nets, insecticide treatment of dogs
- No effective vaccine available

(Google images)

American tryphanosomiasis (chagas disease)







(Rhodnius prolixus)

Acute phase



(Chagoma)



(Romana)

Chronic phase

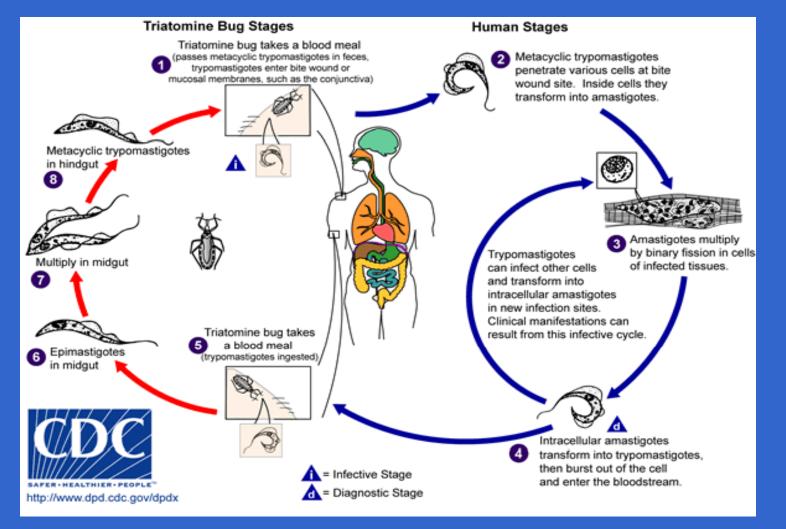


(Cardiomegaly)



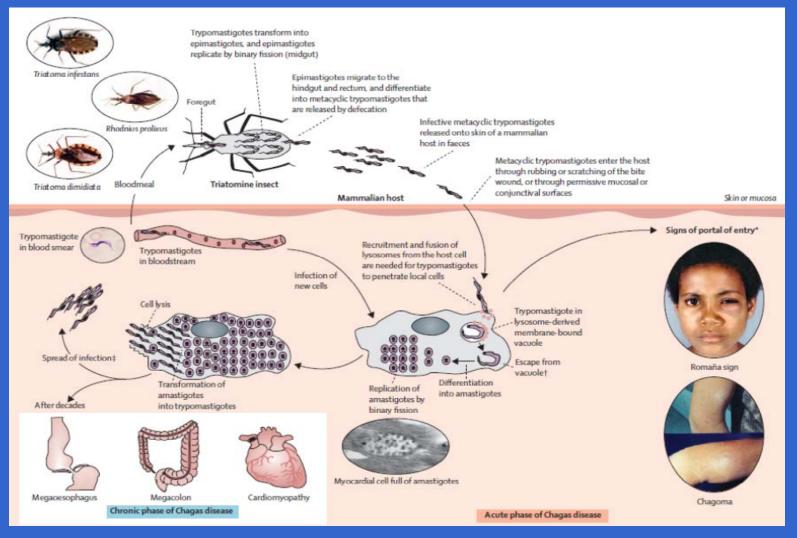
(Megaesophagus/colon) Google images/ Rassi *et al.*,2010)

Life cycle of Tryphanosoma cruzi



(Google images)

Chagas disease pathogenesis



(Rassi et al., 2010)

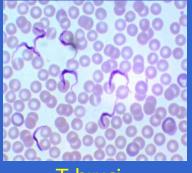
Diagnosis

- Acute phase: blood smear, buffy coat and serum precipitate examination
- Congenital infections: Cord blood examination, IgG serodiagnosis
- Chronic phase: Serodiagnosis, PCR and clinical examination

Treatment and prevention

- Anti-tryphanosomal treatment is recommended for all forms of infection
- Benznidazole and nifurtimox are every effective againest T cruzi
- Prevention: vector control and elimination of non-vector mediated spread

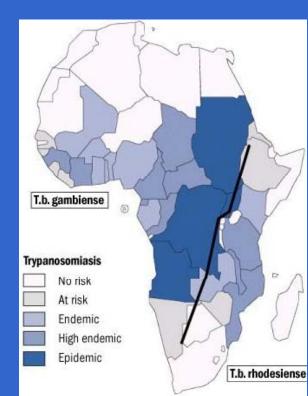
African tryphanosomiasis (sleeping sickness)



T bruci







Haemolymphatic phase



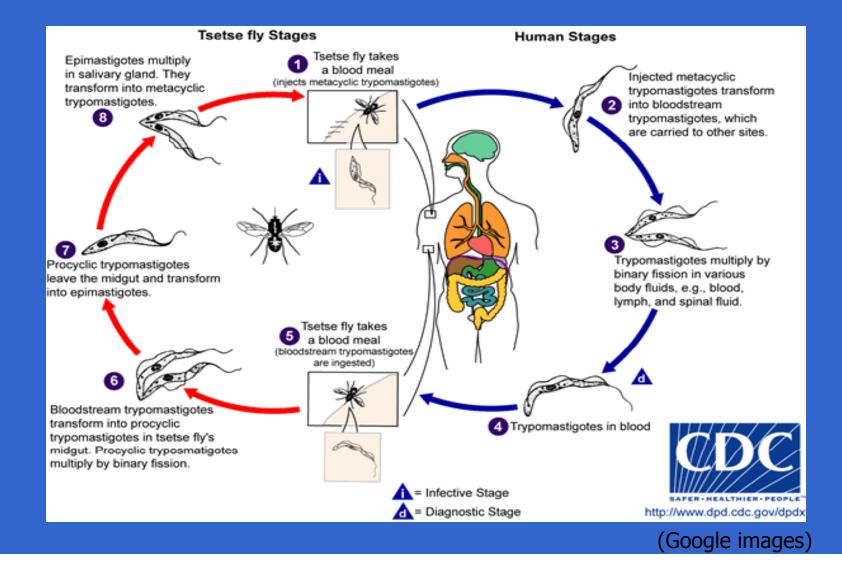


Chancre Winterbottom's sign Neurologhical phase

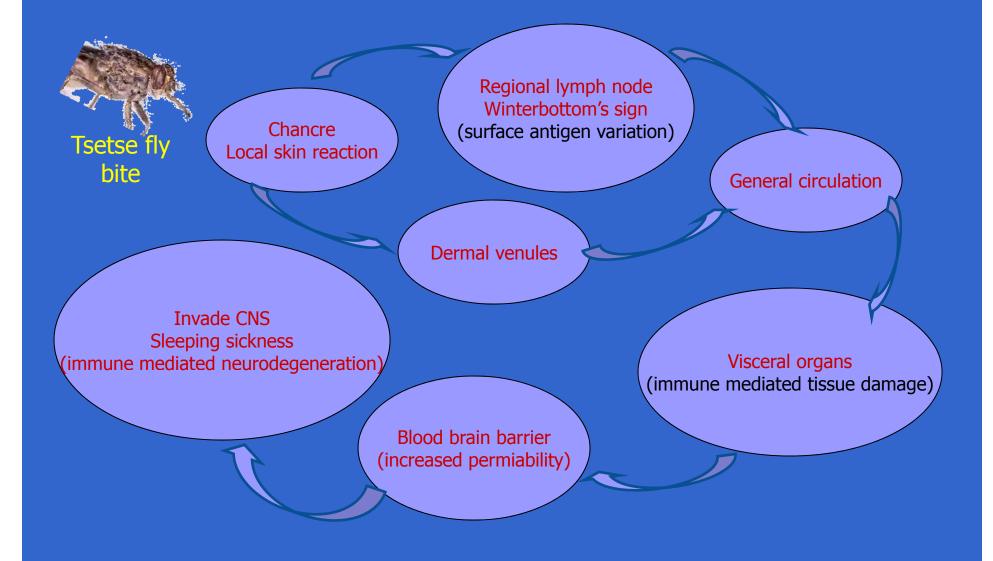


(Google images)

Life cycle of Tryphanosoma bruci



Sleeping sickness pathogenesis



Diagnosis

- Card Agglutination Test for Trypanosomiasis (CATT) for antibody
- Card Indirect Agglutination Test (CIATT) tests for antigens
- Microscopic examination of blood smear, lymph node aspiration
- Examination of CSF for white blood count and tryphanosoma organism

Treatment and prevention

- Haemolymphatic phase: treated with Pentamidine and Suramin
- Neuronal phase: treated by Melarsoprol, effornithine and nifurtimox/ effornithine combination
- WHO: programme for surveillance and control of african trypahnosomiasis
- Tsetse fly control: poisoning and draining water bodies

HIV and protozoal parasite co-infection

- HIV co-infection: disease incidence, prevalence and pathogenesis
- HIV/leishmania co-infection: altered visceral leishmaniasis prevalence
- HIV/leishmania co-infection: influences disease transmission
- HAART: reduced new infections but fail to eliminate relapses
- The HIV and leishmania infections are mutually beneficial
- Altered disease pathogenesis: T cruzi/HIV co-infection

Summary

- Leishmania and tryphanosoma: endemic in developing and under developing countries, affecting large population size
- Leishmania infection: transmitted by plebotomus flies, exhibits cutaneous, visceral and muco-cutaneous forms
- Post kala azar dermal leishmaniasis: not fatal but involves serious public health concern
- Chagas disease: transmitted by bug, characterized by acute self-limiting infection and a chronic infection affecting heart and digestive organs
- Sleeping sickness: transmitted by tse-tse fly, infection involves haemolym phatic and neuronal phases
- HIV-1 co-infection: seen in both endemic and non-endemic countries, leads to altered disease pattern, proves fatal if not managed well

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