# Recombinant Human HO1 Protein(Trx Tag)

**Catalog Number: PDEH100474**

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

## Description

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|---|---|
| **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

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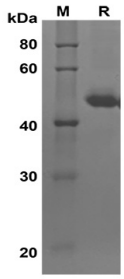
|---|---|
| **Purity** | > 95% as determined by reducing SDS-PAGE. |
| **Endotoxin** | < 10 EU/mg of the protein as determined by the LAL method |
| **Storage** | Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 ℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months. |
| **Shipping** | This product is provided as lyophilized powder which is shipped with ice packs. |
| **Formulation** | Lyophilized from a 0.2 μm filtered solution in PBS with 5% Trehalose and 5% 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

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.