# Fc gamma RIIIA/CD16a Protein, Human (CHO, His)

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| **Cat. No.:** | HY-P75207 |
| **Synonyms:** | Low affinity immunoglobulin gamma Fc region receptor III-A; FcRIIIa; FcR-10; CD16a; FCGR3A; IGFR3 |
| **Species:** | Human |
| **Source:** | CHO |
| **Accession:** | P08637 (G17-Q208) |
| **Gene ID:** | 2214 |
| **Molecular Weight:** | Approximately 23.3 kDa |

## PROPERTIES

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| **Appearance** | Lyophilized powder. |
| **Formulation** | Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. |
| **Endotoxin Level** | <1 EU/μg, determined by LAL method. |
| **Reconsititution** | It is not recommended to reconstitute to a concentration less than 100 μg/mL in $ddH_2O$. |
| **Storage & Stability** | Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage. |
| **Shipping** | Room temperature in continental US; may vary elsewhere. |

## DESCRIPTION

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| **Background** | Fc gamma RIIIA/CD16a Protein serves as a receptor for the invariable Fc fragment of immunoglobulin gamma (IgG), optimally activated upon binding clustered antigen-IgG complexes displayed on cell surfaces, initiating antibody-dependent cellular cytotoxicity (ADCC). This process involves the lysis of antibody-coated cells, preventing inappropriate effector cell activation in the absence of an antigenic trigger. The protein mediates IgG effector functions on natural killer (NK) cells, binding antigen-IgG complexes generated during infection to trigger NK cell-dependent cytokine production and degranulation. Fc gamma RIIIA/CD16a is crucial in generating memory-like adaptive NK cells that efficiently eliminate virus-infected cells via ADCC. It regulates NK cell survival, proliferation, and prevents NK cell progenitor apoptosis. As an Fc-binding subunit, it associates with CD247 and/or FCER1G adapters to form functional signaling complexes, leading to intracellular signaling cascades that drive NK cell activation. The protein also plays a role in mediating the antitumor activities of therapeutic antibodies, triggering TNFA-dependent ADCC of IgG-coated tumor cells and enhancing ADCC in response to afucosylated IgGs. In the context of Dengue virus infection, Fc gamma RIIIA/CD16a is involved in pathogenesis through an antibody-dependent enhancement (ADE) mechanism, facilitating virus entry into myeloid cells and subsequent viral replication during secondary infections. |

**Caution: Product has not been fully validated for medical applications. For research use only.**

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