

Summary

Production Name	ND5 Rabbit Polyclonal Antibody
Description	Rabbit Polyclonal Antibody
Host	Rabbit
Application	WB,ELISA
Reactivity	Human,Rat,Mouse

Performance

Conjugation	Unconjugated
Modification	Unmodified
lsotype	lgG
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	MT-ND5
Alternative Names	MT-ND5; MTND5; NADH5; ND5; NADH-ubiquinone oxidoreductase chain 5; NADH
	dehydrogenase subunit 5
Gene ID	4540.0
SwissProt ID	P03915.The antiserum was produced against synthesized peptide derived from human
	MT-ND5. AA range:328-377

Application

Dilution Ratio	WB 1:500 - 1:2000. ELISA: 1:10000. Not yet tested in other applications.
Molecular Weight	70kD

Product Name: ND5 Rabbit Polyclonal Antibody Catalog #: APRab14464



Background

catalytic activity:NADH + ubiquinone = NAD(+) + ubiquinol.,disease:Defects in MT-ND5 are a cause of complex I mitochondrial respiratory chain deficiency [MIM:252010]. Complex I (NADH-ubiquinone oxidoreductase), the largest complex of the mitochondrial respiratory chain, contains more than 40 subunits. It is embedded in the inner mitochondrial membrane and is partly protruding in the matrix. Complex I deficiency is the most common cause of mitochondrial disorders. It represents largely one-third of all cases of respiratory chain deficiency and is responsible for a variety of clinical symptoms, ranging from neurological disorders to cardiomyopathy, liver failure, and myopathy., disease: Defects in MT-ND5 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-ND5 are a cause of Leigh syndrome (LS) [MIM:256000]. LS is a severe neurological disorder characterized by bilaterally symmetrical necrotic lesions in subcortical brain regions., disease: Defects in MT-ND5 are a cause of mitochondrial encephalomyopathy with lactic acidosis and stroke-like episodes syndrome (MELAS) [MIM:540000]. MELAS is a genetically heterogenious disorder, characterized by episodic vomiting, seizures, and recurrent cerebral insults resembling strokes and causing hemiparesis, hemianopsia, or cortical blindness, disease:Defects in MT-ND5 are associated with features of myoclonic epilepsy associated with ragged-red fibers (MERRF) [MIM:545000]. MERRF is a mitochondrial encephalomyopathy characterized by myoclonic seizures. The prevalence in the general population of Europe has been estimated at 0.9 in 100'000 individuals, but the disease seems to be more common in the USA. Patients usually present during adolescence or early adulthood with myoclonic epilepsy, sometimes with neurosensory deafness, optic atrophy, short stature or peripheral neuropathy., function: Core subunit of the mitochondrial membrane respiratory chain NADH dehydrogenase (Complex I) that is believed to belong to the minimal assembly required for catalysis. Complex I functions in the transfer of electrons from NADH to the respiratory chain. The immediate electron acceptor for the enzyme is believed to be ubiquinone, similarity: Belongs to the complex I subunit 5 family, catalytic activity:NADH + ubiquinone = NAD(+) + ubiquinol., disease:Defects in MT-ND5 are a cause of complex I mitochondrial respiratory chain deficiency [MIM:252010]. Complex I (NADH-ubiquinone oxidoreductase), the largest complex of the mitochondrial respiratory chain, contains more than 40 subunits. It is embedded in the inner mitochondrial membrane and is partly protruding in the matrix. Complex I deficiency is the most common cause of mitochondrial disorders. It represents largely one-third of all cases of respiratory chain deficiency and is responsible for a variety of clinical symptoms, ranging from neurological disorders to cardiomyopathy, liver failure, and myopathy, disease: Defects in MT-ND5 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-ND5 are a cause of Leigh syndrome (LS) [MIM:256000]. LS is a severe neurological disorder characterized by bilaterally symmetrical necrotic lesions in subcortical brain regions, disease: Defects in MT-ND5 are a cause of mitochondrial encephalomyopathy with lactic acidosis and stroke-like episodes syndrome (MELAS) [MIM:540000]. MELAS is a genetically heterogenious disorder, characterized by episodic vomiting, seizures, and recurrent

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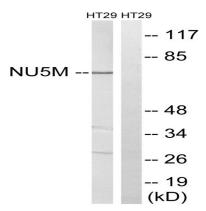


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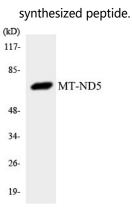
Research Area

Oxidative phosphorylation; Parkinson's disease;

Image Data



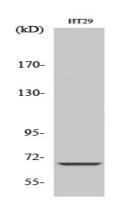
Western blot analysis of lysates from HT-29 cells, using MT-ND5 Antibody. The lane on the right is blocked with the



Western blot analysis of the lysates from Jurkat cells using MT-ND5 antibody.

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Western Blot analysis of various cells using ND5 Polyclonal Antibody diluted at 1: 1000

Note

For research use only.