Recombinant Mouse Ceruloplasmin/CP protein (His tag)

Catalog Number:PDEM100040

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

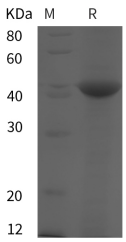
Description

Synonyms	CERU_MOUSE;EC:1.16.3.1;Ferroxidase
Species	Mouse
Expression Host	E.coli
Sequence	Tyr729-Gly1061
Accession	Q61147
Calculated Molecular Weight	36.5 kDa
Observed molecular weight	40 kDa
Tag	N-His

Properties

Purity	> 95 % as determined by reducing SDS-PAGE.
Endotoxin	Please contact us for more information.
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 sterile PBS, pH 7.4. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 95 % as determined by reducing SDS-PAGE.

Background

Ceruloplasmin is a blue, copper-binding (6-7 atoms per molecule) glycoprotein. It has ferroxidase activity oxidizing Fe2+ to Fe3+ without releasing radical oxygen species. It is involved in iron transport across the cell membrane. Provides Cu2+ ions for the ascorbate-mediated deaminase degradation of the heparan sulfate chains of GPC1. May also play a role in fetal lung development or pulmonary antioxidant defense. Defects in CP are the cause of aceruloplasminemia (ACERULOP) [MIM:604290]. It is an autosomal recessive disorder of iron metabolism characterized by iron accumulation in the brain as well as visceral organs. Clinical features consist of the triad of retinal degeneration, diabetes mellitus and neurological disturbances. Note=Ceruloplasmin levels are decreased in Wilson disease, in which copper cannot be incorporated into ceruloplasmin in liver because of defects in the copper-transporting ATPase 2.

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