



[Science Archives \(ISSN:2582-6697\)](http://www.sciencearchives.org)

Journal homepage: [www.sciencearchives.org](http://www.sciencearchives.org)



<https://doi.org/10.47587/SA.2023.4211>

Review Article



## Human galectines and their contribution in the chronic colonic inflammation

Atika Alahmed <sup>✉1</sup>, Eman Al-Rubae <sup>2</sup>, and Talal H. Noon<sup>3</sup>

<sup>1</sup>Department of Oral Medicine, College of Dentistry, University of Baghdad, Iraq

<sup>2</sup>Department of Basic Science, College of Dentistry, University of Baghdad, Iraq

<sup>3</sup>Department of Medicine, Al-Zahraa College of Medicine, University of Basra, Iraq

Received: May 18, 2023/ Revised: June 7, 2023/Accepted: June 15, 2023

(✉) Corresponding Author: [atika.alahmed@gmail.com](mailto:atika.alahmed@gmail.com)

### Abstract

Galectins are a group of 16 members that all share an amino acid sequence named carbohydrate recognition domain that binds to a beta-galactoside and produces multiple intra- and extra-cellular functions such as modulating inflammation. Inflammatory bowel disease is a chronic inflammatory disorder of the large and small intestines that comprises two diseases, Crohn disease and Ulcerative Colitis. Ulcerative colitis showed a persistent inflammatory process that affects mainly the colon, this chronic colonic inflammation is affected by several internal and environmental factors. Many galectins are classically expressed in certain gut areas. Organ-specific distribution and abundance of some Galectins make them an important material for many researchers and a useful therapeutic target for different diseases, therefore; their role in colonic inflammation is reviewed to manifest their correlation to chronic diseases such as ulcerative colitis.

**Keywords:** Inflammatory Bowel Disease, Ulcerative Colitis, Galectins, Colonic Inflammation.

### Introduction

Ulcerative Colitis (UC) is a chronic inflammatory disorder affecting the large intestine with a relapsing and remitting course. The type and location of colonic lesions, the presenting symptoms along with the remission rate and prognosis all are suggestive of the underlying inflammatory process that is affected by different genetic, immune, and environmental factors (Porter et al., 2022). UC along with Crohn's disease (CD) collectively grouped under the term inflammatory bowel disease (IBD), UC may comprise an unusual activation of T-cells found in mucosa against the commensal microbiota leading to and deregulation of intestinal function and stimulating an inflammatory response along with the increased secretion of IL- 5 & IL-13 (Neurath, 2014).

Galectins are a subfamily of lectins that have a great affinity to bind to  $\beta$ -galactosides in carbohydrate-containing molecules such as glycoproteins or glycolipids, the first member was discovered back in 1975 the others were subsequently discovered, till now, up to 16 members have been discovered

in mammals, 12 of them recognized in humans (Fig. 1) (Varki et al., 2022). Galectins are synthesized in the cytosol, afterward, they are transported to the nucleus to participate in DNA-protein interactions (as in transcription) or secreted out of the cell (to be in action on the plasma membrane). Galectins demonstrate a functional variation, where the binding to the endogenous glycans can assist in cell-to-cell interactions, cell signaling and regulation of several intracellular activities; while the binding to the exogenous glycans such as those on pathogens will support the immune functions (Liu et al., 2020).

The connections between galectins and any carbohydrate-containing molecule are mediated via a part named carbohydrate recognition domain (CRD) which usually involves approximately 130 amino acid residues strongly folded into antiparallel  $\beta$ -sheets configuration, this strongly folded structure of the CRD may help to explain the requirement for that number of amino acids in the CRD and the degree of CRD's resistance to the protease activity (Verki et al., 2022). Depending on the number and type of organization of CRDs in each, galectins have been classified

into three major groups (Sanjurjo et al., 2022): Proto-type Galectins: This group includes galectin-1 (Gal-1), 2, -7, -10, -11, -13, -14, -15 and -16 contain a solitary CRD and may link together as homo-dimers (monomers); Chimera-type Galectins: This group has only one member which is galectin-3 (Gal-3), it is characterized by comprising a single CRD and

an extremity (long chains) rich in glycine, proline, and tyrosine amino acids through them Gal-3 can form pentamers; Tandem-repeat Galectins: This group contains galectin-4, -8, -9, and -12 which comprise two different CRDs coupled by a peptide conjugate that can be up to 50 amino acids in extent.

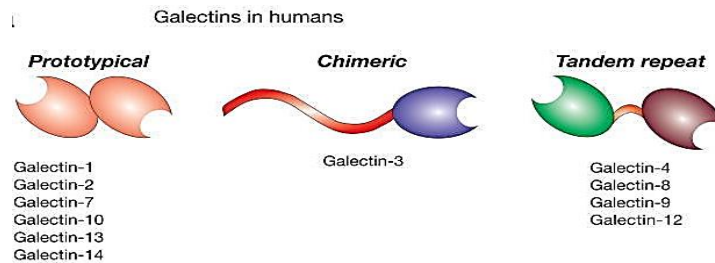


Fig. 1 Types of Galectins in human (Varki et al., 2022)

### Galectins role in the colonic inflammation

Even though Galectins are normally tissue-associated but their levels in the serum can considerably rise upon tissue damage or as a result of infection and inflammation to help in the activation of immune response and the body defenses grant the opportunity for these galectins to be used as valuable biomarkers for diagnosis and prognosis of several disorders (Thiemann and Baum, 2016). Human Galectins participate in the regulation of immune and inflammatory systems homeostasis by regulating cell signaling and chemotaxis as well as cytokine secretion, it is important to notice that the same galectin can have pro- or anti-inflammatory responses, conditioned upon the type of stimulus (stimulatory or inhibitory), microenvironment and target cells in which they are found and the nature of the pathological condition. As well, the response of immune cells to the galectins is also governed by the glycosylation of surface receptors to create active galectin ligands to start diverse signaling programs such as immune cell activation, trafficking, and survival (Yaseen et al., 2020). The role of each member in this family in inflammatory and immune processes, in general, will be revised in this review (including the latest 10 years).

**Galectin-1:** In the thymus, Gal-1 affects the apoptosis of activated T-cells (but not resting T-cells) and control their clonal expansion (sandblad et al., 2017). In the areas of inflammation, studies suggest that Gal-1 enhances anti-inflammatory action and contributes to dynamically resolve acute inflammation where it reduces neutrophils recruitment and enhance their clearance as well as supports the conversion of macrophages in to M2 phenotype (that promote Th2 action and resolve the active inflammation and advance tissue repairs) and influence dendritic cells maturation toward IL-10 secretion that favors T-cells tolerance and suppression (Law et al., 2020). Gal-1 also (through the interaction with CD45-ligand) increase the apoptosis of the T-cells (especially Th1 &

Th17 but not Th2), furthermore, to discontinue an active immune response, activated T-cells themselves can produce Gal-1 in an autocrine mechanism to induce their own apoptosis and the action of Gal-1 goes toward activation and expansion of regulatory T-cell (T-reg) and IL-10 secretion (Cerliani et al., 2017). The placental Gal-1 has an anti-inflammatory action as well (as seen in the cases of preeclampsia) where it is up-regulated as a fetal reaction to the maternal systemic infection or the conjunctival Gal-1 that aims to reduce allergic conjunctivitis (Hazarbassanov, 2022). UC pathogenesis involves abnormally activated mucosal T-cells against the commensal microbiota leading to the deregulation of normal intestinal function and stimulating an inflammatory response, studies found that Gal-1,-3, -4, and -9 have higher expression in the colon than in the small intestine, and principally Gal-1 was up-regulated in inflamed areas than in the normal tissue samples (Papa Gobbi et al., 2016) which have been explained as an attempt of Gal-1 to: 1)increase the activated T-cell apoptosis, 2)to subside the levels of pro-inflammatory cytokines, 3)to restore mucosal homeostasis and enterocytes viability and 4) eventually perseverance of gut inflammation (Law et al., 2020).

**Galectin-2:** It was first recognized in 1992, it is mainly expressed in the gut and in a lesser extent in the placenta and heart. Its normal function in the gut includes binding with the mucin to increase epithelial protection, also binds with other proteins on the mucosal surface of gut to help in accelerating wound healing (Xu et al., 2021). During the inflammation of the gastrointestinal tract (GIT), Gal-2 was found to bind with T-cells surface glycans, binding to CD3 or CD7 initiates the production of IL-5 and IL-10 (anti-inflammatory cytokines) while the secretion of IFN- $\gamma$  and TNF- $\alpha$  (pro-inflammatory cytokines) is inhibited, this binding also induces the T-cells apoptosis which helps to reduce the inflammatory response and support wound repairing thru preserving the colonic crypts integrity and epithelial architecture as revealed by

experimentally induced colitis (Yu et al., 2020). In response to Gal-2, is increased, all the above effects were studied in mice and fewer similar studies were conducted in humans, in line

with this, a study in 2020 showed that no significant difference in Gal-2 levels was detected in the serum between healthy and ulcerative colitis patients (Negedu et al., 2023).




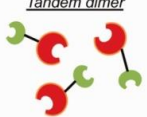




Galectin classification	Immune cell	Galectin	Cell functional response
<p><i>Non-covalent homodimer</i></p>  <p>Galectin-1, -2, -5, -7, -10, -11, -13, -14, -15</p>	T cell	 <p>galectin-1, -2, -3, -4, -9</p>	<ul style="list-style-type: none"> <li>↑ apoptosis</li> <li>↓ inflammatory cytokine secretion</li> <li>↑ regulatory cytokine secretion</li> <li>↓ cytotoxicity</li> <li>↑ or ↓ T cell receptor activation</li> </ul> <p>Reviewed in (41)</p>
	neutrophil	 <p>galectin-1, -3</p>	<ul style="list-style-type: none"> <li>↑ migration (36)</li> <li>↑ adhesion to ECM glycoproteins (40)</li> </ul>
<p><i>Tandem dimer</i></p>  <p>Galectin-4, -6, -8, -9, -12</p>	pre-B cell	 <p>galectin-1</p>	<ul style="list-style-type: none"> <li>↑ pre-B cell/stromal cell synapse formation (38)</li> <li>↑ pre-B cell receptor clustering and signal initiation (38)</li> </ul>
	dendritic cell	 <p>galectin-1</p>	<ul style="list-style-type: none"> <li>↑ migration (35)</li> <li>↑ tolerogenic cytokine secretion (37)</li> <li>↓ co-stimulator expression (37)</li> <li>↓ inflammatory cytokine secretion (37)</li> </ul>
<p><i>Non-covalent homopentamer</i></p>  <p>Galectin-3</p>	macrophage	 <p>galectin-3</p>	<ul style="list-style-type: none"> <li>↑ alternative activation (39)</li> </ul>

Fig. 2 An overview of how the Galectins modulate immune cell function (Farhadi and Hudalla, 2016)

**Galectin-3:** Gal-3 expression was noticed in many types of cells such as: epithelial cells of colon and small intestine, corneal and conjunctival epithelia, epithelial cells of lung, thymus, kidney and prostate as well as cells of immune system (Sciacchitano et al., 2018). Regarding the inflammation, Gal-3 has an interesting action as it is reliant on the type of organ and type of injury, it helps in the neutrophils and mast cells activation and attraction in addition to the macrophage activation and all these cells are involved in the acute colonic inflammation, the chronic inflammation also reveals the strong role of Gal-3 regarding T-cells activation, differentiation and survival along with initiation of tissue fibrosis and scarring with later organ failure (Yu et al., 2020). It is important to know that Gal-3 has also immunosuppressive effects when it is related to adaptive immunity, where the studies found that this molecule inhibits Th1 differentiation, prevents T cell proliferation, enhances phagocytosis of apoptotic cells, supports wound healing and induces T cell apoptosis, (through an apoptotic mechanism dissimilar from that of Gal-1 (Block et al., 2016). The important and unique action of Gal-3 in the colonic inflammation was explained by the study of Yu and coworkers (2020) who revealed that the experimentally induced colitis passed through induction and progression phases. During the induction phase, Gal-3 enhances the pro-inflammatory action of colonic macrophages and dendritic cells that showed an increased infiltration and an increased secretion of TNF- $\alpha$ , IL-6 and IL-18 cytokines. Whereas in the progression phase, the macrophages turned into M2 anti-inflammatory phenotype along with production of TGF- $\beta$  and

IL-10 as well as initiating of the expansion of T-regs that suppress the action of the activated Th1 & Th17 all of these events will reduce the in-progress response and the level of circulating Gal-3 even though it is not correlated with the disease activity but does relate to its progress (Sciacchitano et al., 2018).

**Galectin-4:** Gal-4 is primarily expressed in the gut where in normal conditions it helps in stabilizing the lipid molecules on the gut mucosa, also has a bactericidal effect against Escherichia coli (*E. coli*) O86 and can help in restoring the injured intestinal areas (Kim et al., 2013). In UC, there were controversial results due to different experimental colitis, in one of them the role of Gal-4 was similar function of Gal-2 where it reduces mucosal inflammation through enhancing the apoptosis of activated T-cells, reducing pro-inflammatory cytokine secretion and promoting wound-healing while in another one, Gal-4 increased the secretion of IL-6 that is a known to exacerbate which needs additional studies to determine the universal role of Gal-4 in intestinal inflammation (Cao and Gue, 2016). In another study on mice, Gal-4 was found to bind with CD14 on the monocytes inducing their differentiation into (macrophage-like cells) and enhancing their phagocytic capacity, the study also stated that these cells (in contrary to the normal intestinal macrophages) have innate immune receptors (CD14) and can secrete pro-inflammatory cytokines such as IL-1 & IL-6 (Hong et al., 2019). In recent study, Fundora and coworkers (2022) revealed an elevated expression of Gal-4 in neonatal intestinal

injury including spontaneous intestinal perforation (SIP) and necrotizing enterocolitis (NEC) when compared to controls but whether its elevation was related to the pro- or anti-inflammatory mechanism was inconclusive as well.

**Galectin-5:** Gal-5 and Gal-6 are primarily found in rodents (not humans).

**Galectin-7:** It was first described in 1995, is largely expressed by stratified squamous epithelia (keratinocyte specific) such as skin, cornea, tongue, esophagus, stomach and anus, its function mainly involves the cell proliferation, differentiation, adhesion and migration as well as re-epithelialization of the wounds (Lin et al., 2021). A study in (2018) by Luo and colleagues showed that resting T cells did not respond to Gal-7, whereas activated T cells had responded and secreted higher levels of TNF- $\alpha$  and IFN- $\gamma$ , they also found that Gal-7 had more proliferative effect on Th1 than Th2 increasing the pro-inflammatory cytokines secretion and reducing the level of the regulatory IL-10 & transforming growth factor- $\beta$  (TGF- $\beta$ ) on the other hand, which will explain the immune-enhancement role of Gal-7 especially in the autoimmune diseases and grafts rejection.

**Galectin-8:** Gal-8 is expressed in the liver, cardiac muscle, kidney, brain and endothelial cells. The in vitro studies showed that in the lymphoid organs, Gal-8 had pro-apoptotic activity on the immature T-cells helping in the maturation and tolerance processes, while in the peripheral blood, Gal-8 induced T-cell proliferation with the corresponding secretion of cytokines as seen in the inflammatory and autoimmune processes (Pardo et al., 2017). Different reports revealed that Gal-8 may have dual actions on T cells that are found in the areas of active inflammation by governing T-cell expansion through activating the apoptotic machinery and enhancing T-reg action (Tribulatti, 2020). Gal-8 also induces the proliferation of B-cells with the subsequent maturation and antibodies secretion with similar stimulatory action is applied by Gal-8 on the innate immunity cells (dendritic cells, neutrophils and monocytes). The dual effect of Gal-8 on the activated and naïve cells is related to the distinct surface glycosylation profile of these cells and the type of cellular signaling produced (Carabelli et al., 2018).

**Galectin-9:** It has the same distribution as Gal-1 & -3, especially in the thymus, liver, heart, skeletal muscles, brain and kidney, similar to them, it can modulate immunity through different aspects (O'Brien, 2018). Gal-9 supports the maturation and phagocytosis activity of the dendritic cells, also its effect on the T-cells is concentration related where it shows suppressive effects by induction of apoptosis of T-cells (Th1, Th 17 and cytotoxic T-cells) and by suppressing B cell signaling when it is found in high concentrations, but in lower concentrations it usually helps the Th1 to be expanded and activated and to secrete more cytokines such as IFN- $\gamma$  and TNF $\alpha$  (John, and Mishra, 2016). Experimentally induced colitis studies revealed that Gal-9 can attenuate colitis activity through its ability to enhance epithelial repairing along with

the induction of Th1 apoptosis and expansion of T-regs (Robinson et al., 2020). A recent study had stated a possible factor in IBD pathogenesis included instable membrane of the lysosome (found in the intestinal paneth cells) that leads to leakage of their contents and eventually triggering cell death and tissue damage, and this study results also showed that Gal-9 assisted in the lysosomal membrane integrity and cell survival (Sudhakar et al., 2020).

**Galectin-10:** It is human specific and expressed exclusively by cells of the immune system (neutrophils, eosinophils, basophils, and has regulatory action of T-reg cells), as the IBD has a characteristic eosinophilic infiltration that may secrete their by-products, afterward damaging the intestinal integrity causing ulcerations and abscess, Gal-10 has been linked to IBD pathogenesis because of its abundance in the human eosinophils (Melo et al., 2020). In UC patients, accumulation of eosinophil is believed to induce secretion of substance P causing obvious mucosal and neuronal damage (Tomizawa et al., 2022).

**Gal-11 and Gal-15** has been reported in sheep.

**Galectin-12:** It is expressed mainly in adipocytes where it participates in regulating lipolysis, fatty acid and body energy metabolism and also expressed at small levels in the heart, pancreas, thymus and peripheral leukocytes where it shows pro-apoptotic function and anti-proliferative function (Katzenmaier et al., 2019) most of the published studies are dealing with Gal-12's role in the metabolic disorders, obesity, and diabetes mellitus, otherwise, in the CRC, Gal-12 was down-regulated creating uncontrolled proliferation that assists immune evasion and protects the tumor from apoptotic stimuli, hence favoring the process of tumorigenesis (Wan et al., 2018).

**Galectin -13, -14, and -16:** They have placenta-specific expression, Gal-13 can induce activated T-cells apoptosis where which could be vital to preventing maternal immune cells from attacking the fetus (maternal immune tolerance), therefore; females with preeclampsia were showing lower serum level of Gal-13 (Sammar et al., 2019). In line with this, Gal-14 has been related to the placental development (trophoblast invasion and migration), normalizes the maternal immune tolerance by encouraging apoptosis of T-cells and other leukocytes, and eventually found to be downregulated in early pregnancy loss and preeclampsia (wang et al., 2021). Gal-16 has unique ligand binding patterns and is found in the placental tissue as well as in the brain and some cancers. In the placenta, Gal-16 was reported to regulate signal transduction pathways in B or T cells at the maternal-fetal interface maintaining the pregnancy integrity. While in the brain tissues, it plays a role in cellular differentiation and programmed cell death as seen in Gal-16's association with Alzheimer's disease (Blois et al., 2019). Regarding its role in cancers, Gal-16 in breast cancer is associated with the clinical pattern, histological type, disease stage and familial history of having cancer, and was reported as being higher by two-fold

in chronic myeloid leukemia than in healthy controls (Kaminker and Timoshenko, 2021).

## Conclusions

In human there are 12 members of Galectin family have been discovered and studied, some of them are widely distributed throughout tissues such as Gal-1, -3, and -9 but others are tissue or cell-specific such as Gal-2 in the large intestine, Gal-10 in the human eosinophils and Gal-13 in placenta. Although Gal-2 only presentation is in the large intestine and the Gal-4 is highly expressed in the gut as a whole but reports found that Gal-1 & Gal-3 are the most important players in the colonic inflammation, these molecules have great modulatory function on the immune and inflammatory compartments and they have been related to the pathogenesis of UC significantly either by being anti- or pro- inflammatory effectors. Gal-1, -3 & -9 were discovered earlier that permits the researchers to apply a lot of studies (animal & human) on them in different disorders including UC.

## Conflict of Interest

The author hereby declares no conflict of interest.

## Consent for publication

The author declares that the work has consent for publication.

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#### How to cite this article

Alahmed, A., Al-Rubae, E. and Noon, T. H. (2023). Human Galectines and their contribution in the chronic colonic inflammation (review). *Science Archives*, Vol. 4(2), 141-146. <https://doi.org/10.47587/SA.2023.4211>

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