abcam

Product datasheet

Anti-POLG antibody [EPR7296] ab128899



重组 RabMAb

23 References 5 图像

概述

产品名称 Anti-POLG抗体[EPR7296]

描述 兔单克隆抗体[EPR7296] to POLG

宿主 Rabbit

适用于: WB 经测试应用

种属反应性 与反应: Mouse, Rat, Human

免疫原 Synthetic peptide within Human POLG aa 1150-1200. The exact sequence is proprietary.

阳性对照 MCF7, HepG2, 293T, RAW 264.7 and C6 cell lysates.

常规说明 This product is a recombinant monoclonal antibody, which offers several advantages including:

- High batch-to-batch consistency and reproducibility

- Improved sensitivity and specificity

- Long-term security of supply

- Animal-free production

For more information see here.

Our RabMAb® technology is a patented hybridoma-based technology for making rabbit monoclonal antibodies. For details on our patents, please refer to **RabMAb**® **patents**.

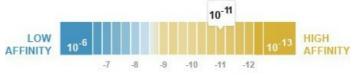
性能

形式 Liquid

存放说明 Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C.

Avoid freeze / thaw cycle. Stable for 12 months at -20°C.

 $K_D = 1.50 \times 10^{-11} M$ 解离常数(KD)



Learn more about K_D

存储溶液 pH: 7.20

Preservative: 0.01% Sodium azide

Constituents: PBS, 40% Glycerol, 0.05% BSA

纯**度** Protein A purified

克隆 单克隆

克隆编号 EPR7296

同种型 IgG

应用

The Abpromise guarantee Abpromise™承诺保证使用ab128899于以下的经测试应用

"应用说明"部分 下显示的仅为推荐的起始稀释度;实际最佳的稀释度/浓度应由使用者检定。

应用	Ab评论	说明
WB		1/1000 - 1/10000. Detects a band of approximately 140 kDa (predicted molecular weight: 140 kDa).

靶标

功能

疾病相关

Involved in the replication of mitochondrial DNA.

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 astrocytosis 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.

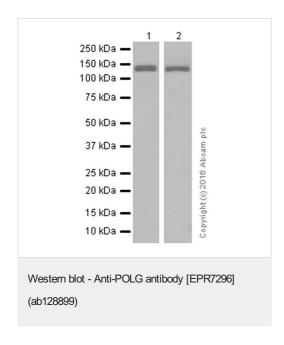
Belongs to the DNA polymerase type-A family.

Mitochondrion.

图片

序列相似性

细胞定位



All lanes : Anti-POLG antibody [EPR7296] (ab128899) at 1/5000 dilution (purified)

Lane 1 : HEK-293T (human embryonic kidney) whole cell lysate
Lane 2 : HepG2 (Human liver hepatocellular carcinoma cell line)
whole cell lysate

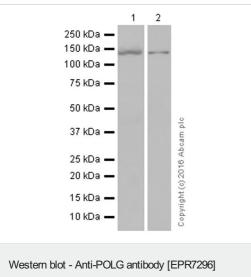
Lysates/proteins at 20 µg per lane.

Secondary

All lanes : Goat Anti-Rabbit IgG H&L (HRP) (<u>ab97051</u>) at 1/20000 dilution

Predicted band size: 140 kDa Observed band size: 140 kDa

Blocking and diluting buffer: 5% NFDM/TBST.



(ab128899)

All lanes: Anti-POLG antibody [EPR7296] (ab128899) at 1/1000 dilution (purified)

Lane 1: RAW 264.7 (Mouse macrophage cell line transformed with Abelson murine leukemia virus) whole cell lysate

Lane 2: C6 (Rat glial tumor cell line) whole cell lysate

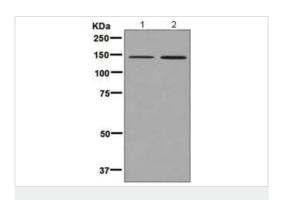
Lysates/proteins at 20 µg per lane.

Secondary

All lanes: Goat Anti-Rabbit IgG H&L (HRP) (ab97051) at 1/20000 dilution

Predicted band size: 140 kDa Observed band size: 140 kDa

Blocking and diluting buffer: 5% NFDM /TBST.



Western blot - Anti-POLG antibody [EPR7296] (ab128899)

All lanes: Anti-POLG antibody [EPR7296] (ab128899) at 1/1000 dilution (unpurified)

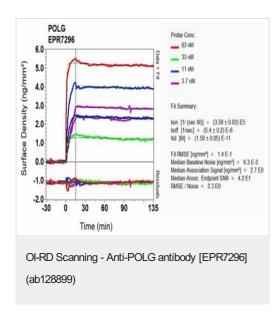
Lane 1: MCF7 cell lysate Lane 2: 293T cell lysate

Lysates/proteins at 10 µg per lane.

Secondary

All lanes: HRP labelled goat anti-rabbit at 1/2000 dilution

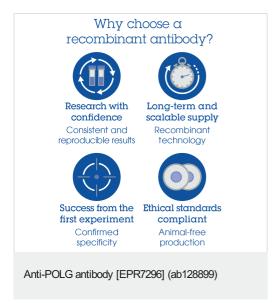
Predicted band size: 140 kDa



Equilibrium disassociation constant (K_D)

Learn more about K_D

Click here to learn more about K_D



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