

**Blood Group Antigen Lewis B Antibody - With BSA and Azide**  
**Mouse Monoclonal Antibody [Clone 2-25LE; same as LWB01 ]**  
**Catalog # AH10472**

### Specification

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#### Blood Group Antigen Lewis B Antibody - With BSA and Azide - Product Information

Application	,14,3,
Primary Accession	<a href="#">P21217</a>
Other Accession	<a href="#">2525</a> , <a href="#">169238</a>
Reactivity	Human, Guinea Pig
Host	Mouse
Clonality	Monoclonal
Isotype	Mouse / IgG1, kappa
Calculated MW	45kDa KDa

#### Blood Group Antigen Lewis B Antibody - With BSA and Azide - Additional Information

Gene ID 2525

#### Other Names

Galactoside 3(4)-L-fucosyltransferase, 2.4.1.65, Blood group Lewis alpha-4-fucosyltransferase, Lewis FT, Fucosyltransferase 3, Fucosyltransferase III, FucT-III, FUT3, FT3B, LE

#### Format

200ug/ml of Ab purified from Bioreactor Concentrate by Protein A/G. Prepared in 10mM PBS with 0.05% BSA & 0.05% azide. Also available WITHOUT BSA & azide at 1.0mg/ml.

#### Storage

Store at 2 to 8°C. Antibody is stable for 24 months.

#### Precautions

Blood Group Antigen Lewis B Antibody - With BSA and Azide is for research use only and not for use in diagnostic or therapeutic procedures.

#### Blood Group Antigen Lewis B Antibody - With BSA and Azide - Protein Information

Name FUT3 ([HGNC:4014](#))

Synonyms FT3B, LE

#### Function

Catalyzes the transfer of L-fucose, from a guanosine diphosphate-beta-L-fucose, to both the subterminal N-acetyl glucosamine (GlcNAc) of type 1 chain (beta-D-Gal-(1->3)-beta-D-GlcNAc) glycolipids and oligosaccharides via an alpha(1,4) linkage, and the subterminal glucose (Glc) or GlcNAc of type 2 chain (beta-D-Gal-(1->4)-beta-D- GlcNAc) oligosaccharides via an alpha(1,3) linkage, independently of the presence of terminal alpha-L-fucosyl-(1,2) moieties on the terminal galactose of these acceptors (PubMed:<a href="http://www.uniprot.org/citations/11058871" target="\_blank">11058871</a>, PubMed:<a href="http://www.uniprot.org/citations/12668675"

target="\_blank">12668675</a>, PubMed:<a href="http://www.uniprot.org/citations/1977660" target="\_blank">1977660</a>). Through its catalytic activity, participates in the synthesis of antigens of the Lewis blood group system, i.e. Lewis a (Le(a)), lewis b (Le(b)), Lewis x/SSEA-1 (Le(x)) and lewis y (Le(y)) antigens (PubMed:<a href="http://www.uniprot.org/citations/11058871" target="\_blank">11058871</a>, PubMed:<a href="http://www.uniprot.org/citations/12668675" target="\_blank">12668675</a>, PubMed:<a href="http://www.uniprot.org/citations/1977660" target="\_blank">1977660</a>). Also catalyzes the transfer of L-fucose to subterminal GlcNAc of sialyl- and disialyl-lactotetraosylceramide to produce sialyl Lewis a (sLe(a)) and disialyl Lewis a via an alpha(1,4) linkage and therefore may regulate cell surface sLe(a) expression and consequently regulates adhesive properties to E-selectin, cell proliferation and migration (PubMed:<a href="http://www.uniprot.org/citations/11058871" target="\_blank">11058871</a>, PubMed:<a href="http://www.uniprot.org/citations/12668675" target="\_blank">12668675</a>, PubMed:<a href="http://www.uniprot.org/citations/27453266" target="\_blank">27453266</a>). Catalyzes the transfer of an L-fucose to 3'-sialyl-N-acetyllactosamine by an alpha(1,3) linkage, which allows the formation of sialyl-Lewis x structure and therefore may regulate the sialyl-Lewis x surface antigen expression and consequently adhesive properties to E-selectin (PubMed:<a href="http://www.uniprot.org/citations/11058871" target="\_blank">11058871</a>, PubMed:<a href="http://www.uniprot.org/citations/29593094" target="\_blank">29593094</a>). Prefers type 1 chain over type 2 acceptors (PubMed:<a href="http://www.uniprot.org/citations/7721776" target="\_blank">7721776</a>). Type 1 tetrasaccharide is a better acceptor than type 1 disaccharide suggesting that a beta anomeric configuration of GlcNAc in the substrate is preferred (PubMed:<a href="http://www.uniprot.org/citations/7721776" target="\_blank">7721776</a>). Lewis- positive (Le(+)) individuals have an active enzyme while Lewis-negative (Le(-)) individuals have an inactive enzyme (PubMed:<a href="http://www.uniprot.org/citations/1977660" target="\_blank">1977660</a>).

#### Cellular Location

Golgi apparatus, Golgi stack membrane; Single- pass type II membrane protein  
Note=Membrane-bound form in trans cisternae of Golgi

#### Tissue Location

Highly expressed in stomach, colon, small intestine, lung and kidney and to a lesser extent in salivary gland, bladder, uterus and liver.

#### Blood Group Antigen Lewis B Antibody - With BSA and Azide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

#### Blood Group Antigen Lewis B Antibody - With BSA and Azide - Images

#### Blood Group Antigen Lewis B Antibody - With BSA and Azide - Background

The Lewis histo-blood group system comprises a set of fucosylated glycosphingolipids that are synthesized by exocrine epithelial cells and circulate in body fluids. The glycosphingolipids function in embryogenesis, tissue differentiation, tumor metastasis, inflammation, and bacterial adhesion. They are secondarily absorbed to red blood cells giving rise to their Lewis phenotype. This gene is a member of the fucosyltransferase family, which catalyzes the addition of fucose to precursor

polysaccharides in the last step of Lewis antigen biosynthesis. It encodes an enzyme with alpha(1,3)-fucosyltransferase and alpha(1,4)-fucosyltransferase activities. Lewis blood group antigens are carbohydrate moieties structurally integrated in mucous secretions. Lewis antigen system alterations have been described in gastric carcinoma and associated lesions. Anomalous expression of Lewis B antigen has been found in some non-secretory gastric carcinomas and colorectal cancers.

### **Blood Group Antigen Lewis B Antibody - With BSA and Azide - References**

Richman, P.I. et al. 1987. Monoclonal antibodies to human colorectal epithelium: markers for differentiation and tumour characterization. *Int. J. Cancer*. 39: 317-328. Rouger, P.h., et al, eds. 1987. Proceedings of the first international work- shop on monoclonal antibodies against human red blood cells and related antigens (Paris, 1987). *Blood Transfusion Immunohaematology* 30: 353- 720. Bara, J. et al. 1988. Immunochemical characterization of mucins. Polypeptide (M1) and polysaccharide (A and Leb) antigens. *Biochem. J.* 254: 185-193. Torrado J, Correa P, Ruiz B, Bernardi P, Zavala D, Bara J. Lewis antigen alterations in gastric cancer precursors. *Gastroenterology*. 1992 Feb;102(2):424-30. Creuzot-Garcher C, Guertzider V, Assem M, Bron AM, Delannoy P, Bara. Alteration of sialyl Lewis epitope expression in pterygium. *J. Invest Ophthalmol Vis Sci*. 1999 Jul;40(8):1631