

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human β -1,4-Galactosyltransferase 1/B4GalT1 in direct ELISAs.
Source	Monoclonal Mouse IgG ₁ Clone # 394709
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human β -1,4-Galactosyltransferase 1/B4GalT1 Ala44-Ser398 Accession # P15291
Conjugate	Alexa Fluor 350 Excitation Wavelength: 346 nm Emission Wavelength: 442 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.

Immunocytochemistry Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

β 4GalT1 is one of seven β 1,4 galactosyltransferases that transfer galactose in a β 1,4 linkage to acceptor sugars including GlcNAc, and Glc, and Xyl. By sequence similarity, the β 4GalTs form four groups: β 4GalT1 and β 4GalT2, β 4GalT3 and β 4GalT4, β 4GalT5 and β 4GalT6, and β 4GalT7 (1). β 4GalT1 is unique among the seven enzymes because it can be expressed either as membrane associated form or secreted form (2). The secreted form is restricted to lactating mammary tissues where the enzyme forms a heterodimer with α -lactalbumin to catalyze the synthesis of lactose (3). The membrane form can reside either in the Golgi apparatus, where it adds galactose to N-acetylglucosamine residues, or on cell surface, where it functions as a recognition molecule during a variety of cell to cell and cell to matrix interactions, by binding to specific oligosaccharide ligands on opposing cells or in the extracellular matrix (4). The two enzymatic forms result from alternate transcription initiation sites and post-translational processing (5). Defects in β 4GalT1 are the cause of congenital disorder of glycosylation type 2D (CDG2D) (6). The amino acid sequence of human B4GALT1 is 88%, 87% and 71% identical to that of rat, mouse/canine and chicken.

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