

A Reliable Research Partner in Life Science and Medicine

Recombinant Human HO1 Protein(Trx Tag)

Catalog Number: PDEH100474

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

Description

Species Human

Source E.coli-derived Human HO1 protein Met1-Thr261, with an N-terminal Trx

Calculated MW 48.7 kDa Observed MW 48 kDa Accession P09601

Bio-activity Not validated for activity

Properties

> 95% as determined by reducing SDS-PAGE. **Purity**

Endotoxin < 10 EU/mg of the protein as determined by the LAL method

Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 Storage

°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.

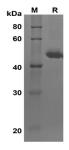
This product is provided as lyophilized powder which is shipped with ice packs. Shipping Lyophilized from a 0.2 µm filtered solution in PBS with 5% Trehalose and 5% Formulation

Mannitol.

Reconstitution 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 HO1 proteins, 2µg/lane of Recombinant Human HO1 proteins was resolved with SDS-PAGE under reducing conditions, showing bands at 48 kDa

Background

Elabscience®

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Heme Oxygenase 1 (HO-1) is an enzyme in endoplasmic reticulum that belongs to the heme oxygenase family. HO-1 cleaves the heme ring at the alpha methene bridge to form Biliverdin. Biliverdin is subsequently converted to Bilirubin by Biliverdin reductase. In physiological state, the highest activity of HO-1 is found in the spleen, where senescent erythrocytes are sequestrated and destroyed. HO-1 activity is highly inducible by its substrate heme and by various no n-heme substances such as heavy metals, bromobenzene, endotoxin, oxidizing agents and UVA. HO-1 is involved in the regulation of cardiovascular function and response to a variety of stressors. Defects in HO-1 are the cause of Heme Oxygenase 1 deficiency, resulting in marked erythrocyte fragmentation and intravascular hemolysis, coagulation abnormalities, endothelial damage, and iron deposition in renal and hepatic tissues.

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