Human Fcγ RI/CD64 Antibody

Antigen Affinity-purified Polyclonal Goat IgG
Catalog Number: AF1257

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Fcγ RI/CD64 in direct ELISAs and Western blots. In direct ELISAs, approximately 40% cross-reactivity with recombinant mouse Fcγ RI is observed and 10% cross-reactivity with recombinant human (rh) Fcγ RIIA and rhFcγ RIIIB is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Fcγ RI/CD64 Gln16-Pro288 Accession # P12314.2
Endotoxin Level	<0.2 EU per 1 µg of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

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	0.1 µg/mL	Recombinant Human Fcγ RI/CD64 (Catalog # 1257-FC)
Blockade of Receptor-ligand Interaction	In a functional ELISA, 1-3 µg/mL of this antibody will block 50% of the binding of 600 ng/mL of human IgG to immobilized Recombinant Human Fcγ RI/CD64 (Catalog # 1257-FC) coated at 1 µg/mL (100 µL/well). At 100 µg/mL, this antibody will block >90% of the binding.	

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.
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 from date of receipt, 2 to 8 °C, reconstituted. ● 6 months from date of receipt, -20 to -70 °C, reconstituted.

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 (~10^{-8} - 10^{-9} M) capable of binding monomeric IgG, whereas the CD16 and CD32 proteins bind IgG with lower affinities (~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, Fc Rγ 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 highly homologous genes (A, B, and C) sharing 98% identity at the nucleotide level have been identified for the human CD64 group (1). Fcγ RI is transmembrane protein with three extracellular Ig-like domains, and it delivers an activating signal via the associated Fc Rγ accessory chain. The genes for Fcγ RIB and Fcγ RIC contain stop codons within their membrane proximal Ig-like domains indicating possible secreted receptors (1, 6). An mRNA splice variant of Fcγ RIB has a deletion of the membrane-proximal Ig-like domain and encodes a putative transmembrane receptor (6). The high affinity recognition of IgG by Fcγ RI permits the triggering of effector responses at low IgG concentrations typical of early immune responses (2). Fcγ RI is expressed constitutively on monocytes and macrophages and can be induced on neutrophils and eosinophils (1, 4). Its expression is up-regulated during bacterial infections and sepsis.

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.
6. Ernst, L. *et al.* (1998) Mol Immunol. **35**:943.