OPEN ACCESS Freely available online

Journal of Glycobiology

ISSN: 2168-958X

Perspective

Molecular Mimicry of Host Glycan's in Infection and Autoimmunity

Lu Ba Shi*

Department of Orthopedics, First Hospital of China Medical University, Shenyang, China

DESCRIPTION

Molecular mimicry of host glycan's is a concept in immunology and microbiology where pathogens, such as bacteria, viruses, or parasites, develop surface molecules (usually glycoproteins or glycolipids) that structurally resemble host glycan's. This mimicry allows the pathogen to evade the host's immune system by camouflaging itself as a part of the host, making it less likely to be recognized and attacked by the immune response. This phenomenon is often associated with autoimmune diseases and infections.

Pathogens produce glycoproteins or glycolipids with structures that are similar to host glycan's. This structural similarity allows the pathogen's surface molecules to be mistaken for self-components by the host's immune system. When the immune system encounters these pathogen-produced molecules, it may recognize them as self and not mount a strong immune response. This is because the immune system is designed not to attack the body's own cells and molecules. The presence of molecular mimicry can lead to autoimmune diseases. In some cases, the immune system may become confused and mistakenly target the host's own tissues and cells that contain similar glycan's, leading to autoimmune reactions. By avoiding detection and immune attack, pathogens can establish infections and persist within the host for extended periods, causing diseases.

Streptococcus pyogenes and Rheumatic fever

Streptococcus pyogenes, also known as Group A Streptococcus (GAS), is a bacterium responsible for a wide range of human infections, including strep throat, scarlet fever, impetigo, and invasive diseases such as cellulitis and necrotizing fasciitis. One of the most significant and well-known complications associated with *Streptococcus pyogenes* infection is rheumatic fever.

Rheumatic fever is an autoimmune inflammatory disease that can occur as a complication of untreated or inadequately treated *Streptococcus pyogenes* infections, particularly strep throat. It is characterized by an abnormal immune response in which the immune system mistakenly attacks and damages various parts of the body, including the heart, joints, skin, and the central nervous system. The development of rheumatic fever is believed to result from molecular mimicry. *Streptococcus pyogenes* has surface molecules with structural similarities to host tissues, including cardiac tissue. When the immune system responds to the bacterial infection, it may cross-react with host tissues, particularly the heart. It can present with various symptoms, including fever, joint pain and swelling, skin rashes and inflammation of the heart.

Rheumatic fever can damage the heart, leading to a condition known as rheumatic heart disease. This condition can result in permanent heart valve damage, which can cause long-term heart problems and complications. It is preventable by promptly diagnosing and treating *Streptococcus pyogenes* infections with antibiotics, such as penicillin. Timely treatment can prevent the development of rheumatic fever. In cases of rheumatic fever, treatment typically involves anti-inflammatory medications, such as aspirin, and antibiotics to eradicate any remaining streptococcal bacteria. For severe cardiac complications, surgical interventions like valve replacement may be necessary.

Campylobacter jejuni and Guillain-Barré syndrome

Campylobacter jejuni is a bacterium commonly associated with foodborne illness and is a leading cause of bacterial gastroenteritis in many parts of the world. Guillain-Barré Syndrome (GBS) is a neurological disorder that can occur as a rare and severe complication following a *Campylobacter jejuni* infection.

Campylobacter jejuni is often transmitted to humans through the consumption of contaminated food, particularly undercooked poultry, unpasteurized milk, and contaminated water. When a person is infected with *Campylobacter jejuni*, they may develop symptoms of gastroenteritis, including diarrhea, abdominal pain, fever, and nausea. Molecular mimicry is believed to be a contributing factor in the development of Guillain-Barré syndrome following Campylobacter infection. The bacterium's surface Lipooligosaccharides (LOS) have structures that resemble gangliosides, which are molecules found on nerve cells. In some

Correspondence to: Lu Ba Shi, Department of Orthopedics, First Hospital of China Medical University, Shenyang, China, E-mail: lbshi@6766.edu.cn

Received: 15-Aug-2023, Manuscript No. JGB-23-27627; **Editor assigned:** 18-Aug-2023, PreQC No. JGB-23-27627 (PQ); **Reviewed:** 31-Aug-2023, QC No. JGB-23-27627; **Revised:** 07-Sep-2023, Manuscript No. JGB-23-27627 (R); **Published:** 15-Sep-2023, DOI: 10.35841/2168-958X.23.12.252.

Citation: Shi LB (2023) Molecular Mimicry of Host Glycan's in Infection and Autoimmunity. J Glycobiol. 12:252.

individuals, the immune response generated against *Campylobacter jejuni* infection can cross-react with gangliosides in the peripheral nervous system, resulting in an autoimmune response. The immune system attacks and damages the myelin sheath (the protective covering of nerve fibers) and can lead to the development of Guillain-Barré syndrome.

Guillain-Barré syndrome is an autoimmune condition characterized by progressive muscle weakness, loss of sensation, and in some cases, paralysis. It usually starts in the legs and can ascend to involve the arms and potentially the muscles that control breathing. GBS is often accompanied by numbness, tingling, and in severe cases, difficulty breathing. The syndrome can be life-threatening if it affects respiratory muscles, requiring hospitalization and sometimes mechanical ventilation.

Diagnosis of Guillain-Barré syndrome typically involves clinical evaluation, nerve conduction studies, and cerebrospinal fluid analysis. There is no specific cure for GBS, but treatment involves managing symptoms, providing supportive care, and sometimes administering Intravenous Immunoglobulin (IVIG) or plasma exchange to reduce the autoimmune response. Many individuals with GBS recover with time and rehabilitation, although recovery can be slow and may take months to years. In some cases, individuals may experience residual neurological deficits or long-term complications.

CONCLUSION

Understanding molecular mimicry is important in both the fields of immunology and microbiology, as it explains about the mechanisms of autoimmune diseases and inform the development of vaccines and treatments to target pathogens that exploit these mimicry mechanisms. It's important to note that rheumatic fever is now relatively rare in developed countries due to improved access to healthcare, antibiotics, and better hygiene practices. While *Campylobacter jejuni* infection can lead to Guillain-Barré syndrome, the development of GBS following such an infection is relatively rare. Most people who contract *Campylobacter jejuni* infections do not develop GBS. However, this association highlights the importance of food safety and prompt treatment of bacterial infections to reduce the risk of complications.