Role of Lectins in Interaction Between Parasites and the Important Insect Vectors

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ABSTRACT

There is growing evidence that lectin-carbohydrate interactions can mediate the infection of parasites to their insect vector. Many insect species are host or vectors of protozoan or metazoan parasites that cause socially and economically important disease such as malaria and leishmaniasis. However, relatively little work has been undertaken concerning the interaction of insect immunity against parasite invasion with respect to lectins activities. Both immune defences (cellular and noncellular) of insect haemolymph react in order to combat the diverse array of natural pathogens and other microorganisms. The most of immune substances are innate, naturally-occurring and nonspecific molecules present in haemolymph. When the physical defences of the insect gut or integument are breached by an invading organism an innate response begins, characterized by immune system's agents such as coagulation, melanization, phagocytosis, encapsulation and nodule formation. Nevertheless, in many cell types such as insect haemocytes, carbohydrates are known to be crucially involved in cell-cell interactions and many studies have addressed the role of carbohydrates and carbohydrate-binding molecules in the adhesion of parasites to their host. As mentioned above, one candidate for attachment and invasion may be lectins or lectin-like molecules that are known to mediate cell-to-cell interaction. In order to the basic understanding of pathogens transmission by vectors, in this article, the interaction between parasites and insect vectors has been reviewed with respect to role of lectins molecules.

INTRODUCTION

Several authors suggest that agglutinin and/or lectins of different insects are important for both the establishment of infection and the development of the parasites in the gut and/or haemolymph of the vectors (14, 3, 15,31).

Lectins are defined as carbohydrate-binding proteins or glycoproteins of non-immune origin or as carbohydrate-binding proteins other than antibodies or enzymes (23). By definition, lectins are polyvalent, oligomeric, nonimmunoglobulin that bind carbohydrate, agglutinate cells (e.g., RBC, bacteria and viruses) or precipitate polysaccharides, glycoproteins or other glycoconjugates (49, 24).

The sources of lectins in nature are not only plants, but also viruses, bacteria, fungi, parasites, invertebrates and vertebrates (24). To detect lectin interactions, the lectin can be conjugated with various labels such as fluorescein (FITC and TRITC), enzymes (horseradish peroxidase), biotin (for binding to avidin), radioactive isotopes and colloidal gold (23). Lectins can also be used directly in agglutination assays or for purification and analysis of glycoconjugates on lectin-sorbents (33). Therefore, lectins have been employed to describe surface carbohydrate of various insect cells and tissues such as haemocytes (43), gut surface (44, 11), and salivary glands (37, 34, 2).

Lectins also probably function as determinants of pathogen transmission by arthropods (11). Thus, they have been reported to be involved in insect-parasite interactions (36, 15, 19, 12).

Generally, most parasites such as *Leishmania* and *Plasmodium* use specific receptor-ligand interaction for the purposes of adhesion

and migration within the vector (19), and it is likely that similar interactions are used by trypanosomes and other protozoan parasites (15). However, in many insects, the precise nature of the receptor molecules that effect parasite tropism is not clear.

Insect Lectins

In insects, lectins serve a variety of functions, principally, non-self recognition (15), and also they are involved in phagocytosis, encapsulation, melanization and clotting (11). Thus, lectins with distinct sugar specificities involve in recognition and protective roles in immune defence against microbial pathogens protozoans (4).

The first milestone paper on tissue specific lectin of a vector was published on the kissing bug *Rhodnius prolixus* by Pereira *et al.* (1981). However, most results on vector lectins have come from research on tsetse flies and have shown that tissue specific lectins might play the crucial role in control of tsetse fly infection by African trypanosomes (26, 27). There are evidences indicating that the biosynthesis of insect lectins take place mainly in the fat body although agglutinins are also reported to be produced by the sex organs (50), haemocytes (1) and in some cases are associated with cell membrane (5, 25).

Several studies on insect haemolymph lectins showed that these molecules are heterogenous and exhibit a degree of

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ultispecificity (base on carbohydrate inhibition studies) in their sugar reactivity the RBC surface (15, 41).

Generally, the physiological functions of the insect lectins and their interactions with transmitted pathogens have been described as: (a) regulation of differentiation processes and morphogenesis, (b) self and non-self recognition in immune and defence reactions (42), (c) regulatory role of vector infections (refractoriness or susceptibility) by transmitted pathogen or parasites, killing factors (26), (d) differentiation factors of a vector-specific developmental stage of the pathogen (59, 22, 30).

Lectins in Parasites

Lectins play a role in attachment of some parasites to the host cells, e.g. a lectin was found in *Entamoeba histolytica* which mediates in adhesion of trophozoites to monolayers of human cells (6). The other possible function of lectins in protozoans include binding to red blood cells by *Plasmodium falciparum* (48), cell-to-cell interaction with *Cryptosporidium parvum* (54) and determination of the particular site of infection with *Eimeria* species (33). It has been found that a lectin-like component with broader specificity on the surface of *L. amazonensis* promastigotes (13)

It has been suggested that merozoites of *P. falciparum* have lectin-like proteins on their surfaces that bound to red blood cell carbohydrate receptors (10). The parasite invasion *in vitro* could be specifically blocked by GlcNAc, GalNAc and NeuNAc. Also, an assay based on resetting of erythrocytes was found to be blood-group specific, with high affinity of rosette binding to groups A,B and AB (GalNAc and galactose terminal saccharide). Thus, it is suggested that the relative protection of belonging to the O blood group against malaria may be explained by this lectin interaction (13).

Mosquito-Parasites/Pathogens Interaction

Mosquitoes are vector of disease such as malaria, filariases and also the most important vector of arboviruses. In *Aedes aegypti*, a diversity in carbohydrates moieties on the basal membrane surface of the salivary gland have been demonstrated which may be related to the invasion of these glands by sporozoites (37).

The midgut glycoproteins of the malaria vector *A. tessellates* were partially characterized by gel electrophoresis and lectin binding (39). These glycoproteins are specific for WGA and Con A indicating the presence of N-linked core oligosaccharides in many proteins. Chitotrios added to a bloodmeal also inhibited parasite development in the mosquito which may indicate GlcNAc residues in mosquito midgut glycoproteins and/or midgut chitin and proteoglycan function as recognition sites for malaria parasites (39). Recently, the complex mixture of oligosaccharide expressed on the midgut of the mosquito, *An. stephensi* were characterized by lectin blotting and by digestion with a range of endo- and exo-glycosidases (62).

It has been shown the specificity of the interaction between sporozoites of malarial parasites and the salivary glands of mosquitoes by implanting the salivary in susceptible and nonsusceptible mosquitoes (39). Specific lectins were used to block *Plasmodium gallinaceum* sporozoites from invading the salivary glands of *Aedes aegypti*, suggesting that glycosylated surface molecules on the basal lamina of female salivary glands may serve as receptors, for invasion by malarial sporozoites (4). This study also showed on the overall glycoprotein characteristics of the male and female salivary glands of *Ae. aegypti* and identified glycoproteins, which appear to be specific to the female salivary glands and to block *P. gallinaceum* sporozoite invasion. In this study, it has been also showed that Soy bean agglutinin (SBA) inhibited the *Plasmodium gallinaceum* invasion into the salivary gland of *Aedes aegypti* while *Dolichos biflorus* agglutinin (DBA) did not (4) and both lectins nominally recognise GalNAc. Also inhibition sugars in *Ae. aegypti* increase the infection rates and migration of *Brugia pahangi* microfilariae (5). This enhancement was greater for the refractory strain of *Ae. aegypti.* It was postulated that the sugars act by blocking gut and/or peritrophic matrix carbohydrate binding proteins such as lectins or lectin-like molecules, which would normally inhibit microfilariae migration However for the most part the role of lectins and carbohydrate in mosquito with respect to pathogen/parasites interaction remains undefined.

Kissing Bugs and Trypanosomatid Interaction

Although many triatomine species are able to transmit the disease, most of the knowledge on the vector-parasites interactions with respects to the possible role of lectins or lectins-like molecules has been derived from few model species, mostly *Rhodnius prolixus* and *Triatoma infestans.* However, lectin activities have been detected in various tissues such as haemolymph, crop and midgut of kissing bugs (36, 3, 41, 30). The agglutination of midgut lectins with *T. cruzi* epimastigotes also indicated that the possible role of gut lectins in interaction with the parasite (30). There is evidences show these compounds involved in *T. cruzi* development. Furthermore, it has been shown that the binding capacity of the gut lectin was species and even stage specific. While epimastigotes of *T. cruzi* were efficiently agglutinated, no agglutination was occurred with trypomastigotes of the same species nor with some other protozoans, which are not natural inhabitance of the bugs.

It has been shown that the adhesion of some trypanosomes to the cell surface of the host, is mediated by lectins or lectin-like molecules (63, 7). In addition, with the American trypanosome, *T. cruzi,* invasion is activated by the enzyme, trans-sialidase, which transfers sialic acid between host glycoconjugates and the parasites (45).

In a study of *T. rangeli* in *R. prolixus*, it has been found that forms of the pathogen in the salivary glands differed from those in other tissues by reacting with a specific lectin (31). The

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results indicating that the membrane of *T. rangeli* in the salivary glands of the vector contains β-D-galactose, but this sugar is absent from all other developmental stages of this trypanosome.
The lectin in haemolymph of *R. prolixus* enhances the development of *T. rangeli* in the haemolymph of the vector (41,31). It has been suggested that lectins could influence the development of *T. cruzi* in triatomine bugs (36).

Sandfly and Leishmania Interaction

Lectins appear to play an important role in sandfly/Leishmania interaction, for example galactosamine increase the rates of *Leishmania major* infection in its vectors, indicating the inhibition of a lectin or lectin-like molecule (57). The authors also cited that a bloodmeal containing a carbohydrate inhibitor for the gut lectin, galactosamine, significantly increased *Phletobomus duboscqi* susceptibility to infection of *Leismania major*. In addition, it has been stated that since the sandfly, *P. papatasi*, feeds on plant sap containing both sugar and lectins then these may affect development of *Leismania* parasites and their transmission to vertebrate host (46). The presence of lectin in leishmania parasites has also been greatly studied. A lectin-like component with broader specificity on the surface of *L. amazonensis* promastigotes was found (13).
Sandfly lectins agglutinating human RBCs were firstly reported from Lysates of heads, midguts, and hindguts of *Phlebotomus papatasi* (58), which unexpectedly, the head lysates agglutinin activity originates from the haemolymph. Then, this study has been extended to compare lectin activity of three *Phlebotomus* species (*P. papatasi, P. perniciousus and P. perfiliewi*) against human and dog RBC and also against promastigotes of various *Leishmania infantum* strains (15). However, high lectin levels were found in abdominal as well as foregut but not in the hindgut of sandflies (55, 56).
Bloodmeal also effect on the levels of lectin or stimulate the lectin secretion into the midgut. In unfed females the agglutination activities was associated mainly with microvillar surface of midgut epithelium and was present also free in the midgut lumen. In fed ones the gut activity was elevated and the lectin was secreted into the midgut lumen and passes though the peritrophic matrix into the peritrophic space (11). The lectin activity response against bloodmeal depend on species differed and has ranging from two fold in *Lu carmelinoi* up to sixteen fold in *P. duboscqi* and such differences in lectin response may influence the sandflies abilities to support the development of various *Leishmania* species (11).
The agglutination activity of promastigotes of various *Leishmania* species or strains have been found different and natural parasite agglutination titres were observed in some natural vector-parasite combinations (58, 52, 51). Species with high hemagglutination titres usually gave high parasite agglutination titers. Intra-specific in agglutination of *Le. majour* variability strains by sandfly lysates was related to varying virulence of the strains to the laboratory mice (53).
Inhibitory effects of sugar on the parasites /sandflies agglutination were reported by several authors. For example, agglutination of *L. major* and *L. donovani* promastigotes by gut extracts of *P. papatasi* and *Lu. longipalpis* were inhibited mainly by mannosamine , galactosamine and N-acetyl-glucosamine (53). Also lipophosphoglycan (LPG), the major glycoconjugates on the surface of *Leishmania* promastigotes, was shown as a

strong inhibitor of sandfly gut lectin. During metacyclogenesis of promastigotes the LPG undergoes extensive modifications. This change in *L. major* consists of regulation in the number of side chains expressing terminal β-linked galactose in favour of those terminating with arabinose (29). This process is responsible for the control of stage-specific adhesion of promastigotes to sandfly microvilli (38).
Generally, in sandfly, the secreted lectins into the gut and membrane-bound lectin-like receptor play an important role in vector-parasites interactions. The lectin-like receptors on the microvilli might enable attachment of promastigotes to sandfly midgut epithelium while secreted gut lectin may inhibit *Leishmania* development in sandfly midgut (11). The agglutinin activity found on *Leishmania* surface (47) was lower in log-phase promastigotes which display inherent capacity to attach to midgut microvilli than in metacyclics (52). The metacyclics are not longer able to attach to the midgut surface. Thus, the lectin on the parasites is not likely to be involved in this attachment.
However, it was shown that sandflies feed on plant saps which contain sugar and lectins, the development of *Leishmania* parasites and their transmission to vertebrate host were affected (46).

Tsetse Flies and Trypanosomatid Interaction

In trypanosome-tsetse fly interactions, the vector lectins play a dual role; lectins released into the midgut not only lyse the trypanosomes and prevent midgut infections but also provide a signal for the maturation of established trypanosomes to the procyclic form (27, 28). On the other hand, the role of lectin(s) in the maturation of trypanosomes in tsetse has showed that although midgut lectins promote cell death, but are essential for trypanosome maturation (26). The authors suggested that the agglutination of trypanosomes in the fly midgut by binding to the procyclic surface coat, prior to their establishment in the ecto-peritrophic space indicate the midgut lectin is responsible for maturation of the parasite. Subsequently, feeding glucosamine or N-acetylglucosamine to *G. m. moristans* significantly increases the development of parasite in the midgut while D-glucosamine inhibits the killing of procyclic trypanosomes taken as an infective feed (27). Surface labelling experiments indicate the presence of at least 25 such components

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in *T. rhodesiense* procyclics, it is possible that one or more of these is involved in the recognition/attachment event (32). Similar trans-sialidase activity has also been found in others African trypanosomes, *T. brucei brucei, T. b. gambienie, T. b. rhodesiense, T.vivax* and *T. congolense* (9) which may involved in the parasites attachment.

Bloodmeal increase the lectin activity of midgut. Starved *G. palpalis* gut showed little lectin activity while in blood-fed the trypanoagglutinins become elevated to peak titres 2-3 days after a bloodmeal and fall to initial levels five days later (56). This change in lectin activities is thought to be responsible for differences in susceptibility of teneral and non-teneral flies. Flies fed before infective feeds were able to remove trypanosomes from their midgut more quickly than flies infected as tenerals (59).

However, the mechanism of lectin/trypanosome interaction in tsetse flies seems to be complex and involved with different binding proteins and enzymes.

DISCUSSION

Insect vectors and parasites lectins may involve in the interaction between the host cell and pathogens which affect the life cycle of the parasites. Blood-meal may enhance the lectin activity or secretion of lectin in the vector gut of some vector groups which may reflect the higher risk of contamination of gut content during ingestion of the blood. Also, lectin interaction in some vectors is sex depended and in female is higher than male while the female need blood to mature the eggs and this increase. Lectin in haemolymph or gut may responsible for maturation/development of parasites as well as promoting the pathogen death. This may indicates that the parasites escape form insect defence by changing their forms which benefit the parasites to complete their life cycle.

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