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Short Communication

Glycan Recognition by Galectins in Transfusion and Immune Processes

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DESCRIPTION

The ABO blood group system represents one of the most extensively studied antigenic systems in human biology, with direct relevance to transfusion medicine and broader physiological processes. These blood group antigens are carbohydrate structures expressed on the surface of red blood cells as well as on epithelial and endothelial cells. Their distribution extends into secretions and tissues, reflecting their synthesis through glycosyltransferase enzymes that modify precursor oligosaccharides. Beyond their established role in compatibility during blood transfusion, ABO antigens participate in interactions with endogenous lectins, including galectins, which recognize specific glycan patterns. The relationship between ABO blood groups and galectins has attracted attention for its potential implications in immune defence, inflammation, and host–pathogen interactions.

Galectins participate in innate immunity by recognizing glycan structures on pathogens and host cells. They can bind to bacterial, viral, and parasitic components, contributing to pathogen recognition and clearance. In addition to direct antimicrobial effects, galectins modulate immune cell behaviour by influencing cytokine production, apoptosis, and cell migration. The interaction between galectins and ABO antigens may shape these processes by altering the availability of binding sites or modifying receptor clustering on cell membranes. For example, differences in glycosylation patterns associated with blood group phenotypes may affect how galectins cross-link glycoproteins, thereby influencing signaling pathways involved in immune activation.

The role of galectins in apoptosis and cell survival further extends their relevance to immune regulation. By binding to glycan structures on cell surfaces, galectins can trigger signaling pathways that lead to programmed cell death or survival, depending on the context. ABO antigen expression may influence these processes by altering glycan availability and receptor organization. In immune cells, such modulation can affect the balance between activation and tolerance, shaping overall immune function. These mechanisms highlight the broader significance of glycan–lectin interactions in maintaining physiological balance.

In transfusion practice, consideration of glycan interactions beyond classical antigen–antibody compatibility may contribute to more refined approaches to donor–recipient matching. While current protocols focus primarily on ABO and Rh systems, future strategies may incorporate additional factors related to glycosylation patterns and lectin interactions. Such approaches could improve transfusion outcomes, especially in patients with complex medical conditions or those requiring long-term transfusion support. However, further research is needed to establish the clinical significance of these interactions and to develop practical methods for their assessment.

The interplay between ABO blood groups and galectins also has potential implications for therapeutic development. Galectin inhibitors and modulators are being explored in various clinical contexts, including cancer and inflammatory diseases. Understanding how ABO-related glycan variations influence galectin activity may inform the design of targeted therapies or personalized treatment strategies. In addition, manipulation of glycan structures on therapeutic cells or biomaterials could enhance their compatibility and function within the host.

CONCLUSION

The relationship between ABO blood groups and galectins represents an intersection of transfusion medicine and innate immunity grounded in glycan biology. ABO antigens define distinct carbohydrate patterns that influence how galectins interact with cells, affecting immune recognition, inflammation, and cellular communication. While the primary role of ABO compatibility in transfusion remains centered on preventing antibody-mediated reactions, additional layers of interaction involving galectins may contribute to subtle variations in clinical outcomes. Continued research into these mechanisms will deepen understanding of how glycan–lectin interactions shape human physiology and may support the development of more precise and effective medical interventions

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