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Research review paper

# Protozoa lectins and their role in host–pathogen interactions

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ABSTRACT

Lectins are proteins/glycoproteins of non-immune origin that agglutinate red blood cells, lymphocytes, fibroblasts, *etc.*, and bind reversibly to carbohydrates present on the apposing cells. They have at least two carbohydrate binding sites and their binding can be inhibited by one or more carbohydrates. Owing to carbohydrate binding specificity of lectins, they mediate cell–cell interactions and play role in protozoan adhesion and host cell cytotoxicity, thus are central to the pathogenic property of the parasite. Several parasitic protozoa possess lectins which mediate parasite adherence to host cells based on their carbohydrate specificities. These interactions could be exploited for development of novel therapeutics, targeting the adherence and thus helpful in eradicating wide spread of protozoan diseases. The current review highlights the present state knowledge with regard to protozoal lectins with an emphasis on their haemagglutination activity, carbohydrate specificity, characteristics and also their role in pathogenesis notably as adhesion molecules, thereby aiding the pathogen in disease establishment.

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**Contents**



*Abbreviations:* MIC, micronemal protein 1; TML, *Tritrichomonas mobilensis* lectin; TFL, *Tritrichomonas foetus* lectin; TSL, *Tritrichomonas suis* lectin; Tcon TS-LD, *Trans*-sialidase lectin domain from *Trypanosoma congolense*; Gal/GalNAc, Galactose/*N*-acetylgalactosamine; p 30, protein 30; HU, haemagglutination unit; LLGP, lectin-like glycoprotein; MBP, mannose binding protein; HBP, heparin binding protein; SDS-PAGE, sodium dodecyl sulfate polyacrylamide gel electrophoresis; a.a., amino acid.

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## 1. Introduction

Lectins are proteins/glycoproteins of non-immune origin which agglutinate cells including erythrocytes, lymphocytes and microbial cells, or precipitate glycoconjugates through sugar specific binding sites (Goldstein and Poretz, 1986; Sharon and Lis, 2003). They bind non-covalently to specific sugars on apposing cells and mediate cell–cell interactions. Exceptionally, some lectins bind to cell surfaces but do not result in agglutination (non-agglutinating lectins), as affinity of lectin molecule to its receptor varies according to the variations in their carbohydrate receptor (Northrop and Connor, 2008). Toxic non-agglutinating lectins and non-toxic agglutinating lectins have been reported from seed extracts of *Ricinus communis* (Olsnes and Pihl, 1973). Due to specific binding of both these proteins to animal cell surfaces *via* specific sugar residues, these have been properly termed as lectins (Callow, 1976). Haemagglutination is a major attribute of lectin activity and has been classically and routinely used for detection and characterization of lectins (Ambrosi et al., 2005). However, cell agglutination is not a defining feature of lectins and a broader definition includes carbohydrate binding proteins other than enzyme or antibody (Barondes, 1988). Peumans and Van Damme (1995) concluded that the prerequisite for a protein to be termed as lectin is presence of atleast one non-catalytic domain that binds to a carbohydrate, specifically and reversibly. Certain carbohydrate binding proteins do not precipitate glycoconjugates or agglutinate cells due to presence of single carbohydrate binding site. Thus various proteins behaving differently in terms of their agglutination and/or glycoconjugate precipitation are included under the term "lectins" (Peumans and Van Damme, 1995).

Owing to their ability to identify subtle variations in carbohydrate structures, lectins have been the subject of intense research. Lectins are excellent candidates to mediate parasite interactions with complementary ligands on host cells and thus play a significant role in cell–cell interactions (Singh et al., 2011a; Singh and Walia, 2014a). Lectins from microbes have been attributed with varied biomedical applications (Singh and Bhari, 2013; Singh et al., 2010, 2015a, 2016a; Tiwary and Singh, 1998, 1999). A panorma of microfungal lectins possess antimicrobial (Singh et al., 2013; Singh and Thakur, 2014), mitogenic (Singh and Walia, 2014b; Singh et al., 2014, 2015b, 2015c) and immunomodulatory (Singh et al., 2011b) activities, and also have potential roles in mycoparasitism and host–parasite interactions (Singh et al., 2011c). Based on their unique carbohydrate binding specificities, a number of lectins have been reported from plants, animals, fungi, bacteria, protozoa and viruses *etc.* (Singh et al., 1999). Data survey from various internet sources reveals that protozoal lectins constitute only 2.2% of total microbial lectins (Fig. 1). A number of protozoa such as *Entamoeba* (Petri et al., 1987), *Giardia* (Ward et al., 1990), *Cryptosporidium* (Joe et al., 1994), *Trichomonas*, (Roussel et al., 1991) and *Plasmodium* (Jungery, 1985) *etc.* possess lectin-like proteins. In protozoans, lectins mediate parasite adherence to host cells, thus play a significant role in various cell-to-cell interactions (Thea et al., 1992), in binding to red blood cells (Schoppert et al., 1996), and in cytopathogenicity (Petri et al., 2002; Roussel et al., 1991). Keeping in view the pivotal role of lectins in protozoa pathogenesis, the current review focusses on haemagglutination activity, carbohydrate specificity and characteristics of various protozoa lectins and also lays emphasis on role of these lectins in disease establishment and pathogenesis.

## 2. Haemagglutination activity of protozoa lectins

A remarkable property of lectins is to agglutinate human blood type erythrocytes, and based on their specificity to agglutinate, they are classified as specific/non-specific. Erythrocyte susceptibility to certain lectins occurs upon its mild enzymatic treatment due to exposure of masked sites on/within erythrocyte membrane. Haemagglutination activity of various protozoan lectins is enlisted in Table 1. Surface membrane-associated lectin from *Giardia lamblia* trophozoites avidly agglutinates rabbit erythrocytes, primarily after trypsin treatment of erythrocytes (Farthing et al., 1986). Protease treatment leads to *de novo* exposure of underneath cryptantigen by removing glycol-coat from erythrocyte surface. Based on the data present, a unique

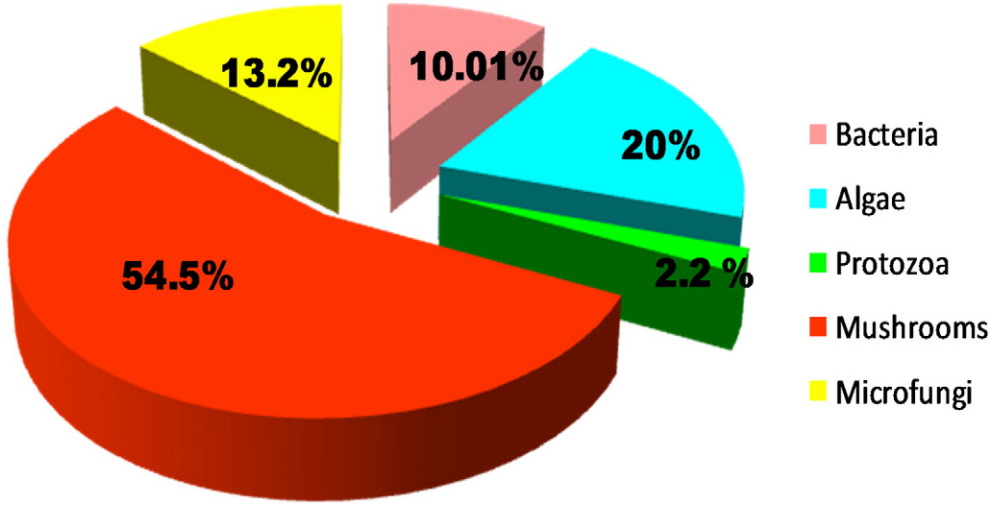


**Fig. 1.** Distribution of protozoal lectins among various microbial groups (Data surveyed from various internet sources).

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