

**DESCRIPTION**

**Source** Mouse myeloma cell line, NS0-derived human Fc gamma RIIB/C (CD32b/c) protein  
Ala46-Pro217, with a C-terminal 10-His tag  
Accession # P31994

**N-terminal Sequence Analysis** Ala46

**Predicted Molecular Mass** 21 kDa

**SPECIFICATIONS**

**SDS-PAGE** 25-35 kDa, reducing conditions

**Activity** Measured by its ability to bind human IgG with an estimated  $K_d < 200$  nM.

**Endotoxin Level** <1.0 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 100 µg/mL in sterile PBS.

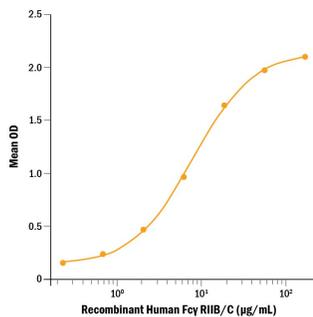
**Shipping** The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

**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.
- 3 months, -20 to -70 °C under sterile conditions after reconstitution.

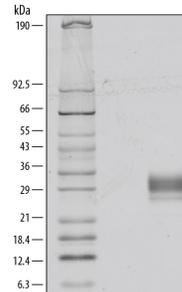
**DATA**

**Binding Activity**



Recombinant Human Fcγ RIIB/C (CD32b/c) (Catalog # 1875-CD) binds human IgG with an estimated  $K_d < 200$  nM.

**SDS-PAGE**



1 µg/lane of Recombinant Human Fcγ RIIB/C (CD32b/c) was resolved with SDS-PAGE under reducing (R) conditions and visualized by silver staining, showing multiple bands at 27-35 kDa.

**BACKGROUND**

Receptors for the Fc region of IgG (Fcγ Rs) are members of the Ig superfamily that function in the activation or inhibition of immune responses such as degranulation, phagocytosis, ADCC (antibody-dependent cellular toxicity), cytokine release, and B cell proliferation (1-3). The Fcγ Rs have been divided into three classes based on close relationships in their extracellular domains; these groups are designated Fcγ RI (also known as CD64), Fcγ RII (CD32), and Fcγ RIII (CD16). Each group may be encoded by multiple genes and exist in different isoforms depending on species and cell type. The CD64 proteins are high affinity receptors ( $\sim 10^{-8}$ - $10^{-9}$  M) capable of binding monomeric IgG, whereas the CD16 and CD32 proteins bind IgG with lower affinities ( $\sim 10^{-6}$ - $10^{-7}$  M) only recognizing IgG aggregates surrounding multivalent antigens (1, 4). Fcγ Rs that deliver an activating signal either have an intrinsic immunoreceptor tyrosine-based activation motif (ITAM) within their cytoplasmic domains or associate with one of the ITAM-bearing adapter subunits, FcRγ or ζ (3, 5). The only inhibitory member in human and mouse, Fcγ RIIb, has an intrinsic cytoplasmic immunoreceptor tyrosine-based inhibitory motif (ITIM). The coordinated functioning of activating and inhibitory receptors is necessary for successful initiation, amplification, and termination of immune responses (5).

Three distinct genes encode the human CD32 group, and the protein products are designated Fcγ RIIa, B, and C (1). These receptors are glycoproteins of approximately 40 kDa having two extracellular Ig-like domains. The Fcγ RII proteins share 94-99% amino acid identity in their extracellular domains but differ substantially in their transmembrane and cytoplasmic domains. Fcγ RIIa associates with FcRγ, and delivers an activating signal upon ligand binding (3, 5). In contrast, Fcγ RIIb delivers an inhibitory signal. Fcγ RIIc represents an unequal cross-over event between the IIA and IIB genes. Its extracellular domain shares 99% amino acid identity with Fcγ RIIb, but a portion of the cytoplasmic domain is closely related to Fcγ RIIa. Fcγ RII proteins are expressed on cells of both myeloid and lymphoid lineages as well as on cells of non-hematopoietic origin.

**References:**

1. van de Winkel, J. and P. Capes (1993) *Immunol. Today* **14**:215.
2. Raghaven, M. and P. Bjorkman (1996) *Annu. Rev. Cell Dev. Biol.* **12**:181.
3. Ravetch, J. and S. Bolland (2001) *Annu. Rev. Immunol.* **19**:275.
4. Takai, T. (2002) *Nature Rev. Immunol.* **2**:580.
5. Ravetch, J. and L. Lanier (2000) *Science* **290**:84.