

DESCRIPTION

Source	Human embryonic kidney cell, HEK293-derived		
	Human PILR-β (Gln20-Ala189) Accession # Q9UKJ0-1	IEGRMD	Human IgG ₁ (Pro100-Lys330)
	N-terminus		C-terminus

N-terminal Sequence Analysis No results obtained. Gln20 predicted.

Structure / Form Disulfide-linked homodimer

Predicted Molecular Mass 45.7 kDa (monomer)

SPECIFICATIONS

SDS-PAGE 57-66 kDa, reducing conditions

Activity Measured by its binding ability in a functional ELISA. Recombinant Human CL-P1/COLEC12 (Catalog # 2690-CL) immobilized at 2 µg/mL, 100 µg/mL, can bind to Recombinant Human PILR-β Fc Chimera with an ED₅₀ of 0.12-0.72 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

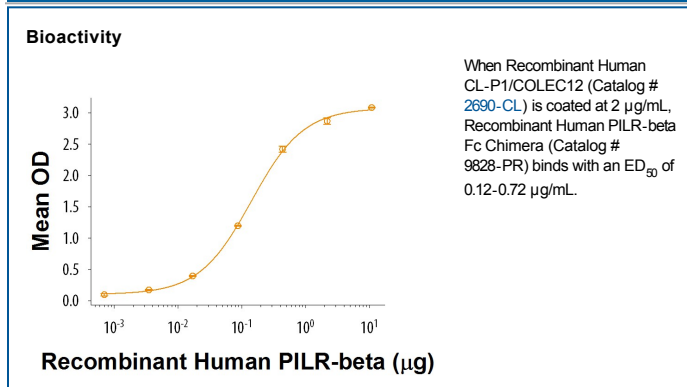
Reconstitution Reconstitute at 500 µg/mL in PBS.

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



BACKGROUND

Paired immunoglobulin-like type 2 receptor beta (PILR- β) is a type I transmembrane (TM) glycoprotein belonging to the Ig superfamily. PILR- β is the activating counterpart to the immunoreceptor tyrosine-based inhibitory motif (ITIM) containing PILR- α inhibitory receptor (1). Mature human PILR- β is a 208 amino acid (aa) protein containing a 172 aa V-type Ig-like extracellular domain (ECD) with a siglec-like fold, a single TM, and a truncated cytoplasmic tail (2, 3). The TM of PILR- β contains a positively-charged residue which interacts with immunoreceptor tyrosine-based activation (ITAM)-bearing adaptor molecules (2). The ECD of mature human PILR- β shares 40% aa sequence identity with its mouse counterpart. PILR- β is expressed on myeloid cells, such as natural killer, macrophage, and dendritic cells, as well as resident cells of the central nervous system, such as microglial cells (2,4). It is a binding partner for DAP12 and CD99, and has been shown to play an important role in innate immunity and inflammation (4-6). The PILR- α/β pair have also been shown to regulate cell signaling via association with SHP-1 (7). Experiments studying the effects of *S. aureus* and *T. gondii* infections in mice have shown that up-regulation of PILR- β led to significantly lower survival rates while knock-down of PILR- β or activation of PILR- α resulted in significantly less inflammation and increased pathogen clearance (4,5).

References:

1. Wilson, M.D. *et al.* (2006) *Physiol. Genomics* **27**:201.
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3. Lu, Q. *et al.* (2014) *PNAS* **111**:8221.
4. Tato, C.M. *et al.* (2012) *PLoS One* **7**:e31690.
5. Banerjee, A. *et al.* (2010) *Infect. Immun.* **78**:1353.
6. Tabata, S. *et al.* (2008) *J. Biol. Chem.* **283**:8893.
7. Mousseau, DD. *et al.* (2000) *J. Biol. Chem.* **275**:4467.