



ND5 rabbit pAb

Cat No.:ES2905

For research use only

Overview

Product Name	ND5 rabbit pAb
Host species	Rabbit
Applications	WB;ELISA
Species Cross-Reactivity	Human;Rat;Mouse;
Recommended dilutions	Western Blot: 1/500 - 1/2000. ELISA: 1/10000. Not yet tested in other applications.
Immunogen	The antiserum was produced against synthesized peptide derived from human MT-ND5. AA range:328-377
Specificity	ND5 Polyclonal Antibody detects endogenous levels of ND5 protein.
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Storage	Store at -20°C. Avoid repeated freeze-thaw cycles.
Protein Name	NADH-ubiquinone oxidoreductase chain 5
Gene Name	MT-ND5
Cellular localization	Mitochondrion inner membrane ; Multi-pass membrane protein .
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Clonality	Polyclonal
Concentration	1 mg/ml
Observed band	70kD
Human Gene ID	4540
Human Swiss-Prot Number	P03915
Alternative Names	MT-ND5; MTND5; NADH5; ND5; NADH-ubiquinone oxidoreductase chain 5; NADH dehydrogenase subunit 5
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



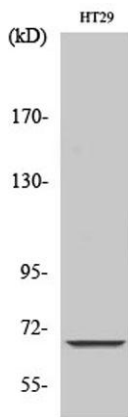


oxidoreductase), the largest complex of the mitochondrial respiratory chain, contains more than 40 subunits. It is embedded in the inner mitochondrial membrane and is partly protruding in the matrix. Complex I deficiency is the most common cause of mitochondrial disorders. It represents largely one-third 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. 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. 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. 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 heterogeneous disorder, characterized by episodic vomiting, seizures, and recurrent cerebral insults resembling strokes and causing hemiparesis, hemianopsia, or cortical blindness. 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

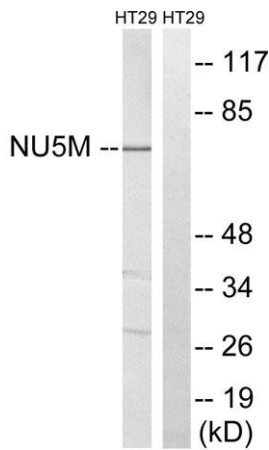




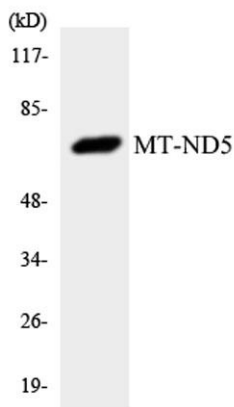
neuropathy.,function:Core subunit of the mitochondrial membrane respiratory chain NADH dehydrogenase (Complex I) that is believed to belong to the minimal assembly required for catalysis. Complex I functions in the transfer of electrons from NADH to the respiratory chain. The immediate electron acceptor for the enzyme is believed to be ubiquinone.,similarity:Belongs to the complex I subunit 5 family.,



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



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



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





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