

## Recombinant Human KL Protein (His Tag)

**Catalog Number:** PDEH100852

**Note:** Centrifuge before opening to ensure complete recovery of vial contents.

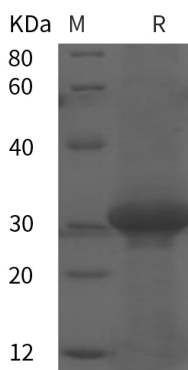
### Description

<b>Species</b>	Human
<b>Source</b>	E.coli-derived Human KL protein Phe765-Ser981, with an N-terminal His
<b>Calculated MW</b>	23.8 kDa
<b>Observed MW</b>	31 kDa
<b>Accession</b>	Q9UEF7
<b>Bio-activity</b>	Not validated for activity

### Properties

<b>Purity</b>	> 95% as determined by reducing SDS-PAGE.
<b>Endotoxin</b>	< 10 EU/mg of the protein as determined by the LAL method
<b>Storage</b>	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
<b>Shipping</b>	This product is provided as lyophilized powder which is shipped with ice packs.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with 5% Trehalose and 5% Mannitol.
<b>Reconstitution</b>	It is recommended that sterile water be added to the vial to prepare a stock solution of 0.5 mg/mL. Concentration is measured by UV-Vis.

### Data



SDS-PAGE analysis of Human KL proteins, 2 µg/lane of Recombinant Human KL proteins was resolved with SDS-PAGE under reducing conditions, showing bands at 31 kDa.

### Background

#### For Research Use Only

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Klotho, also called Klotho-alpha, is the founding member of the Klotho family within the glycosidase-1 superfamily. Klotho is expressed in areas concerned with calcium regulation, predominantly in the kidney distal convoluted tubules, but also in the brain choroid plexus (which produces cerebrospinal fluid) and the parathyroid. A 130 kDa form found in plasma and cerebrospinal fluid is produced by proteolytic cleavage of the glycosylated 135 kDa full-length Klotho. A truncated 549 aa isoform predicted by alternative mRNA splicing has been detected in human and mice. A prominent intracellular 120 kDa form of Klotho is localized to endoplasmic reticulum and Golgi membranes. The phenotype of Klotho-deficient mice resembles premature aging, including arteriosclerosis, osteoporosis, skin atrophy, infertility, emphysema and premature death. Conversely, excess Klotho extends lifespan. Klotho acts as a cofactor for interaction of FGF-23 with FGF R1. This interaction negatively regulates 1 alpha-hydroxylase, the rate-limiting enzyme in the synthesis of 1,25(OH)<sub>2</sub>D<sub>3</sub> (vitamin D). Klotho deficient mice show severe hyperphosphatemia and ectopic calcification of soft tissues due to excess vitamin D. Both Klotho and Klotho beta are cofactors for FGF19 binding. Klotho also shows glucuronidase activity which activates the renal ion channel TRPV5 to reabsorb urinary calcium. Klotho has been reported to downregulate insulin or IGF-1 signaling in adipocytes, to bind and antagonize Wnt molecules, and to facilitate release of parathyroid hormone.

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