

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse Siglec-E in direct ELISAs and Western blots. In direct ELISAs, less than 3% cross-reactivity with recombinant human (rh) Siglec-6, rhSiglec-7, and rhSiglec-9 is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse Siglec-E Gln20-Phe355 Accession # Q6PJ50
Conjugate	Alexa Fluor 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 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.

Western Blot	Optimal dilution of this antibody should be experimentally determined.
Immunohistochemistry	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

Siglecs are sialic acid specific I-type lectins that are characterized by an extracellular domain (ECD) with an N-terminal Ig-like V-set domain followed by varying numbers of Ig-like C2-set domains (1, 2). Mouse Siglec-E, also known as Myeloid Inhibitory Siglec (MIS), is an 80 - 85 kDa member of the CD33-related subfamily of Siglecs. It consists of a 335 amino acid (aa) ECD with one Ig-like V-set domain and two Ig-like C2-set domains, a 21 aa transmembrane segment, and a 93 aa cytoplasmic domain that contains two immunoreceptor tyrosine-based inhibitory motifs (ITIM) (3, 4). Rodent and primate Siglec gene families have significantly diverged, and Siglec-9 is the most likely human ortholog of mouse Siglec-E (1). Within the ECD, mouse Siglec-E shares 56% and 80% aa sequence identity with human Siglec-9 and rat Siglec-E, respectively. Siglec-E is expressed as a heavily N-glycosylated disulfide-linked homodimer and shows binding preference for disialic acids in the α2-8 linkage (3, 5). It is expressed on the surface of several hematopoietic cell types including neutrophils, NK cells, monocytes, peritoneal macrophages and B1 cells, and splenic myeloid dendritic cells and marginal zone B cells (5). Tyrosine phosphorylation of the cytoplasmic ITIMs mediates the association of Siglec-E with the phosphatases SHP-1 and SHP-2 (3, 4). Siglec-E is up-regulated and additionally phosphorylated following cellular stimulation by a variety of TLR agonists (6). Siglec-E signaling negatively regulates the LPS-induced production of TNF-α and IL-6 by macrophages (4, 6). Its up-regulation in macrophages parallels the development of endotoxin tolerance (6). Siglec-E recognition of sialylated determinants on virulent *T. cruzi* contributes to the suppression of dendritic cell IL-12 p40 production (7).

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