

EndoG Antibody [7G1G10]
Catalog # ASC11984**Specification****EndoG Antibody [7G1G10] - Product Information**

Application	WB
Primary Accession	Q14249
Other Accession	Q14249 , 24638471
Reactivity	Human, Rat
Host	Mouse
Clonality	Monoclonal
Isotype	IgG1
Application Notes	EndoG monoclonal antibody can be used for detection of EndoG by Western blot at 5 - 10 µg/mL.

EndoG Antibody [7G1G10] - Additional Information

Gene ID	2021
Target/Specificity	ENDOG;

Reconstitution & Storage

EndoG monoclonal antibody can be stored at -20°C, stable for one year.

Precautions

EndoG Antibody [7G1G10] is for research use only and not for use in diagnostic or therapeutic procedures.

EndoG Antibody [7G1G10] - Protein Information

Name ENDOG

Function

Endonuclease that preferentially catalyzes the cleavage of double-stranded 5-hydroxymethylcytosine (5hmC)-modified DNA (PubMed: [25355512](http://www.uniprot.org/citations/25355512)). The 5hmC-modified nucleotide does not increase the binding affinity, but instead increases the efficiency of cutting and specifies the site of cleavage for the modified DNAs (By similarity). Shows significantly higher affinity for four-stranded Holliday junction over duplex and single-stranded DNAs (By similarity). Promotes conservative recombination when the DNA is 5hmC-modified (PubMed: [25355512](http://www.uniprot.org/citations/25355512)). Promotes autophagy through the suppression of mTOR by its phosphorylation-mediated interaction with YWHAG and its endonuclease activity-mediated DNA damage response (PubMed: [33473107](http://www.uniprot.org/citations/33473107)). GSK3-beta mediated phosphorylation of ENDOG enhances its interaction with YWHAG, leading to the release of TSC2 and PIK3C3 from YWHAG resulting in mTOR pathway suppression and autophagy initiation (PubMed: [33473107](http://www.uniprot.org/citations/33473107))

target="_blank">33473107). Promotes cleavage of mtDNA in response to oxidative and nitrosative stress, in turn inducing compensatory mtDNA replication (PubMed:29719607).

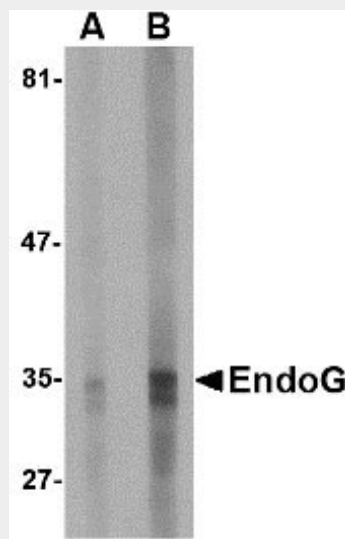
Cellular Location
Mitochondrion.

EndoG Antibody [7G1G10] - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

EndoG Antibody [7G1G10] - Images



Western blot analysis of EndoG expression in HepG2 cell lysate with EndoG antibody at (A) 5 and (B) 10 µg/mL.

EndoG Antibody [7G1G10] - Background

EndoG Monoclonal Antibody: The fragmentation of nuclear DNA is a hallmark of apoptotic cell death. The activities of caspase and nuclease are involved in the DNA fragmentation. Caspase-activated deoxyribonuclease (CAD), also termed DNA fragmentation factor (DFF40), is one such nuclease, and is capable of inducing DNA fragmentation and chromatin condensation after cleavage by caspase-3 of its inhibitor ICAD/DFF45. Caspase and CAD independent DNA fragmentation also exists. Recent studies demonstrated that another nuclease, endonuclease G (EndoG), is specifically activated by apoptotic stimuli and is able to induce nucleosomal fragmentation of DNA independently of caspase and DFF/CAD. EndoG is a mitochondrion-specific nuclease that translocates to the nucleus and cleaves chromatin DNA during apoptosis. The homologue of mammalian EndoG is the first mitochondrial protein identified to be involved in

apoptosis in *C. elegans*. EndoG also cleaves DNA in vitro.

EndoG Antibody [7G1G10] - References

Li LY, Luo X, Wang X. Endonuclease G is an apoptotic DNase when released from mitochondria. *Nature* 2001; 412:95-9.

Parrish J, Li L, Klotz K, et al. Mitochondrial endonuclease G is important for apoptosis in *C. elegans*. *Nature* 2001; 412:90-4

Hengartner MO. Apoptosis. DNA destroyers. *Nature* 2001; 412:27, 29.

Widlak P, Li LY, Wang X, et al. Action of recombinant human apoptotic endonuclease G on naked DNA and chromatin substrates: cooperation with exonuclease and DNase I. *J. Biol. Chem.* 2001; 276:48404-9.