Product name:	COX15 Rabbit Polyclonal Antibody
Cat number:	ABN09267
Conjugate:	Unconjugated
Size:	100µL
Clone:	Polyclonal
Concentration:	1mg/ml
Host:	Rabbit
Isotype:	IgG
Immunogen:	The antiserum was produced against synthesized peptide derived from human COX15. AA range:181-230
Reactivity:	Human,Mouse,Rat
Applications:	IHC 1:100-1:300,ICC/IF 1:50-1:200,ELISA 1:5000-1:10000
Purification:	Affinity purification
Form:	Liquid
Buffer:	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Storage:	Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles.

Background:

Cytochrome c oxidase (COX), the terminal component of the mitochondrial respiratory chain, catalyzes the electron transfer from reduced cytochrome c to oxygen. This component is a heteromeric complex consisting of 3 catalytic subunits encoded by mitochondrial genes and multiple structural subunits encoded by nuclear genes. The mitochondrially-encoded subunits function in electron transfer, and the nuclear-encoded subunits may function in the regulation and assembly of the complex. This nuclear gene encodes a protein which is not a structural subunit, but may be essential for the biogenesis of COX formation and may function in the hydroxylation of heme O, according to the yeast mutant studies. This protein is predicted to contain 5 transmembrane domains localized in the mitochondrial inner membrane. Alternative splicing of this gene generates two transcript variants divergingdisease:Defects in COX15 are a cause of cytochrome c oxidase deficiency (COX deficiency) [MIM:220110]. COX deficiency is a clinically heterogeneous disorder. The clinical features range from isolated myopathy to severe multisystem disease with onset from infancy to adulthood.,disease:Defects in COX15 are a cause of Leigh syndrome [MIM:256000]. Leigh syndrome is an early-onset progressive neurodegenerative disorder characterized by delayed onset of symptoms, hypotonia, feeding difficulties, failure to thrive, motor regression and brainstem signs. Diagnosis is confirmed by the presence of focal, bilateral lesions in one or more areas of the central nervous system including the brainstem, thalamus, basal ganglia, cerebellum and spinal cord.,function:May be involved in the biosynthesis of heme A.,pathway:Porphyrin metabolism; heme A biosynthesis; heme A from heme O: step 1/1.,similarity:Belongs to the COX15/ctaA family.,tissue specificity:Predominantly found in tissues characterized by high rates of oxidative phosphorylation (OxPhos), including muscle, heart, and brain.,