

DESCRIPTION

Source	Chinese Hamster Ovary cell line, CHO-derived human Klotho protein		
	Human Klotho (Glu34-His549) Accession # Q9UEF7	Human Fractalkine Mucin-like Stalk (Phe103-Thr338) Accession # P78423	6-His tag
	N-terminus		C-terminus
N-terminal Sequence Analysis	Glu34		
Predicted Molecular Mass	84 kDa		

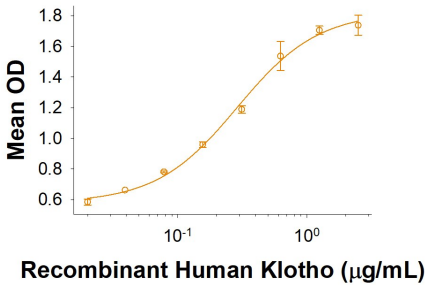
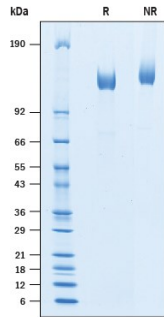
SPECIFICATIONS

SDS-PAGE	110-140 kDa, under reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When Recombinant Human FGF-23 (Catalog # 2604-FG) is immobilized at 5 µg/mL (100 µL/well), Recombinant Human Klotho Mucin Stalk Chimera His-tag (Catalog # 10308-KL) binds with an ED ₅₀ of 0.2-1.8 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	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <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

<p>Binding Activity</p>  <p>When Recombinant Human FGF-23 (Catalog # 2604-FG) is immobilized at 5 µg/mL, 100 µL/well, Recombinant Human Klotho Mucin Stalk Chimera His-tag (Catalog # 10308-KL) binds with an ED₅₀ of 0.2-1.8 µg/mL.</p>	<p>SDS-PAGE</p>  <p>2 µg/lane of Recombinant Human Klotho Mucin Stalk Chimera His-tag (Catalog # 10308-KL) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 110-140 kDa.</p>
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BACKGROUND

Klotho, also called alpha-Klotho (α -Klotho), is the founding member that along with β -Klotho and γ -Klotho, form the Klotho family within the glycosidase-1 superfamily (1, 2). α -Klotho is a type I transmembrane protein consisting of a large extracellular domain (ECD) containing glycosidase-like domains (KL1 and KL2), a single transmembrane domain and a short intracellular domain. Alternative mRNA splicing of the ECD of α -Klotho results in a circulating protein known as soluble α -Klotho (s-Klotho), which has been detected in both humans and mice (3, 4). In addition to the s-Klotho form, a 130 kDa form found in plasma and cerebrospinal fluid and a prominent intracellular 120 kDa form of α -Klotho have also been identified (3, 4). The mature ECD of full length human α -Klotho shares 87% and 90% identity with mouse and rat α -Klotho, respectively. Due to highly conserved sequences between α -Klotho forms, it is difficult to differentiate s-Klotho from the other short forms in vivo (5). Although α -Klotho was identified ~20 years ago, its function remains incompletely understood. α -Klotho shows weak glucuronidase activity which activates the renal ion channel TRPV5 to reabsorb urinary calcium (10). α -Klotho acts as a cofactor for interaction of FGF-23 with FGF R1 (6). This interaction negatively regulates 1 α -hydroxylase, the rate-limiting enzyme in the synthesis of 1,25(OH)₂D₃ (vitamin D) (7). s-Klotho functions as a hormonal factor and is involved in anti-aging, anti-oxidation, modulation of ion-transport, and Wnt signaling (8). Both α -Klotho and β -Klotho are cofactors for FGF19 binding (9). The phenotype of α -Klotho-deficient mice resembles premature aging, including arteriosclerosis, osteoporosis, skin atrophy, infertility, emphysema and premature death (2). α -Klotho deficient mice show severe hyperphosphatemia and ectopic calcification of soft tissues due to excess vitamin D (2-7). Conversely, excess α -Klotho extends lifespan (6).

References:

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