

An Overview on Specificity of Glycan-Binding Proteins Associated with Microbial Lectins

Bipin Aloor^{*}

Department of Microbiology, Davangere University, Karnataka, India

DESCRIPTION

Glycan-binding proteins, also known as lectins, are a diverse group of proteins that have the ability to specifically recognize and bind to carbohydrates, particularly complex sugar molecules called glycans [1,2]. These proteins are essential in various biological processes across all domains of life. They possess specialized carbohydrate-binding domains that allow them to recognize and bind to specific carbohydrate structures, including monosaccharides, oligosaccharides, and complex glycans.

Specificity of glycan-binding proteins

The binding specificity of lectins is determined by the arrangement and chemical properties of sugar molecules within the carbohydrate target. Glycan-binding proteins are classified into several families based on their structural and functional characteristics. Common families of lectins include C-type lectins, galectins, ficolins, and siglecs, among others [3,4]. Each lectin family has its own characteristic carbohydrate-binding domain and binding specificity. Few examples are:

- Concanavalin A (ConA) is a lectin protein derived from the seeds of the jack bean plant (*Canavalia ensiformis*). It is one of the most well-known and widely studied lectins in the field of glycobiology and molecular biology. ConA is known for its strong affinity for specific carbohydrate structures, particularly mannose and glucose residues [5,6].
- Galectins are a family of lectin proteins found in various organisms, including animals, and have an affinity for β -galactoside-containing carbohydrates. They are known for their diverse roles in various biological processes, including cell adhesion, signaling, immune regulation, and cancer progression.
- Siglecs, or sialic acid-binding immunoglobulin-like lectins, found on immune cells, particularly on white blood cells like lymphocytes, monocytes, and neutrophils. Siglecs are expressed in immune regulation and cell-cell interactions [7,8].

Microbial lectins

Hemagglutinins: Hemagglutinins are microbial lectins that agglutinate (clump together) red blood cells (erythrocytes) by binding to specific carbohydrates on their surfaces. They are often found in bacteria and viruses during the initial stages of infection. By binding to host erythrocytes, these lectins can facilitate the attachment of pathogens to host tissues. Influenza viruses are known for their hemagglutinins, which bind to sialic acid receptors on host cells [9].

Adhesins: Adhesins are microbial lectins that bind to carbohydrates on host cell surfaces, allowing microorganisms to adhere to host tissues and establish infections. Adhesins can also be involved in biofilm formation. Inhibiting the binding of adhesins to host cells could prevent the establishment of infection. Fimbriae in bacteria like *Escherichia coli* and *Neisseria gonorrhoeae* are adhesins that promote bacterial adherence to host cells [10].

Toxins: Toxin lectins are microbial lectins that, when bound to host carbohydrates, can have harmful effects on host cells and tissues. They are often part of the virulence factors of pathogens. They can disrupt host cell membranes, interfere with host cell signaling, and modulate immune responses, leading to damage or disease. Ricin, a toxin lectin derived from the castor bean plant, binds to specific carbohydrates on host cell surfaces and disrupts protein synthesis, leading to cell death.

CONCLUSION

Microbial lectins are essential components of various microorganisms that contribute to their ability to interact with and infect host cells. Hemagglutinins, adhesins, and toxin lectins serve different roles in host-pathogen interactions, with hemagglutinins and adhesins promoting adherence to host tissues, while toxin lectins can have detrimental effects on host cells. Understanding the mechanisms of microbial lectincarbohydrate interactions is essential for developing strategies to combat microbial infections and diseases.

Correspondence to: Bipin Aloor, Department of Microbiology, Davangere University, Karnataka, India, E-mail: bi.al@gmail.com

Received: 15-Jun-2023, Manuscript No. JGB-23-26980; **Editor assigned:** 19-Jun-2023, PreQC No. JGB-23-26980 (PQ); **Reviewed:** 4-Jul-2023, QC No. JGB-23-269780; **Revised:** 11-Jul-2023, Manuscript No. JGB-23-26980 (R); **Published:** 19-Jul-2023, DOI: 10.35841/2168-958X.23.12.250.

Citation: Aloor B (2023) An Overview on Specificity of Glycan-Binding Proteins Associated with Microbial Lectins. J Glycobiol. 12:250.

Copyright: © 2023 Aloor B. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original author and source are credited.

OPEN ACCESS Freely available online

Aloor B

REFERENCES

- 1. Goldstein IJ, Hughes RC, Monsigny M, Osawa T, Sharon N. What should be called a lectin?. Nature. 1980;285(5760):66.
- 2. Ashwell G, Harford J. Carbohydratespecific receptors of the liver. Annu Rev Biochem. 1982;51(1):531-554.
- Drickamer K. Two distinct classes of carbohydrate-recognition domains in animal lectins. J Biol Chem. 1988;263(20):9557-9560.
- 4. Powell LD, Varki A. L-type lectins. J Biol Chem.1995; 270(24): 14243-14246.
- 5. Lee RT, Lee YC. Affinity enhancement by multivalent lectincarbohydrate interaction. Glycoconj J. 2000;17(7-9):543-551.
- Casu B, Lindahl U. Structure and biological interactions of heparin and heparan sulfate. Adv Carbohydr Chem Biochem. 2001;57:159-206.

- 7. Esko JD, Selleck SB. Order out of chaos: Assembly of ligand binding sites in heparan sulfate. Annu Rev Biochem. 2002;71:435-471.
- 8. Drickamer K, Taylor ME. Identification of lectins from genomic sequence data. Methods Enzymol. 2003;362:560-567.
- Lee JK, Baum LG, Moremen K, Pierce M. The X-lectins: A new family with homology to the Xenopus laevis oocyte lectin XL-35. Glycoconj J 2004;21(8-9):443-450.
- Clynes RA, Towers TL, Presta LG, Ravetch JV. Inhibitory Fc receptors modulate in vivo cytoxicity against tumor targets. Nat Med. 2000;6(4):443-446.