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Life Cycle of *Leishmania Donovani*; causative agent of visceral leishmaniasis : A Review

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ABSTRACT

Leishmaniasis is transmitted by the bite of infected female phlebotomine sandflies. The sandflies inject the infective stage (i.e., promastigotes) from their proboscis during blood meals. Promastigotes that reach the puncture wound are phagocytized by macrophages and other types of mononuclear phagocytic cells. Promastigotes transform in these cells into the tissue stage of the parasite (i.e., amastigotes) , which multiply by simple division and proceed to infect other mononuclear phagocytic cells. Parasite, host, and other factors affect whether the infection becomes symptomatic and whether cutaneous or visceral leishmaniasis results. Sandflies become infected by ingesting infected cells during blood meals. In sandflies, amastigotes transform into promastigotes, develop in the gut and migrate to the proboscis.

Keywords: Phlebotomine, Sandflies, Amastigotes, Promastigotes, Leishmaniasis

I. INTRODUCTION

Visceral leishmaniasis (VL) is a vector-borne disease caused by replication of parasites in macrophages, mononuclear phagocytic system. The *Leishmania* parasite has a digenetic complex life cycle with an extracellular developmental stage in the insect vector, a female phlebotomine sandfly (Lievin-Le and Loiseau, 2015) and a developmental stage in mammals, which is mostly intracellular. The parasite undergoes major morphological and physiological transformations during the different stages of its life cycle. Phlebotomine female sandflies are dipteran insects within the family of Psychodidae. The transmission of leishmaniasis is attributed to about 70 of around 1000 known sandfly species Those belonging to the genus Lutzomyia are prevalent in the New World and those belonging to the genus Phlebotomus are prevalent in the Old World. Phlebotomine sandflies are not active during the day and seek out cool and relatively humid dark niches which allow them to survive in hot and dry climates. Indeed, they become active at dusk when the temperature drops and humidity rises (Murray et al., 2005).

Leishmania parasites need female phlebotomine sand flies not only to complete their life cycle but also to propagate i.e. it acts as a vector. There are many adaptations like secretion of phosphoglycans, which protect the parasite from digestive enzymes; production of chitinases that degrade the stomodeal valve of the sand fly; secretion of a neuropeptide \that arrests midgut and hindgut peristalsis; and attaching to the midgut to avoid expulsion (Kamhawi, 2006). In sandflies, development of the parasite occurs in the alimentary canal with the formation of a motile, flagellated and elongated form termed as promastigote. The promastigote multiplies and matures in the insect midgut into an infective metacyclic form which migrates to its proboscis. When sandflies bite mammalian host for blood meal the metacyclic promastigotes reach the punctured wound (Tripathi *et al*., 2007). A typical inoculum contains around 100-1000 metacyclic promastigotes. Once inside the host they quickly get phagocytised by leucocytes, particularly macrophages, neutrophils and dendritic cells. The parasites which are internalized by these cells through phagocytosis are trafficked to endosomes and lysosomes. The resulting phagolysosome, also called parasitophorous vacuole (PV), is an acidified and hydrolytically active compartment where the *Leishmania* parasite is not only capable to survive but also to multiply (Antoine *et al*., 1998). Then they immediately undergo a further morphological change by shedding its flagellum and taking on an ovoid shape hence termed ‘amastigote’ and possibly a metabolic change with a switch to anaerobic metabolism under acidic conditions found chiefly in the

phagolysosome compartment. The parasite's persistence in such a hostile environment is attributed to its differentiation in amastigotes, which resist to macrophage hydrolases (Desjardins and Descoteaux, 1997). Phagocytic cells are ruptured by amastigotes to further infect other cells, thus eventually damaging the whole reticuloendothelial system and to sustain the development.

II. REFERENCES

[1] Antoine, J. C., Prina, E., Lang, T. and Courret, N. (1998). The biogenesis and properties of the parasitophorous vacuoles that harbour Leishmania in murine macrophages. Trends Microbiol. 6, 392-401.

[2] Desjardins, M. and Descoteaux, A. (1997). Inhibition of phagolysosomal biogenesis by the Leishmania lipophosphoglycan. J. Exp. Med. 185(12): 2061–2068.

[3] Kamhawi, S. (2006). Phlebotomine sand flies and Leishmania parasites: friends or foes? Trends Parasitol. 22(9): 439-445.

[4] Lievin-Le Moal V, Loiseau PM. 2015. Leishmania hijacking of the macrophage intracellular compartments. FEBS J. doi: 10.1111/febs.13601. Epub ahead of print

[5] Murray, H.W., Berman, J.D., Davies, C.R. and Saravia, N.G. (2005). Advances in leishmaniasis. Lancet 366(9496):1561-1577.

[6] Tripathi, P., Singh, V. and Naik, S. (2007). Immune response to Leishmania, paradox rather than paradigm. FEMS Immunol. Med. Microbiol. 51(2): 229-242.