10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

PMS2 Polyclonal Antibody

Catalog # AP71987

Product Information

Application	WB, IHC-P, IF, ICC, E
Primary Accession	P54278
Reactivity	Human, Rat, Mouse
Host	Rabbit
Clonality	Polyclonal
Calculated MW	95797

Additional Information

Gene ID	5395
Other Names	PMS2; PMSL2; Mismatch repair endonuclease PMS2; DNA mismatch repair protein PMS2; PMS1 protein homolog 2
Dilution	WB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. Immunofluorescence: 1/200 - 1/1000. ELISA: 1/10000. Not yet tested in other applications. IHC-P~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. Immunofluorescence: 1/200 - 1/1000. ELISA: 1/10000. Not yet tested in other applications. IF~~1:50~200 ICC~~N/A E~~N/A
Format	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.
Storage Conditions	-20°C

Protein Information

Name	PMS2 (HGNC:9122)
Function	Component of the post-replicative DNA mismatch repair system (MMR) (PubMed:30653781, PubMed:35189042). Heterodimerizes with MLH1 to form MutL alpha. DNA repair is initiated by MutS alpha (MSH2-MSH6) or MutS beta (MSH2-MSH3) binding to a dsDNA mismatch, then MutL alpha is recruited to the heteroduplex. Assembly of the MutL-MutS-heteroduplex ternary complex in presence of RFC and PCNA is sufficient to activate endonuclease activity of PMS2. It introduces single-strand breaks near the mismatch and thus generates new entry points for the exonuclease EXO1 to degrade the strand containing the mismatch. DNA methylation would prevent cleavage and therefore assure that only the newly mutated DNA strand is going to be corrected. MutL alpha (MLH1-PMS2) interacts physically with the clamp loader subunits of DNA polymerase III, suggesting that it may play a role to recruit the DNA polymerase III to the site of the MMR. Also implicated in DNA damage signaling, a process which induces cell cycle arrest and can lead to

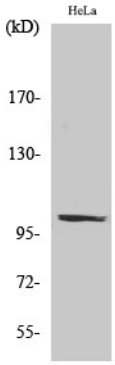
apoptosis in case of major DNA damages. Possesses an ATPase activity, but in the absence of gross structural changes, ATP hydrolysis may not be necessary for proficient mismatch repair (PubMed:35189042).

Cellular Location Nucleus

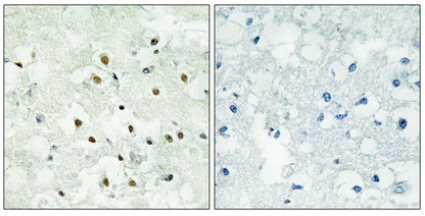
Background

Component of the post-replicative DNA mismatch repair system (MMR). Heterodimerizes with MLH1 to form MutL alpha. DNA repair is initiated by MutS alpha (MSH2-MSH6) or MutS beta (MSH2- MSH3) binding to a dsDNA mismatch, then MutL alpha is recruited to the heteroduplex. Assembly of the MutL-MutS-heteroduplex ternary complex in presence of RFC and PCNA is sufficient to activate endonuclease activity of PMS2. It introduces single-strand breaks near the mismatch and thus generates new entry points for the exonuclease EXO1 to degrade the strand containing the mismatch. DNA methylation would prevent cleavage and therefore assure that only the newly mutated DNA strand is going to be corrected. MutL alpha (MLH1-PMS2) interacts physically with the clamp loader subunits of DNA polymerase III, suggesting that it may play a role to recruit the DNA polymerase III to the site of the MMR. Also implicated in DNA damage signaling, a process which induces cell cycle arrest and can lead to apoptosis in case of major DNA damages.

Images



Western Blot analysis of various cells using PMS2 Polyclonal Antibody cells nucleus extracted by Minute TM Cytoplasmic and Nuclear Fractionation kit (SC-003,Inventbiotech,MN,USA).



Immunohistochemical analysis of paraffin-embedded Human brain. Antibody was diluted at 1:100(4°,overnight). High-pressure and temperature Tris-EDTA,pH8.0 was used for antigen retrieval. Negetive contrl (right) obtaned from antibody was pre-absorbed by immunogen peptide.