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Research Article

Mechanisms Involved in the Arthropod Transmission of Leishmania *spp.*

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Abstract

Due to the great complexity of the transmission of Leishmania spp parasites. Due to its arthropod vectors and the general need to gather the existing information in precise and rigorously commented writings, this document was prepared, based on the consultation of scientific articles of proven methodological quality and with reproducible results, focused on vector-parasite interactions and parasite-host cell as a tool for the adjustment or design of government health programs aimed at hindering or interrupting transmission and thereby controlling American tegumentary and visceral leishmaniasis.

EPIDEMIOLOGICAL ASPECTS IN THE TRANSMISSION OF LEISHMANIASIS

The burden of global morbidity and mortality is greatly contributed to by vector-borne diseases, in addition to being a main factor in perpetuating economic inequality [1]. Vector-borne diseases have as a great ally the accelerated pace of growth of environmental problems (especially the deforestation caused by urbanization and the increase in agriculture and mining, and with it the redistribution of reservoirs and vectors with the promotion of greater proximity between all those involved in parasitic transmission) [2], as they contribute to the emergence and resurgence of these pathologies mainly in tropical and subtropical geographic areas, where many countries have low economic income, and therefore serious difficulties in investing in the control of such severe health scourges [3,4].

Parasitic infections transmitted by arthropods and insects are favored in their incidence and prevalence in tropical and subtropical jungle ecosystems by geographic factors (with varied biotypes as a result of rapid differentiation towards peripheral refuges due to the multiplication of jungle plant species) [5], biological (of the parasite, insect, reservoirs and susceptible), social and economic that characterize them and that favor contact between the human being and the transmitter, for example, in leishmaniasis (it is accepted that these protozoa are originally parasites of secondarily adapted arthropods to vertebrates: first to the lower ones and more recently to mammals), where human interventions in the geographical environment such as deforestation, urbanization, migration and urban marginality drive the evolution towards the origin of new biocenoses towards the domicile and peridomicile; and where there is also a lack of specific control strategies such as those designed for other parasitic diseases that can reduce the number of cases of infection, since in the insect vector the transmission dynamics are not completely clarified [6].

ARTHROPOD TRANSMITTER AND ETIOLOGICAL AGENT OF LEISHMANIASIS

The causative agents of leishmaniasis are protozoa of the genus *Leishmania* spp., *Trypanosomatid* parasites [7]. They are protozoa that present unusual biological characteristics (such as post-transcriptional regulation of gene expression) and are divergent (with two main forms) [8], whose life cycle includes an insect vector and a susceptible vertebrate host, in the digestive tract of the first the *Leishmania* spp. it replicates as procyclic promastigote (motile flagellated extracellular forms of the insect midgut, non-infective for vertebrates) and as metacyclic promastigotes (in the thoracic midgut and in the proboscis) without the ability to divide, but infective for mammalian vertebrates when they are inoculated through the insect bite, metacyclic promastigotes differentiate into intracellular aflagellate amastigotes (proliferate by binary division) after they are phagocytosed by macrophages [9]. Hence the complexity of the vector transmission process of *Leishmania* spp. [6,10,11].

Therefore, it is convenient to evaluate the transmission

capacity after human intervention in the natural habitat of the binomial insect vector (*Lutzomyia* spp. in the new world and *Phlebotomus* spp. in the old world) and *Leishmania* spp. in relation to human housing (structures with little or extensive exposure to the surrounding environment), primary forest (distance between it and the home), parasite dispersal and adaptation phenomenon (active or passive contamination and inoculation) [6,12], blood preference of the insect (degree of anthropophilia, linked to the trophic relationship between the infected vector and the healthy host to be infected) and the role of a species as a vector (primary or secondary) this in direct relation to the preference for sugar in the diet, abundance and susceptibility physiological to the parasites, the physiological age and parity (nulliparous and parous, the latter have greater epidemiological value than the crude infection rate of the total vector population examined) [13].

TRANSMISSION MECHANISM OF LEISHMANIA SPP. BY ITS TRANSMITTING ARTHROPOD

In relation to the physiological susceptibility of the vector to *Leishmania* spp. parasites, that is, the vector-parasite specificity, the general biology and molecular environment of the intestine of the vector, the frequency of blood feeding, the period of the gonadotrophic cycle must be considered in relation to the development cycle of the parasite and the temperature at which the vector is maintained [14]. Regarding the molecular environment of the vector's intestine, it is crucial that the parasite remains attached to the intestinal wall to avoid being evacuated along with the peritrophic membrane after processing the blood intake [15]; for this, the terminal sugars of the surface molecule of cellular lipophosphoglycan from the surface of the protozoan with lectins from the vector intestine [16].

It is presumed that the success in the transmission of *Leishmania* spp. it is conditioned in the intestine of the vector by the type of sugar ingested (it can interfere with the attachment of the parasite to the intestinal wall), for example, *Lu logipalpis* ingests sucrose and *Lu evansi* fructose, with *Lu longipalpis* being a better transmitter of *Leishmania* than *Lu. Evansi* [15,16]. The ingestion behavior of the vector also plays a role in transmission; in this sense, it is believed that the parasite causes the insect to increase the frequency of bites on the host's skin, thereby increasing the probability of transmission [17]. Another key element in the transmission of *Leishmania* spp. it is the vector's saliva, since it has been described that its inoculum with protozoa enhances parasitic infectivity (allows parasite density to increase), because it has anticoagulant substances. In this same order of ideas, salivary secretions with a lower concentration of erythema-inducing peptide, specifically maxidilan, contribute enormously to the success of the infection [18,19].

Now, from the point of view of the *Leishmania* spp. parasite it is likely that its colonization modifies the fluid mechanoreceptors present in the cibarium and proboscis of the vector to facilitate its transmission. The progress of parasite transmission of *Leishmania* spp. seems to be favored, in the host, by the temperature of the skin, that is, temperatures equal to or lower than 29°C (detected in lower limbs, areas of the body where the highest number of cases of skin lesions caused by *Leishmania* spp.) substantially increase the probability of infection, because the complement sees its biological activity (cytolysis of promastigotes) reduced as a host defense mechanism against this protozoan [6,20].

CONCLUSION

In conclusion or summary the changing environmental conditions within the invertebrate and vertebrate host determine the parasite *Leishmania* spp. its different morphological forms and the success in fulfilling its life cycle, thus there are several membrane effectors attached or released extracellularly that allow survival and transmission [21]; Just as the social behavior of human beings also intervenes in the maintenance of the life cycle of this protozoan, therefore [22], the information provided in this document is recommended for the design of health programs to control such an important public health problem [23].

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