

Human FCAR/CD89 Biotinylated Antibody

Antigen Affinity-purified Polyclonal Sheep IgG Catalog Number: BAF3939

DECODIDEION		
DESCRIPTION		
Species Reactivity	Human	
Specificity	Detects human FCAR in Western blots. In Western blots, less than 1% cross-reactivity with recombinant human (rh) Fcγ R1α, rhF	cγ R2α,
	and rhFcγ R3β is observed.	
Source	Polyclonal Sheep IgG	
Purification	Antigen Affinity-purified	
Immunogen	Mouse myeloma cell line NS0-derived recombinant human FCAR	
	Gln22-Asn227	
	Accession # P24071	
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.	
APPLICATIONS		
Please Note: Optimal diluti	utions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.	
	Recommended Sample	
	Concentration	
Western Blot	0.1 μg/mL Recombinant Human FCAR/CD89 (Catalog # 3939-FA)	
PREPARATION AND	D STORAGE	
Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.	
Shipping	The product is shipped at ambient temperature. 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. 	
	 6 months, -20 to -70 °C under sterile conditions after reconstitution. 	

FCAR, also called FcaRl 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, FcRy, 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 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 downregulated as monocytes differentiate to tissue macrophages (12). On neutrophils, a significant amount of FCAR lacks FcRy, 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:

- 1. Wines, B. D. and P. M. Hogarth (2006) Tissue Antigens 68:103.
- Otten, M. A. and M. van Egmon (2004) Immunol. Lett. 92:23.
- Montiero, R. C. and J. G. J. van de Winkel (2003) Annu. Rev. Immunol. 21:177.
- Maliszewski, C. R. et al. (1990) J. Exp. Med. 172:1665.
- Launay, P. et al. (1999) J. Biol. Chem. 274:7216.
- Pasquier, B. et al. (2005) Immunity 22:31.
- Shen, L. et al. (2001) Blood 97:205.
- 8. Herr, Y. et al. (2003) Nature 423:614.
- Patry, C. et al. (1996) J. Immunol. 156:4442.
- Togo, S. et al. (2003) FEBS Lett. 535:205. 10.
- van der Boog, P. J. M. et al. (2002) J. Immunol. 168:1252.
- Hamre, R. et al. (2003) Scand. J. Immunol. 57:506. 12.

