

DESCRIPTION

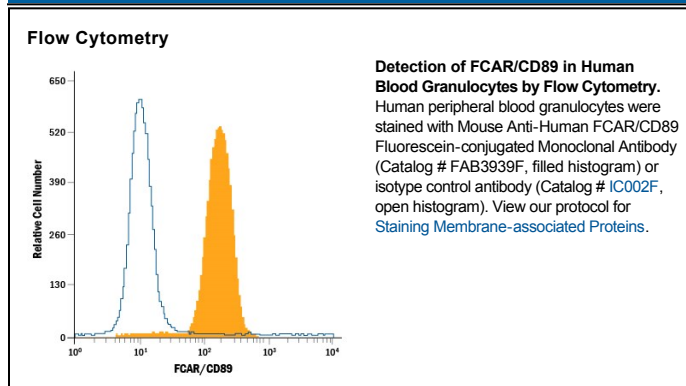
Species Reactivity	Human
Specificity	Detects human FCAR/CD89 in direct ELISAs. In direct ELISAs, no cross-reactivity with Fcγ RIA, RIIA, or RIIB is observed.
Source	Monoclonal Mouse IgG ₁ Clone # 488032
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	NS0 mouse myeloma cell line transfected with human FCAR/CD89 Gln22-Lys287 Accession # P24071
Conjugate	Fluorescein Excitation Wavelength: 488 nm Emission Wavelength: 515-545 nm (FITC)
Formulation	Supplied in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details. *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.

	Recommended Concentration	Sample
Flow Cytometry	10 μL/10 ⁶ cells	See Below

DATA



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. <ul style="list-style-type: none"> 12 months from date of receipt, 2 to 8 °C as supplied.

BACKGROUND

FCAR, also known as FcαRI or CD89, is a variably glycosylated 50-100 kDa myeloid-specific type I transmembrane (TM) Fc receptor for IgA that is a member of the multichain immune recognition receptor (MIRR) family (1-3). Human FCAR contains a 21 amino acid (aa) signal sequence and extracellular (ECD), TM and cytoplasmic domains of 206, 19 and 41 aa, respectively (4). Arg230 within the TM domain supports interaction with the ITAM-containing signaling subunit, FcRγ, 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 splice variants have been reported, but only two have been identified as proteins (9, 10). The α.2 form, which lacks 22 aa just prior to the TM domain, is exclusively expressed in alveolar macrophages. The α.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 downregulated as monocytes differentiate to tissue macrophages (12). On neutrophils, a significant amount of FCAR lacks FcRγ, 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). FcRγ signaling is required for these and for transport to late endosomes (5-7). Human FCAR shows 55-58% aa identity with rat, horse and cow FCAR. No ortholog occurs in mouse. FCAR structure resembles the KIR/ILT/LIR/MIR family more than other IgA receptors, including pIgR, Fcα/μR, Asialoglycoprotein Receptor (ASGR1) and Transferrin Receptor (TfR) (1-3).

References:

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