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Commentary Open Access

Parasite Lectins: More than Adhesion Molecules?

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Commentary

Several studies demonstrate the involvement of lectins in the recognition of carbohydrates present on the surface of different kinds of pathogens [1-3], including parasites of the genus *Leishmania* [4,5]. The recognition often involves the modulation of the host immune response due to the activation of signaling pathways initiated by the stimulation of lectins present on the surface of cells from immune system by carbohydrates from pathogens [1,2]. On the other hand, lectins from microorganisms are also involved in infections: Heparin-Binding Protein (HBP) from *Trypanosoma cruzi* are involved in processes of amastigote and epimastigotes adhesion to the mammalian host cells and to the intestinal epithelium of insects, respectively [6-8]. Furthermore, galactose/N-acetylgalactosamine (Gal/GalNAc) lectin is involved in the infection by *Entamoeba histolytica*, responsible for the third highest number of death from parasitic diseases in the world [9-11]. Other relevant lectins from parasites are involved in mediating protozoa attachment to the host cells, acting as valuable tools to study pathogenesis of infection: mannose lectin (MBP) from *Acanthamoeba*, causative agent of keratitis, mediates parasite adhesion to the host cells, and may serve as a marker of pathogenicity of this parasite; micronemal protein (MIC1), a *Toxoplasma gondii* adhesin, bind to host sialic acid moieties, playing role in the parasite invasion and virulence; *Tritrichomonas foetus* lectin (TFL), a sialic acid specific lectin, is involved in mucosal surface attachment and immunogenicity of *Tritrichomonas foetus*, a protozoan parasite of the bovine urogenital tract; and *Cryptosporidium parvum Clec* (CpClec), a novel mucin-like glycoprotein with a C-type lectin domain (CTLD), is involved in *Cryptosporidium*-host cell interactions [12].

Unlike what is observed for lectins present in the vertebrate host, plants or microorganisms, the number of works referring to lectins expression on the surface of *Leishmania* parasites are limited [13,14], especially with regard to the role of these molecules in cell adhesion and their influence on host immune response. In this regard, it was found the presence of HBP on *L. braziliensis* promastigotes, suggesting a role of HBP in the interaction of the parasite with the intestinal cells of the insect (*Lutzomyia*) [15-17].

Recently, our research group showed the presence of HBP in promastigotes of *L. infantum chagasi* using Fast Protein Liquid Chromatography (FPLC) with heparin column, providing the basis for further studies on the biological actions of HBP in visceral infection [18]. In this study, HBP was identified on the external membrane of the parasite and also present in the cytoplasm and cytoskeleton, in vesicles, and close to the kinetoplast of the parasite. Furthermore, blocking of HBP from *L. infantum chagasi* by heparin partially prevented the adhesion and internalization of parasites in RAW 264.7 macrophages; however, it is possible that parasite lectins not only participate in adhesion, as the heparin interaction with extracellular HBP from *L. donovani* leads to inhibition of protein kinase C [19]. This evidence, together with the existence of intracellular HBP in *L. infantum chagasi* demonstrated by our research group indicate that lectins can be more than adhesion molecules, possibly participating in intracellular metabolism reactions dependent on specific molecular recognition. Thus, the function of these molecules acting inside the parasite needs to be studied in more details.

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