

PMS2 Antibody (aa623-639, clone 163C1251)
Mouse Monoclonal Antibody
Catalog # ALS12040**Specification**

PMS2 Antibody (aa623-639, clone 163C1251) - Product Information

Application	WB, IHC
Primary Accession	P54278
Reactivity	Human
Host	Mouse
Clonality	Monoclonal
Calculated MW	96kDa KDa

PMS2 Antibody (aa623-639, clone 163C1251) - Additional Information**Gene ID** 5395**Other Names**

Mismatch repair endonuclease PMS2, 3.1.-., DNA mismatch repair protein PMS2, PMS1 protein homolog 2, PMS2, PMSL2

Target/Specificity

A synthetic peptide corresponding to amino acids 623-639 SSLAKRIQLHHEAQQS of human PMS2; GenBank No. NP_000526.1.

Reconstitution & Storage

Short term 4°C, long term aliquot and store at -20°C, avoid freeze thaw cycles.

Precautions

PMS2 Antibody (aa623-639, clone 163C1251) is for research use only and not for use in diagnostic or therapeutic procedures.

PMS2 Antibody (aa623-639, clone 163C1251) - 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

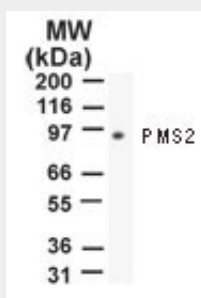
Nucleus

PMS2 Antibody (aa623-639, clone 163C1251) - Protocols

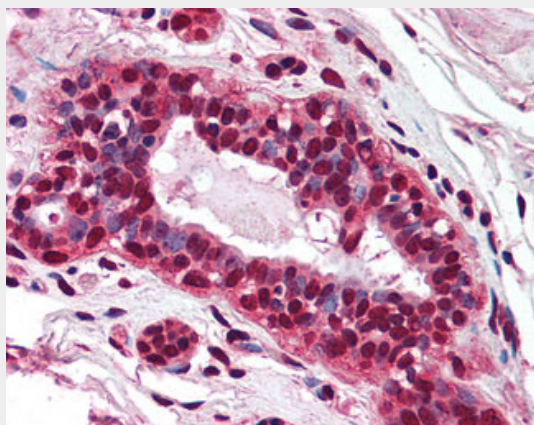
Provided below are standard protocols that you may find useful for product applications.

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

PMS2 Antibody (aa623-639, clone 163C1251) - Images



Western blot of PMS2 using antibody at 2 ug/ml against 10 ug of NIH 3T3 cell lysate.



Anti-PMS2 antibody IHC of human breast.

PMS2 Antibody (aa623-639, clone 163C1251) - 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-MSH6) 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.

PMS2 Antibody (aa623-639, clone 163C1251) - References

Nicolaides N.C.,et al.Nature 371:75-80(1994).
Tabata Y.,et al.Submitted (FEB-2003) to the EMBL/GenBank/DDBJ databases.
Bronner C.E.,et al.Submitted (OCT-1994) to the EMBL/GenBank/DDBJ databases.
Ota T.,et al.Nat. Genet. 36:40-45(2004).
Hillier L.W.,et al.Nature 424:157-164(2003).