**General Information**

**Gene Name Synonym:**
GALA

**Protein Construction:**
A DNA sequence encoding the human GLA (NP_000160.1) (Met 1-Leu 429) was fused with a polyhistidine tag at the C-terminus.

**Source:**  Human

**Expression Host:**  HEK293 Cells

**QC Testing**

**Purity:**  > 97 % as determined by SDS-PAGE

**Endotoxin:**
< 1.0 EU per μg of the protein as determined by the LAL method

**Stability:**
Samples are stable for up to twelve months from date of receipt at -70 °C

**Predicted N terminal:**  Leu 32

**Molecular Mass:**
The secreted recombinant human GLA consists of 409 amino acids and has a predicted molecular mass of 46.8 kDa. In SDS-PAGE under reducing conditions, it migrates as an approximately 50 kDa band.

**Formulation:**
Lyophilized from sterile 50mM Tris, 150mM NaCl, pH 7.5
Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

**Usage Guide**

**Storage:**
Store it under sterile conditions at -20°C to -80°C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

**Reconstitution:**
Detailed reconstitution instructions are sent along with the products.

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**SDS-PAGE:**

![SDS-PAGE Image]

**Alpha-galactosidase A**

**Protein Description**

Alpha-galactosidase A, also known as Alpha-D-galactoside galactohydrolase, Alpha-D-galactosidase A, Melibiase and GLA, is a member of the glycosyl hydrolase 27 family. GLA is used as a long-term enzyme replacement therapy in patients with a confirmed diagnosis of Fabry disease. Defects in GLA are the cause of Fabry disease (FD) which is a rare X-linked sphingolipidosis disease where glycolipid accumulates in many tissues. The disease consists of an inborn error of glycosphingolipid catabolism. FD patients show systemic accumulation of globotriaosylceramide (Gb3) and related glycosphingolipids in the plasma and cellular lysosomes throughout the body. Clinical recognition in males results from characteristic skin lesions (angiookeratomas) over the lower trunk. Patients may show ocular deposits, febrile episodes, and burning pain in the extremities. Death results from renal failure, cardiac or cerebral complications of hypertension or other vascular disease. Deficiency of GLA leads to the accumulation of glycosphingolipids in the vasculature leading to multiorgan pathology. In addition to well-described microvascular disease, deficiency of GLA is also characterized by premature macrovascular events such as stroke and possibly myocardial infarction.

**References**