

Anti-Galectin 3/LGALS3 Antibody Picoband® HRP Conjugated

Catalog Number: PB9081-HRP

About LGALS3

Galectin-3 (GAL3), also known as LGALS3, MAC2 or GALBP, is a member of the lectin family, of which 14 mammalian galectins have been identified. Galectin-3 is encoded by a single gene, it located on chromosome 14, locus q21-q22. It is expressed in the nucleus, cytoplasm, mitochondrion, cell surface, and extracellular space. Studies have also shown that the expression of galectin-3 is implicated in a variety of processes associated with heart failure, including myofibroblast proliferation, fibrogenesis, tissue repair, inflammation, and Ventricular remodeling. Galectin-3 is expressed in various tissues and organs, but is significantly absent in normal hepatocytes.

Overview

Product Name	Anti-Galectin 3/LGALS3 Antibody Picoband® HRP Conjugated
Reactive Species	Human
Clonality	Polyclonal
Formulation	Each vial contains 50% glycerol, 0.9% NaCl, 0.2% Na ₂ HPO ₄ .
Storage Instructions	At -20°C for one year from date of receipt. Avoid repeated freezing and thawing.
Host	Rabbit
Uniprot ID	P17931

Technical Details

Immunogen	E.coli-derived human Galectin 3 recombinant protein (Position: K139-I250). Human Galectin 3 shares 88% and 84% amino acid (aa) sequences identity with mouse and rat Galectin 3, respectively.
Cross Reactivity	No cross-reactivity with other proteins
Isotype	Rabbit IgG
Form	Liquid
Concentration	0.5 mg/mL
Purification	Immunogen affinity purified.
Conjugate	HRP
Suggested Dilutions	The intended application should be selected according to the customer's experimental requirements.

4 Publications Citing This Product

1. PubMed ID: 10.3389/fimmu.2021.639260, Galectin-Receptor Interactions Regulates Cardiac Pathology Caused by Trichinella spiralis Infection.
2. PubMed ID: 10.3892/etm.2019.7227, Expression of galectin-3 and apoptosis in placental villi from patients with missed abortion during early pregnancy
3. PubMed ID: 10.1155/2021/3210586, Insulin Receptor Substrate p53 Ameliorates High-Glucose-Induced Activation of NF-kappaB and Impaired Mobility of HUVECs

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