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Product Data Sheet

FITC anti-Siglec-E

Catalog # / Size:	3985560 / 100 µg 3985555 / 25 µg
Clone:	M1304A01
Isotype:	Rat IgG2a, κ
Reactivity:	Mouse
Concentration:	NULL

Applications:

Applications:	Flow Cytometry
Recommended Usage:	Each lot of this antibody is quality control tested by immunofluorescent staining with flow cytometric analysis. For flow cytometric staining, the suggested use of this reagent is ≤ 0.25 microg per million cells in 100 microL volume. It is recommended that the reagent be titrated for optimal performance for each application.
Application Notes:	This antibody works for western blotting under non-reducing conditions.

Description: Siglecs (sialic acid binding Ig-like lectins) are type I membrane proteins with an extracellular region containing a sialic acid binding V-set Ig-like domain at the N-terminus, followed by varying numbers of C2-set Ig domains. The cytoplasmic tails of all siglecs have tyrosine based motifs with a signaling function. Siglecs are widely expressed on hematopoietic cells, often in a cell-type-specific manner. Their ligands, sialic acids, are negatively charged monosaccharides found on cell-surface glycoproteins and glycolipids. Studies suggest that siglecs may participate in cell-cell interactions or act as receptors for the entry of viral or bacterial pathogens. In addition, the presence of immunoreceptor tyrosine-based inhibitory motifs (ITIM) in their cytoplasmic domain indicates that these molecules may play a role in the suppression of immunoreceptor signaling. Siglec-E is a mouse CD33-related siglec that selectively regulates early recruitment of neutrophils to the lung in acute lung inflammation induced by lipopolysaccharide. Siglec E-deficient mice exhibit exaggerated neutrophil recruitment that is reversible by using a blockade of the β2 integrin, CD11b. In addition, sialidase treatment of fibrinogen reverses the suppressive effect of Siglec-E on CD11b signaling. This suggests that sialic acid recognition by Siglec-E is required for its inhibitory function. These findings indicate that Siglec-E is an important negative regulator of neutrophil recruitment to the lungs and β2 integrin-dependent signaling.

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