Recombinant Cynomolgus Monkey FCAR/CD89

Catalog Number: 9516-FA

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived Gln22-Asn227, with a C-terminal 6-His tag Accession # XP005590398
N-terminal Sequence Analysis	No reults obtained. Gln 22 inferred from enzymatic pyroglutamate treatment revealing Glu23
Predicted Molecular Mass	24 kDa

SPECIFICATIONS

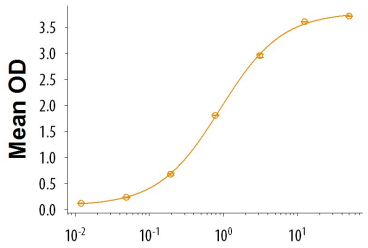
SDS-PAGE	33-70 kDa, reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When Recombinant Human IgA is immobilized at 5 µg/mL, 100 µL/well, the concentration of Recombinant Cynomolgus Monkey FCAR/CD89 that produces 50% of the optimal binding response is 0.5-2.5 µg/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 200 µg/mL in PBS.
Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

DATA

Bioactivity



When Human IgA is coated at 5 µg/mL (100 µL/well), Recombinant Cynomolgus Monkey FCAR/CD89 (Catalog # 9516-FA) binds with an ED_{50} of 0.5-2.5 µg/mL.

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BACKGROUND

FCAR, also called Fc alpha RI or CD89, is a variably glycosylated 50-100 kDa myeloid-specific type I transmembrane (TM) Fc receptor for IgA and is a member of the multi-chain immune recognition receptor (MIRR) family (1-3). Cynomolgus FCAR is predicted to contain a 21 amino acid (aa) signal sequence and 206 aa extracellular (ECD), 19 aa TM and 41 aa cytoplasmic domains (4). The Arg230 within the TM domain of human FCAR supports interaction with the ITAM-containing signaling subunit, FcR gamma, which contains a TM Asp (5-7). Two ECD C2-type Ig-like domains (EC1 and 2) are oriented at right angles (8). Up to two molecules of FCAR can bind one molecule of serum IgA via EC1 (8). Many human FCAR splice variants have been reported, but only two have been identified as proteins (9, 10). The a.2 form, which lacks 22 aa just prior to the TM domain, is exclusively expressed in alveolar macrophages. The a.3 form lacks EC2. FCAR binds monomeric, polymeric and secretory IgA, but does not mediate the barrier function of secretory IgA in mucosal epithelium (1-3). Shedding and circulation of polymeric IgA/FCAR immune complexes has been reported (11). Circulating neutrophils, eosinophils, and monocytes express FCAR (12). Tissue expression of FCAR is mainly from neutrophils; FCAR is down-regulated as monocytes differentiate to tissue macrophages (12). On neutrophils, a significant amount of FCAR lacks FcR gamma, but can still be endocytosed to early endosomes and recycled to the cell surface (5). Binding of serum IgA to FCAR is transient and anti-inflammatory, inhibiting IgG or IgE-induced degranulation (6). Sustained aggregation of FCAR results in inflammatory responses (6). FcR gamma signaling is required for these and for transport to late endosomes (5-7). Within ECD, Cynomolgus FCAR shows 83% aa identity with human FCAR. No ortholog occurs in mouse. FCAR structure resembles the KIR/ILT/LIR/MIR family more than other IgA receptors, including pIgR, Fc alpha /µR, asialoglycoprotein receptor (ASGR1) and transferrin receptor (TfR) (1-3).

References:

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