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Opinion

Essential Galectin Functions in Cell Polarity

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Half a Century of Progress in Galectin Research

Galectin was first discovered by Teichberg in 1975 [1] and is now recognized as a family of endogenous lectins with a high affinity for polysaccharides containing β-galactoside residues [2]. Fifteen galectin family members, classified into three groups, have been identified, all of which have one or two highly conserved carbohydrate recognition domains (CRDs) [2]. They perform diverse biological functions in different cell types and tissues, such as immunity [3], cell adhesion [4], and cell migration [5]. Most of these functions are performed by secreted galectins, and such extracellular functions of galectins have been widely recognized and studied. However, a critical question is what intracellular galectins do because galectin is ubiquitous in the nucleus, cytoplasm, and extracellular space [6]. So far, this area of research remains largely untapped.

Galectin Dependent Targeting of Glycoproteins to the Apical Membrane

Unlike non-polar cells like mesenchymal cells, macrophages, and plasma cells, epithelial cells are characterized by their polarity with structurally and functionally distinct apical and basolateral membranes. An important feature of cell polarity is polarized vesicular trafficking, controlled by precise intracellular sorting machinery that delivers proteins to the apical or basolateral cell surface. The fundamental knowledge about the cellular components and molecular players for polarized membrane protein sorting was established three decades ago [7], and significant progress has been made. One of the most important discoveries is that cytosolic galectins play an essential role in glycoprotein trafficking to generate and maintain epithelial cell polarity, and galectin-mediated cross-linking of glycoproteins is crucial for apical delivery.

In polarized Madin-Darby canine kidney II (MDCK II) cells, after the release of p75NTR from the trans-Golgi network (TGN), the chimera-type galectin, galectin-3 forms a complex with p75NTR through O-glycan binding and stabilizes p75NTR glycoprotein cross-linked complexes [8]. This complex is formed in a vesicular post-Golgi compartment, and this vesicle carrier takes galectin-3 and p75NTR together to the apical membrane [9]. Similarly, galectin-9 of the tandem-repeat class can also influence apical protein transport to maintain MDCK cell polarity [10]. Another tandem repeat-type galectin, galectin-4-dependent glycoprotein delivery to the brush border of intestinal enterocyte-like HT-29 cells utilize a lipid raft-based mechanism. A direct implication of galectin-4 in the recruitment of glycoproteins such as the glycosyl-phosphotidylinositol (GPI)-anchored complement regulatory protein (CD59) with detergent-resistant membranes (DRMs) is through a lectin-type interaction [11]. Of course, galectins assist in transporting much more cargos responsible for cell polarities than mentioned above, such as apical membrane glycoproteins dipeptidylpeptidase-IV (DPP-IV) [12] and β1-integrin [13], and more glycoprotein cargos remain to be discovered.

Potential Clinical Values

The galectin-dependent mechanisms in polarized cells may be of potential value for understanding epithelial cell-derived tumors. Because when epithelial cell atypical hyperplasia develops under precancerous conditions, the cell polarity is lost. Squamous metaplasia of epithelium is one of these examples. Whether galectin-dependent apical protein sorting is impaired in these processes and whether restoring galectin-dependent sorting can at least partially reverse the epithelial cell atypical hyperplasia are all questions that should be explored. Hopefully, such studies will soon flourish and reveal their significant biomedical values.

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