Recombinant Human PILR-α Fc Chimera

Catalog Number: 10802-PR

DESCRIPTION

Source

Chinese Hamster Ovary cell line, CHO-derived human PILR-alpha protein

Human PILR-alpha (Thr25-Thr196) Accession # Q9UKJ1.3	IEGRMD	Human IgG_1 (Pro100-Lys330)

N-terminus C-terminus

N-terminal Sequence Analysis	Thr25
Predicted Molecular Mass	46 kDa

SPECIFICATIONS

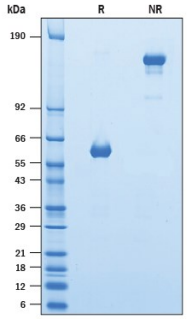
SDS-PAGE	57-64 kDa, under reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When Recombinant Human PANP Fc Chimera (Catalog # 7920-PN) is coated at 5.00 µg/mL (100 µL/well), Recombinant Human PILR- α Fc Chimera binds with an ED_{50} of 0.150- 0.900 µg/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 500 µg/mL in 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, 2 to 8 °C under sterile conditions after reconstitution. • 3 months, -20 to -70 °C under sterile conditions after reconstitution.

DATA

SDS-PAGE



Recombinant Human PILR-alpha Fc Chimera Protein SDS-PAGE. 2 µg/lane of Recombinant Human PILR-alpha Fc Chimera (Catalog # 10802-PR) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 57-64 kDa and 114-128 kDa, respectively.

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BACKGROUND

Paired immunoglobulin-like type 2 receptor-alpha (PILRA) is one of two members that belong to a small family of immunoregulatory Ig-superfamily receptors (1-4). It is a counterpart to PILRB and the PILRs represent one of many pairs of Ig-like domain-containing receptors that participate in immune regulation (1, 2). Mature human PILRA consists of an extracellular domain (ECD) with one V-type Ig-like domain, a transmembrane domain, and a cytoplasmic domain with two immunoreceptor Tyr-based inhibitory (ITIM) motifs. Given that ITIMs are known to interact with phosphatases such as PTPN6 and PTPN11, the presence of these motifs makes PILRA an inhibitory receptor (1-4). The ECD of human PILRA shares 42% and 40% amino acid sequence identity with mouse and rat PILRA, respectively. Three potential isoforms for human PILRA have been reported. PILRA is expressed by neutrophils, macrophages, monocytes, mast cells, APCs, microglia, neurons, cardiac muscle and renal proximal plus pancreatic duct eipthelium (4, 7, 8). It has multiple binding partners, including CD99 (4, 9), glycoprotein B/gB of HSV1 (in human) (7), PANP (PILR-associated neural protein) (8) and NPDC1 plus collectin-12 (10). Although PILRA and PILRB are highly similar in their ECD amino acid sequence, they do not necessarily share the same ligands, as PILRB fails to bind to gB and PANP (8, 10). PILRA binding appears to be dependent upon the presence of a poorly-defined peptide sequence coupled to a sialylated, O-linked carbohydrate motif but its exact function remains unclear (5, 9-12). Up-regulation of PILRA in the early stage of immune reaction and its subsequent binding to CD99 may lead to a down-regulation of the inflammatory response (10). Genome-wide association studies (GWAS) have linked PILRA to Alzheimer's Disease (AD) through association of PILRB with ZCWPW1 (13,14). It was further supported that ZCWPW1 locus SNP rs1476679 was associated with reduced PILRA levels suggesting a potential role for the gene in AD (15). The missense variant (G78R, rs1859788) of PILRA is thought to be the causal allele for the confirmed AD risk locus. The variant reduced the binding of PILRA to several ligands including a novel ligand, complement component 4A, and herpes simplex virus 1 (HSV-1) glycoprotein B (16). The observed protection from AD risk by PILRA G78R variant provided a new candidate for the therapeutic target.

References:

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