

# Anti-POLG antibody ab154310

2 Images

### Overview

<b>Product name</b>	Anti-POLG antibody
<b>Description</b>	Rabbit polyclonal to POLG
<b>Host species</b>	Rabbit
<b>Tested applications</b>	<b>Suitable for:</b> WB, IHC-P
<b>Species reactivity</b>	<b>Reacts with:</b> Human
<b>Immunogen</b>	Recombinant fragment corresponding to Human POLG aa 180-393 (N terminal). Database link: <a href="#">P54098</a>
<b>Positive control</b>	NT2D1, IMR32 and MCF7 whole cell lysates; Human U87 xenograft tissue.
<b>General notes</b>	<p>The Life Science industry has been in the grips of a reproducibility crisis for a number of years. Abcam is leading the way in addressing this with our range of recombinant monoclonal antibodies and knockout edited cell lines for gold-standard validation. Please check that this product meets your needs before purchasing.</p> <p>If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be found below, along with publications, customer reviews and Q&amp;As</p>

### Properties

<b>Form</b>	Liquid
<b>Storage instructions</b>	Shipped at 4°C. Upon delivery aliquot. Store at -20°C or -80°C. Avoid freeze / thaw cycle.
<b>Storage buffer</b>	pH: 7.00 Preservative: 0.01% Thimerosal (merthiolate) Constituents: 78.99% PBS, 1% BSA, 20% Glycerol (glycerin, glycerine)
<b>Purity</b>	Immunogen affinity purified
<b>Clonality</b>	Polyclonal
<b>Isotype</b>	IgG

### Applications

**The Abpromise guarantee** Our [Abpromise guarantee](#) covers the use of ab154310 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
WB		1/500 - 1/3000. Predicted molecular weight: 140 kDa.
IHC-P		1/100 - 1/1000. Perform heat mediated antigen retrieval with citrate buffer pH 6 before commencing with IHC staining protocol. 10mM Citrate buffer

## Target

### Function

Involved in the replication of mitochondrial DNA.

### Involvement in disease

Defects in POLG are the cause of progressive external ophthalmoplegia with mitochondrial DNA deletions autosomal dominant type 1 (PEOA1) [MIM:157640]. Progressive external ophthalmoplegia is characterized by progressive weakness of ocular muscles and levator muscle of the upper eyelid. In a minority of cases, it is associated with skeletal myopathy, which predominantly involves axial or proximal muscles and which causes abnormal fatigability and even permanent muscle weakness. Ragged-red fibers and atrophy are found on muscle biopsy. A large proportion of chronic ophthalmoplegias are associated with other symptoms, leading to a multisystemic pattern of this disease. Additional symptoms are variable, and may include cataracts, hearing loss, sensory axonal neuropathy, ataxia, depression, hypogonadism, and parkinsonism.

Defects in POLG are a cause of progressive external ophthalmoplegia with mitochondrial DNA deletions autosomal recessive (PEOB) [MIM:258450]. PEOB is a severe form of progressive external ophthalmoplegia. It is clinically more heterogeneous than the autosomal dominant forms. Can be more severe.

Defects in POLG are a cause of sensory ataxic neuropathy dysarthria and ophthalmoparesis (SANDO) [MIM:607459]. SANDO is a clinically heterogeneous systemic disorder with variable features resulting from mitochondrial dysfunction. It shares phenotypic characteristics with autosomal recessive progressive external ophthalmoplegia and mitochondrial neurogastrointestinal encephalopathy syndrome. The clinical triad of symptoms consists of sensory ataxic, neuropathy, dysarthria, and ophthalmoparesis.

Defects in POLG are a cause of Alpers-Huttenlocher syndrome (AHS) [MIM:203700]; also called Alpers diffuse degeneration of cerebral gray matter with hepatic cirrhosis. AHS is an autosomal recessive hepatocerebral syndrome. The typical course of AHS includes severe developmental delay, intractable seizures, liver failure, and death in childhood. Refractory seizures, cortical blindness, progressive liver dysfunction, and acute liver failure after exposure to valproic acid are considered diagnostic features. The neuropathological hallmarks of AHS are neuronal loss, spongiform degeneration, and astrogliosis of the visual cortex. Liver biopsy results show steatosis, often progressing to cirrhosis.

Defects in POLG are a cause of mitochondrial neurogastrointestinal encephalopathy syndrome (MNGIE) [MIM:603041]; also known as myoneurogastrointestinal encephalomyopathy. MNGIE is an autosomal recessive disease associated with multiple deletions of skeletal muscle mitochondrial DNA (MtDNA). It is clinically characterized by onset between the second and fifth decades of life, ptosis, progressive external ophthalmoplegia, gastrointestinal dysmotility (often pseudoobstruction), diffuse leukoencephalopathy, thin body habitus, peripheral neuropathy, and myopathy.

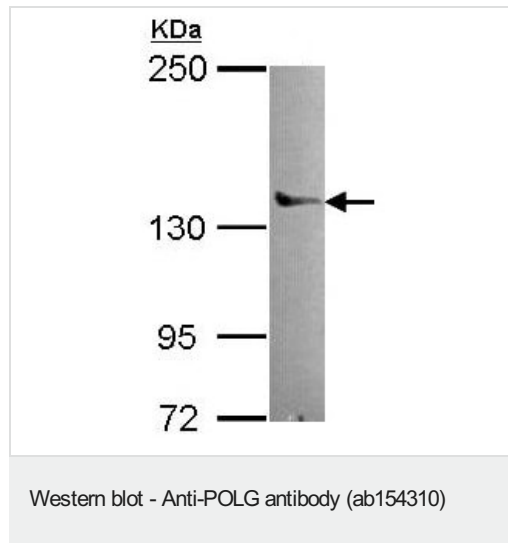
Defects in POLG are a cause of Leigh syndrome (LS) [MIM:256000]. LS is a severe neurological disorder characterized by bilaterally symmetrical necrotic lesions in subcortical brain regions.

**Sequence similarities**

Belongs to the DNA polymerase type-A family.

**Cellular localization**

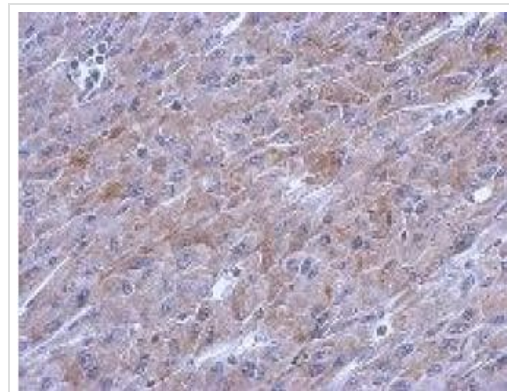
Mitochondrion.

**Images**

Anti-POLG antibody (ab154310) at 1/1000 dilution + NT2D1 whole cell lysate at 30 µg

**Predicted band size:** 140 kDa

5% SDS PAGE



Immunohistochemical analysis of paraffin-embedded Human U87 xenograft tissue labeling POLG with ab154310 at 1/500 dilution.

Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Anti-POLG antibody (ab154310)

**Please note:** All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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