

Summary

Production Name	COX15 Rabbit Polyclonal Antibody
Description	Rabbit Polyclonal Antibody
Host	Rabbit
Application	IHC,ELISA
Reactivity	Human,Mouse,Rat

Performance

Conjugation	Unconjugated
Modification	Unmodified
lsotype	IgG
Clonality	Polyclonal
Form	Liquid
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.
Buffer	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Purification	Affinity purification

Immunogen

Gene Name	COX15	
Alternative Names	COX15; Cytochrome c oxidase assembly protein COX15 homolog	
Gene ID	1355.0	
SwissProt ID	Q7KZN9. The antiserum was produced against synthesized peptide derived from	
	human COX15. AA range:181-230	

Application

Dilution Ratio	IHC 1:100-1:300	ELISA: 1:5000

Molecular Weight

Background

Cytochrome c oxidase (COX), the terminal component of the mitochondrial respiratory chain, catalyzes the electron transfer

Product Name: COX15 Rabbit Polyclonal Antibody Catalog #: APRab09267

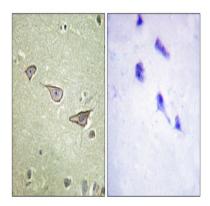


from reduced cytochrome c to oxygen. This component is a heteromeric complex consisting of 3 catalytic subunits encoded by mitochondrial genes and multiple structural subunits encoded by nuclear genes. The mitochondrially-encoded subunits function in electron transfer, and the nuclear-encoded subunits 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 diverging disease: 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.,

Research Area

Oxidative phosphorylation;Porphyrin and chlorophyll metabolism;

Image Data



Immunohistochemistry analysis of paraffin-embedded human brain tissue, using COX15 Antibody. The picture on the right is blocked with the synthesized peptide.

Note

For research use only.