

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived mouse PTK7/CCK4 protein		
	Mouse PTK7/CCK4 (Ala23-Thr696) Accession # Q8BKG3	IEGRMDP	Mouse IgG _{2A} (Glu98-Lys330)
	N-terminus		C-terminus
N-terminal Sequence Analysis	Ala23		
Structure / Form	Disulfide-linked homodimer		
Predicted Molecular Mass	102 kDa		

SPECIFICATIONS

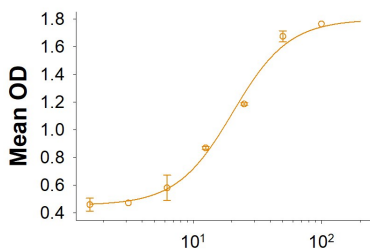
SDS-PAGE	110-130 kDa, under reducing conditions
Activity	Measured by its binding ability in a functional ELISA. Recombinant Mouse PTK7/CCK4 Fc Chimera (Catalog # 10333-TK) binds Biotinylated Recombinant Mouse Wnt-3a (Catalog # BT1324). The ED ₅₀ for this effect is 8-48 µg/mL.
Endotoxin Level	<0.10 EU 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. <ul style="list-style-type: none"> • 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

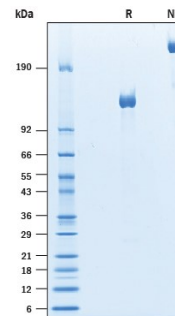
Binding Activity



Recombinant Mouse PTK7/CCK4 (µg/mL)

Recombinant Mouse PTK7/CCK4 Fc Chimera (Catalog # 10333-TK) binds Biotinylated Recombinant Mouse Wnt-3a (Catalog # BT1324). The ED₅₀ for this effect is 8-48 µg/mL.

SDS-PAGE



2 µg/lane of Recombinant Mouse PTK7/CCK4 Fc Chimera (Catalog # 10333-TK) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 110-130 kDa and 220-260 kDa, respectively.

BACKGROUND

Protein tyrosine kinase 7 (PTK7), also known as colon carcinoma kinase 4 (CCK4), is a member of the receptor tyrosine kinase superfamily (1, 2). PTK7 is a type I transmembrane receptor with a large extracellular domain (ECD) containing 7 Ig-like C2 type loops, a transmembrane domain, and a cytoplasmic tyrosine kinase homology domain. However, due to the lack of the DFG triplet motif required for catalytic activity in the kinase domain, PTK7 is considered a pseudokinase (1, 3). The mature ECD of mouse PTK7 shares 92% amino acid sequence identity with human PTK7. PTK7 is expressed in a wide array of tissue types ranging from lung and liver to kidney and placenta and has been linked to a broad range of functions (5). While originally identified as being over-expressed in colon carcinomas, PTK7 has been shown to play a role in embryogenesis, epithelial tissue organization, angiogenesis, cell motility, and survival (1, 6, 7). PTK7 has been shown to be an important regulator of the Wnt signaling pathways, both canonical and non-canonical, and is linked to the regulation of the planar cell polarity pathway (3, 6). Proteolysis of full-length PTK7 by MMPs (matrix metalloproteinases), ADAMs (a disintegrin domain and metalloproteinases), and gamma-secretases results in a soluble N-terminal fragment and several C-terminal, membrane-associated or intracellular proteolytic fragments (3, 6). The soluble form is a co-receptor for the Semaphorin/Plexin and VEGF signaling pathways (8). Deregulation of PTK7 signaling has now been observed in numerous cancers including colon, gastric, lung, and acute myeloid leukemia (1, 3). Recent studies have shown that PTK7 expression promoted increased migration and resistance to apoptosis in leukemic cells and acute myeloid leukemia (AML) blasts, while knock-down of PTK7 induced apoptosis in colorectal carcinoma cells (2, 7). Further, other studies have suggested that the ratio of full-length vs. cleaved protein, not just expression, contributes to PTK7's metastatic effects in cancer (6).

References:

1. Shin, W. *et al.* (2008) *Biochem. Biophys. Res. Commun.* **371**:793.
2. Meng, L. *et al.* (2010) *PLoS ONE* **5**:e14018.
3. Golubkov, V. *et al.* (2010) *J. Biol. Chem.* **285**:35740.
4. Berger, H. *et al.* (2017) *Front. Cell Dev. Biol.* **5**:31.
5. Park, S. *et al.* (1996) *J. Biochem.* **119**:235.
6. Golubkov, V. *et al.* (2014) *J. Biol. Chem.* **289**:24238.
7. Prebet, T. *et al.* (2010) *Blood*. **116**:2315.
8. Peradziryi, H. *et al.* (2012) *Arch. Biochem. Biophys.* **524**:71.