

MMS21 Antibody
Catalog # ASC11132**Specification**

MMS21 Antibody - Product Information

Application	WB
Primary Accession	Q96MF7
Other Accession	NP_775956 , 27734761
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	Predicted: 27 kDa
Application Notes	Observed: 30 kDa KDa MMS21 antibody can be used for detection of MMS21 by Western blot at 0.5 - 1 µg/mL.

MMS21 Antibody - Additional Information

Gene ID	286053
Target/Specificity	
NSMCE2;	

Reconstitution & Storage

MMS21 antibody can be stored at 4°C for three months and -20°C, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.

Precautions

MMS21 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

MMS21 Antibody - Protein Information

Name NSMCE2

Synonyms C8orf36, MMS21

Function

E3 SUMO-protein ligase component of the SMC5-SMC6 complex, a complex involved in DNA double-strand break repair by homologous recombination (PubMed:[16055714](http://www.uniprot.org/citations/16055714), PubMed:[16810316](http://www.uniprot.org/citations/16810316)). Is not be required for the stability of the complex (PubMed:[16055714](http://www.uniprot.org/citations/16055714), PubMed:[16810316](http://www.uniprot.org/citations/16810316)). The complex may promote sister chromatid homologous recombination by recruiting the SMC1-SMC3 cohesin

complex to double-strand breaks (PubMed:16055714, PubMed:16810316). The complex is required for telomere maintenance via recombination in ALT (alternative lengthening of telomeres) cell lines and mediates sumoylation of shelterin complex (telosome) components which is proposed to lead to shelterin complex disassembly in ALT-associated PML bodies (APBs) (PubMed:17589526). Acts as an E3 ligase mediating SUMO attachment to various proteins such as SMC6L1 and TSNAX, the shelterin complex subunits TERF1, TERF2, TIN2 and TERF2IP, RAD51AP1, and maybe the cohesin components RAD21 and STAG2 (PubMed:16055714, PubMed:16810316, PubMed:17589526, PubMed:31400850). Required for recruitment of telomeres to PML nuclear bodies (PubMed:17589526). SUMO protein-ligase activity is required for the prevention of DNA damage-induced apoptosis by facilitating DNA repair, and for formation of APBs in ALT cell lines (PubMed:17589526). Required for sister chromatid cohesion during prometaphase and mitotic progression (PubMed:19502785).

Cellular Location

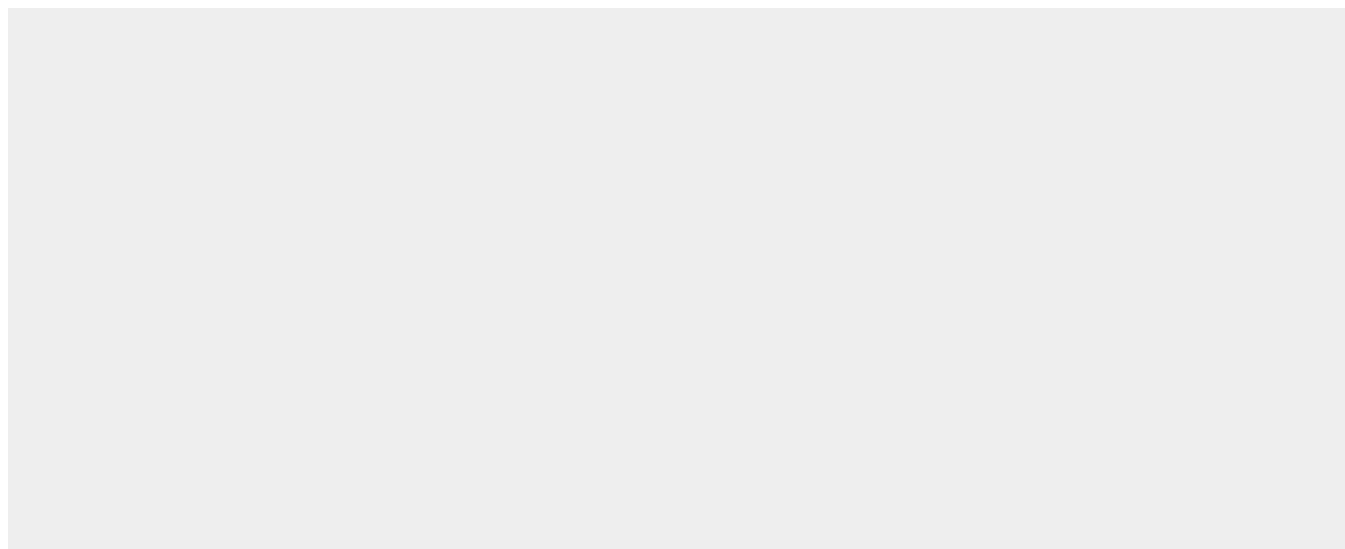
Nucleus. Chromosome, telomere. Nucleus, PML body. Note=Localizes to PML nuclear bodies in ALT cell lines.

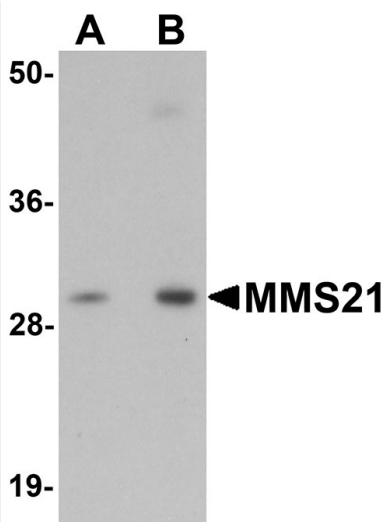
MMS21 Antibody - Protocols

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](#)

MMS21 Antibody - Images





Western blot analysis of MMS21 in 293 cell lysate with MMS21 antibody at (A) 0.5 and (B) 1 μ g/mL.

MMS21 Antibody - Background

MMS21 Antibody: MMS21, also known as NSE2, is a SUMO ligase that in combination with the SMC5/6 complex is required for the prevention of DNA damage induced apoptosis by facilitating DNA repair in human cells. MMS21-dependent sumoylation is integral and important to the cohesion mechanism and mitotic progression; this function appears to be independent of SMC6. MMS21 mediates SUMO attachment to various proteins such as SMC6L1 and TRAX, and possibly the cohesin components RAD21 and STAG2.

MMS21 Antibody - References

Potts PR and Yu H. Human MMS21/NSE2 is a SUMO ligase required for DNA repair. *Mol. Cell. Biol.* 2005; 25:7021-32.
Behlke-Steinert S, Touat-Todeschini L, Skoufias DA, et al. SMC5 and MMS21 are required for chromosome cohesion and mitotic progression. *Cell Cycle* 2009; 8:2211-8.
Bermudez-Lopez M, Ceschia A, de Piccoli G, et al. The Smc5/6 complex is required for dissolution of DNA-mediated sister chromatid linkages. *Nucleic Acids Res.* 2010; 38:6502-12.