

Fc gamma RIIIA/CD16a Protein, Human (F176V, CHO, His)

Cat. No.:	HY-P75210
Synonyms:	Low affinity immunoglobulin gamma Fc region receptor III-A; FcRIIIa; FcR-10; CD16a; FCGR3A; IGFR3
Species:	Human
Source:	CHO
Accession:	P08637 (G17-Q208, F176V)
Gene ID:	2214
Molecular Weight:	40-45 kDa

PROPERTIES

Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 µm filtered solution of PBS, pH 7.2. 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.
Reconstitution	It is not recommended to reconstitute to a concentration less than 100 µg/mL in ddH ₂ O.
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

Background	<p>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.</p>
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Caution: Product has not been fully validated for medical applications. For research use only.

Tel: 609-228-6898

Fax: 609-228-5909

E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA