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InnoCyto Inc.

15375 Barranca Pkwy, Suite I-103
Irvine, CA 92618
Email: info@innocyto.com
Phone: 1-949-418-7543

PE Human CD32a R167 (FcγRIIA) Protein (C-His)

Catalog Number:	820401, 820402
Size:	25 ug, 100 ug
Target Name:	CD32a, FCGR2A, CD32, FCG2 , FCGR2A1, IGFR2
Regulatory Status:	RUO

PRODUCT DETAILS

Application:	Flow Cytometry
Format:	Liquid, PE
Expression Host:	CHO
Species:	Human
Sources:	Recombinant CD32a R167 /Fc gamma RIIa (Ala 36 - Ile 218) with C-terminus His-tag is expressed in CHO cell and conjugated to PE.
Accession Number:	Q92835
Molecular Weight:	The protein has a predicted molecular weight of 21.9 kDa. Under DTT-reducing conditions, it migrates at approximately 35 kDa on SDS-PAGE prior to conjugation.
Affinity Tag:	C-His
Formulation:	1xPBS buffer, pH7.4, 0.09% NaN3 with a carrier protein
Endotoxin level:	Not tested
Protein Concentration:	25μg size is bottled at 0.2 mg/mL concentration. 100 μg size is bottled at lot specific concentration.
Storage and Handling:	Briefly centrifuge the vial upon receipt. An unopened vial may be stored at 2–8°C for up to six months.

BACKGROUND INFORMATION

CD32A R167 refers to the arginine (R) variant at position 167 of the FcγRIIA (CD32A) receptor, one of two common allelic forms resulting from a functionally significant single nucleotide polymorphism. CD32A is the most widespread activating Fc gamma receptor in humans, expressed on monocytes, macrophages, neutrophils, dendritic cells, platelets, and mast cells, where it mediates critical antibody-dependent immune functions. The receptor binds to the Fc portion of IgG antibodies and triggers cellular responses including phagocytosis of antibody-opsonized pathogens, antibody-dependent cellular cytotoxicity (ADCC), immune complex clearance, and inflammatory mediator release. The R167 variant represents one of the two major polymorphic forms, with the alternative being the histidine (H167) variant, and this genetic variation has profound effects on receptor function and clinical outcomes.

Structurally, the R167 variant contains an arginine residue at position 167 within the second extracellular immunoglobulin-like domain (EC2), which constitutes the primary IgG-binding interface. The arginine at this position is a positively charged, bulky amino

acid that affects the electrostatic and steric properties of the binding pocket. Compared to the H167 variant, the R167 form exhibits reduced binding affinity for human IgG2 antibodies due to electrostatic repulsion and suboptimal spatial complementarity with the IgG2 Fc region. This structural difference does not alter the overall receptor architecture, transmembrane domain, or the cytoplasmic immunoreceptor tyrosine-based activation motif (ITAM), but it significantly modulates the efficiency of ligand engagement and subsequent receptor activation thresholds.

The primary ligands for CD32A R167 are IgG antibodies, particularly IgG1 and IgG3 subclasses, when present in immune complexes or aggregated forms. While the R167 variant can bind all IgG subclasses, it shows markedly reduced affinity for IgG2 compared to the H167 variant. This differential binding has functional consequences: the R167 variant is less efficient at mediating immune responses triggered by IgG2-containing immune complexes, which can affect clearance of certain pathogens and immune complexes that preferentially elicit IgG2 responses.

In disease contexts, the CD32A R167 polymorphism has been associated with altered susceptibility to various conditions. Individuals homozygous for the R167 variant show increased risk of systemic lupus erythematosus (SLE) and other autoimmune diseases, likely due to impaired clearance of IgG2-containing immune complexes, leading to their deposition in tissues and chronic inflammation. The R167 variant has also been linked to increased susceptibility to certain bacterial infections, particularly those involving encapsulated organisms where IgG2 antibodies play a protective role. In cardiovascular disease, the R167 variant may influence thrombotic risk through altered platelet FcγRIIA function. Therapeutically, the R167 polymorphism has important implications for antibody-based treatments. Patients carrying the R167 variant may show reduced responses to therapeutic monoclonal antibodies that rely on FcγRIIA-mediated effector functions, particularly those of the IgG2 subclass. This has prompted development of Fc-engineered antibodies with enhanced binding to the R167 variant to ensure therapeutic efficacy across all patient genotypes. Additionally, pharmacogenomic approaches are being explored to stratify patients based on FcγRIIA polymorphisms for personalized selection of antibody therapies. Understanding the R167 variant is crucial for optimizing immunotherapy outcomes and predicting individual responses to antibody-based treatments in cancer, autoimmune diseases, and infectious diseases.

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