



# COX1 Polyclonal Antibody

<b>Catalog No</b>	YP-Ab-05051
<b>Isotype</b>	IgG
<b>Reactivity</b>	Human;Mouse
<b>Applications</b>	WB;ELISA
<b>Gene Name</b>	MT-CO1 COI COXI MTCO1
<b>Protein Name</b>	Cytochrome c oxidase subunit 1 (EC 1.9.3.1) (Cytochrome c oxidase polypeptide I)
<b>Immunogen</b>	Synthesized peptide derived from human protein . at AA range: 380-460
<b>Specificity</b>	COX1 Polyclonal Antibody detects endogenous levels of protein.
<b>Formulation</b>	Liquid in PBS containing 50% glycerol, and 0.02% sodium azide.
<b>Source</b>	Polyclonal, Rabbit,IgG
<b>Purification</b>	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
<b>Dilution</b>	WB 1:500-2000 ELISA 1:5000-20000
<b>Concentration</b>	1 mg/ml
<b>Purity</b>	≥90%
<b>Storage Stability</b>	-20°C/1 year
<b>Synonyms</b>	
<b>Observed Band</b>	56kD
<b>Cell Pathway</b>	Mitochondrion inner membrane ; Multi-pass membrane protein .
<b>Tissue Specificity</b>	Blood,Bone fossil,Bones,Breast cancer,Distant normal tissue,Glioma,Para-can
<b>Function</b>	catalytic activity:4 ferrocytochrome c + O(2) + 4 H(+) = 4 ferricytochrome c + 2 H(2)O.,disease:Defects in MT-CO1 are a cause of anemia sideroblastic acquired idiopathic (AISA) [MIM:516030]; a disease characterized by inadequate formation of heme and excessive accumulation of iron in mitochondria.,disease:Defects in MT-CO1 are a cause of cytochrome c oxidase deficiency (COX deficiency) [MIM:220110]; also called mitochondrial complex IV deficiency. COX deficiency is a clinically heterogeneous disorder. The clinical features are ranging from isolated myopathy to severe multisystem disease, with onset from infancy to adulthood.,disease:Defects in MT-CO1 are a cause of Leber hereditary optic neuropathy (LHON) [MIM:535000]. LHON is a maternally inherited disease resulting in acute or subacute loss of central vision, due to optic nerve dysfunction. Cardiac conduction defects and neurological d
<b>Background</b>	catalytic activity:4 ferrocytochrome c + O(2) + 4 H(+) = 4 ferricytochrome c + 2 H(2)O.,disease:Defects in MT-CO1 are a cause of anemia sideroblastic acquired idiopathic (AISA) [MIM:516030]; a disease characterized by inadequate formation



of heme and excessive accumulation of iron in mitochondria.,disease:Defects in MT-CO1 are a cause of cytochrome c oxidase deficiency (COX deficiency) [MIM:220110]; also called mitochondrial complex IV deficiency. COX deficiency is a clinically heterogeneous disorder. The clinical features are ranging from isolated myopathy to severe multisystem disease, with onset from infancy to adulthood.,disease:Defects in MT-CO1 are a cause of Leber hereditary optic neuropathy (LHON) [MIM:535000]. LHON is a maternally inherited disease resulting in acute or subacute loss of central vision, due to optic nerve dysfunction. Cardiac conduction defects and neurological defects have also been described in some patients. LHON results from primary mitochondrial DNA mutations affecting the respiratory chain complexes.,disease:Defects in MT-CO1 are associated with recurrent myoglobinuria [MIM:550500]. Myoglobinuria consists of excretion of myoglobin in the urine.,function:Cytochrome c oxidase is the component of the respiratory chain that catalyzes the reduction of oxygen to water. Subunits 1-3 form the functional core of the enzyme complex. CO I is the catalytic subunit of the enzyme. Electrons originating in cytochrome c are transferred via the copper A center of subunit 2 and heme A of subunit 1 to the bimetallic center formed by heme A3 and copper B.,pathway:Energy metabolism; oxidative phosphorylation.,similarity:Belongs to the heme-copper respiratory oxidase family.,

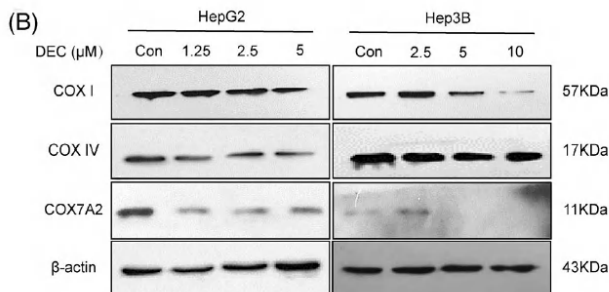
**matters needing attention**

Avoid repeated freezing and thawing!

**Usage suggestions**

This product can be used in immunological reaction related experiments. For more information, please consult technical personnel.

## Products Images



The nature compound dehydrocrenatinine exerts potent antihepatocellular carcinoma by destroying mitochondrial complexes in vitro and in vivo 2022 Feb 02. WB Human