

DESCRIPTION

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| Species Reactivity | Human |
| Specificity | Detects human Fc γ RI in direct ELISAs and Western blots. In Western blots, this antibody does not cross-react with recombinant mouse (rm) Fc γ RI, rmFc γ RII, recombinant human (rh) Fc γ RIIA, rhFc γ RIIIB, or rhFc γ RIIIB. |
| Source | Monoclonal Mouse IgG ₁ Clone # 276426 |
| Purification | Protein A or G purified from hybridoma culture supernatant |
| Immunogen | Mouse myeloma cell line NS0-derived recombinant human Fc γ RI Gln16-Pro288 Accession # P12314.2 |
| Conjugate | Alexa Fluor 405 Excitation Wavelength: 405 nm Emission Wavelength: 421 nm |
| Formulation | Supplied 0.2 mg/mL in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details. *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions. |

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 | 0.25-1 µg/10 ⁶ cells | Human peripheral blood monocytes |

PREPARATION AND STORAGE

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| Shipping | The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below. |
| Stability & Storage | Protect from light. Do not freeze. <ul style="list-style-type: none"> 12 months from date of receipt, 2 to 8 °C as supplied. |

BACKGROUND

Receptors for the Fc region of IgG (Fc γ 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 Fc γ Rs have been divided into three classes based on close relationships in their extracellular domains; these groups are designated Fc γ RI (also known as CD64), Fc γ RII (CD32), and Fc γ 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). Fc γ 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 γ R γ or ζ (3, 5). The only inhibitory member in human and mouse, Fc γ 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). Fc γ RI is transmembrane protein with three extracellular Ig-like domains, and it delivers an activating signal via the associated Fc γ R γ accessory chain. The genes for Fc γ RIB and Fc γ RIC contain stop codons within their membrane proximal Ig-like domains indicating possible secreted receptors (1, 6). An mRNA splice variant of Fc γ 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 Fc γ RI permits the triggering of effector responses at low IgG concentrations typical of early immune responses (2). Fc γ 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.

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