

DESCRIPTION

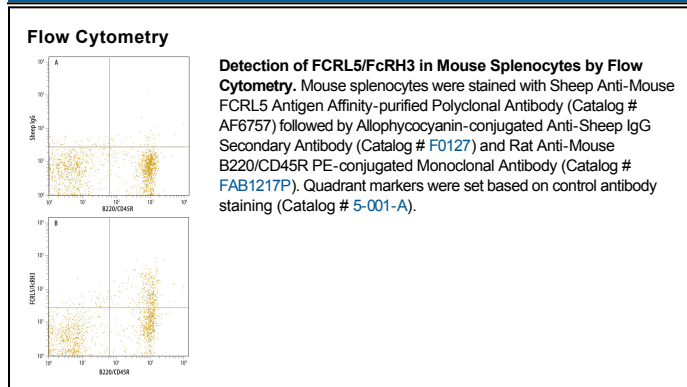
Species Reactivity	Mouse
Specificity	Detects mouse FCRL5/FcRH3 in direct ELISAs. In direct ELISAs, less than 1% cross-reactivity with recombinant human FCRL5 is observed.
Source	Polyclonal Sheep IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse FCRL5/FcRH3 Gln27-Ala496 (predicted) Accession # NP_899045
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

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	2.5 µg/10 ⁶ cells	See Below
CyTOF-ready	Ready to be labeled using established conjugation methods. No BSA or other carrier proteins that could interfere with conjugation.	

DATA



PREPARATION AND STORAGE

Reconstitution	Sterile PBS to a final concentration of 0.2 mg/mL.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> ● 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.

BACKGROUND

Fc Receptor-Like 5 (FCRL5), also known as FcRH3 (FcRH5 in human), IRTA2, and CD307e, is a 90-95 kDa member of the FCRL family of proteins whose amino acid (aa) sequence is reminiscent of that for classical Fc receptors. FCRL molecules are type 1 transmembrane proteins that contain from three to nine immunoglobulin-like domains. They are differentially expressed within the B cell lineage, and can either promote or inhibit B cell proliferation and activation (1, 2, 3). Mature mouse FCRL5 consists of a 470 aa extracellular domain (ECD), a 21 aa transmembrane segment, and a 79 aa cytoplasmic region. The ECD contains five Ig-like domains, while the cytoplasmic region possesses one ITAM-like motif and one immunotyrosine inhibitory motif (ITIM) (1, 3, 4). There are two major alleles for FCRL5 in mouse. The first was just described, and is found in BALB/c plus NZB mouse strains. The second is found in C57BL/6 mice, and differs by eleven scattered aa in the ECD. This creates one additional N-linked glycosylation site, and increases the SDS-PAGE MW by 5 kDa (3). Alternate splicing of mouse FCRL5 generates at least one additional isoform that lacks the first Ig-like domain (aa 3-90 of the mature molecule) (4). Human FCRL5, by contrast, contains up to nine Ig-like domains in a highly variable ECD, and over common regions, mouse and human FCRL5 share 49% aa sequence identity. FCRL5 expression is restricted to mature B lineage cells in lymphoid tissues and blood, and is particularly noted to be expressed on T-independent marginal zone and B1 B cells (3-8). Its ligation inhibits signaling through the B cell antigen receptor (9). Epstein-Barr virus transformation of B cells induces the up-regulation of surface FCRL5 by a direct effect of its EBNA2 protein on FCRL5 gene transcription (10). FCRL5 on B cells functions as a receptor for the orthopoxvirus MHC class I-like protein OMCP (11). And based on the literature and R&D Systems testing, both mouse and human FCRL5 will bind to purified IgG with high affinity (5). In human, the FCRL5 gene maps to the 1q21 chromosomal locus, a common site of rearrangements in B cell malignancies. Notably, the FCRL5 protein is preferentially expressed in cell lines with 1q21 abnormalities, and is up-regulated on tumor cells in some types of B cell malignancies (5, 7, 12-14). In addition, soluble FCRL5 is elevated in the serum of many B cell leukemia patients (13, 15).

References:

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