

DESCRIPTION

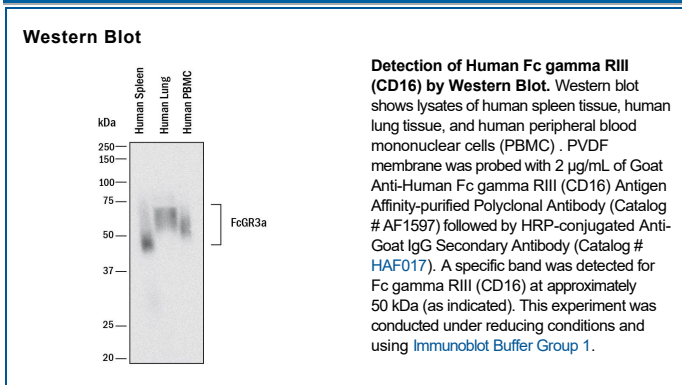
Species Reactivity	Human
Specificity	Detects human Fcγ RIIIA/B (CD16) in direct ELISAs and Western blots. In these formats, approximately 5% cross-reactivity with recombinant human Fcγ RIIA is observed and less than 2% cross-reactivity with recombinant mouse CD16 is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Fcγ RIIIB (R&D Systems, Catalog # 1597-FC) Thr20-Gln208 Accession # O75015
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 either lyophilized or 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
Western Blot	2 μg/mL	See Below
Blockade of Receptor-ligand Interaction	In a functional ELISA, 2-6 μg/mL of this antibody will block 50% of the binding of 5 μg/mL of human IgG to immobilized Recombinant Human Fcγ RIIIB/CD16b (Catalog # 1597-FC) coated at 5 μg/mL (100 μL/well). At 50 μg/mL, this antibody will block >90% of the binding.	

DATA



PREPARATION AND 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. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <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

Receptors for the Fc region of IgG (Fcγ R) are members of the Ig superfamily. Based on their genetic organization and molecular structure, three classes of human Fcγ Rs: RI (CD64), RII (CD32), and RIII (CD16), which generate multiple isoforms, are recognized (1 - 3). These receptors function in the activation or inhibition of immune responses. The activating-type receptor either has, or associates non-covalently with an accessory subunit (Fcγ Rγ or ζ chain) that has an immunoreceptor tyrosine-based activation motif (ITAM) in its cytoplasmic domain. In contrast, the inhibitory receptor (Fcγ RIIb) has a built-in immunoreceptor tyrosine-based inhibitory motif (ITIM) in its own cytoplasmic domain. Fcγ RI is a high-affinity receptor that binds monomeric IgG. Both Fcγ RII and RIII are low-affinity receptors that bind IgG in the form of immune complexes. Two genes for human Fcγ RIII, A and B, encoding a transmembrane receptor and a glycosylphosphatidylinositol (GPI) anchored protein, respectively, have been identified. Three allelic variants of Fcγ RIIb, NA-1, NA-2, and SH, exist. A soluble form of Fcγ RIIb corresponding to the extracellular region of the receptor is produced by proteolytic cleavage and circulates in plasma and other body fluids. The extracellular domains of Fcγ RIIIA and B share 97% amino acid sequence homology. Whereas Fcγ RIIIA is expressed on most effector cells of the immune system including macrophage, monocyte, NK cells, mast cells, eosinophils, dendritic cells, and Langerhans cells, Fcγ RIIb is selectively expressed in neutrophils and eosinophils. Signaling through Fcγ RIIIA results in oxidative burst, cytokine release and phagocytosis by macrophages, antibody-dependent cellular cytotoxicity by natural killer cells and degranulation of mast cells. By contrast, Fcγ RIIb is a decoy receptor that binds IgG complexes without triggering activation. Soluble Fcγ RIIb has a regulatory role in inflammatory processes (4). It interacts with complement receptors CR3 and CR4 on monocytes to induce the production of pro-inflammatory cytokines.

References:

1. van de Winkel, J. and P. Capes (1993) *Immunol. Today* **14**:215.
2. Ravetch, J.V. and S. Bolland (2001) *Annu. Rev. Immunol.* **19**:275.
3. Takai, T. (2002) *Nature Rev. Immunol.* **2**:580.
4. Gauchat, G.J. *et al.* (1996) *J. Immunol.* **157**:1184.