abcam

ab214

Anti-Mre11 antibody [12D7] - BSA and Azide free

Key facts

Isotype	IgG1
Host species	Mouse
Storage buffer	pH: 7.4 Constituents: PBS
Form	Liquid
Clonality	Monoclonal
Immunogen	Synthetic Peptide within Human MRE11 aa 150 to C-terminus. The exact immunogen used to generate this antibody is proprietary information. Database link P49959
Carrier free	Yes
Clone number	12D7
Purification technique	Affinity purification Protein G
Light chain type	kappa
Concentration	Concentration data is not available right now, please try again later Batch concentration finder →

Reactivity data

IP

Expected

Species	Human
Dilution info	-

Notes	For normal lymphoblastoid cell lines.

Not recommended

Species	Mouse
Dilution info	-
Notes	For normal lymphoblastoid cell lines.

Species	Rat
Dilution info	-
Notes	For normal lymphoblastoid cell lines.

Flow Cyt

Tested

Species	Human
Dilution info	1-2 µg for 10^6 Cells
Notes	ab170190 - Mouse monoclonal IgG1, is suitable for use as an isotype control with this antibody.

Not recommended

Species	Mouse, Rat
Dilution info	-
Notes	-

WB

Tested

Species	Human
Dilution info	1/500 - 1/3000
Notes	(see Robinson et al).

Not recommended

Species	Rat
Dilution info	-
Notes	(see Robinson et al).

Species	Mouse
Dilution info	-
Notes	-

ICC/IF

Tested

Species	Human
Dilution info	1/100 - 1/1000
Notes	-

Not recommended

Species	Mouse, Rat
Dilution info	-
Notes	-

Target data

See full target information MRE11

Function

Core component of the MRN complex, which plays a central role in double-strand break (DSB) repair, DNA recombination, maintenance of telomere integrity and meiosis (PubMed:11741547, PubMed:14657032, PubMed:22078559, PubMed:23080121, PubMed:24316220, PubMed:26240375, PubMed:27889449, PubMed:28867292, PubMed:29670289, PubMed:30464262, PubMed:30612738, PubMed:31353207, PubMed:37696958, PubMed:38128537, PubMed:9590181, PubMed:9651580, PubMed:9705271). The MRN complex is involved in the repair of DNA double-strand breaks (DSBs) via homologous recombination (HR), an error-free mechanism which primarily occurs during S and G2 phases (PubMed:24316220, PubMed:28867292, PubMed:31353207, PubMed:38128537). The complex (1) mediates the end resection of damaged DNA, which generates proper single-stranded DNA, a key initial steps in HR, and is (2) required for the recruitment of other repair factors and efficient activation of ATM and ATR upon DNA damage (PubMed:24316220, PubMed:27889449, PubMed:28867292, PubMed:36050397, PubMed:38128537). Within the MRN complex, MRE11

possesses both single-strand endonuclease activity and double-strand-specific 3'-5' exonuclease activity (PubMed:11741547, PubMed:22078559, PubMed:24316220, PubMed:26240375, PubMed:27889449, PubMed:29670289, PubMed:31353207, PubMed:36563124, PubMed:9590181, PubMed:9651580, PubMed:9705271). After DSBs, MRE11 is loaded onto DSBs sites and cleaves DNA by cooperating with RBBP8/CtIP to initiate end resection (PubMed:27814491, PubMed:27889449, PubMed:30787182). MRE11 first endonucleolytically cleaves the 5' strand at DNA DSB ends to prevent non-homologous end joining (NHEJ) and licence HR (PubMed:24316220). It then generates a single-stranded DNA gap via 3' to 5' exonucleolytic degradation to create entry sites for EXO1- and DNA2-mediated 5' to 3' long-range resection, which is required for single-strand invasion and recombination (PubMed:24316220, PubMed:28867292). RBBP8/CtIP specifically promotes the endonuclease activity of MRE11 to clear protein-DNA adducts and generate clean double-strand break ends (PubMed:27814491, PubMed:27889449, PubMed:30787182). MRE11 endonuclease activity is also enhanced by AGER/RAGE (By similarity). The MRN complex is also required for DNA damage signaling via activation of the ATM and ATR kinases: the nuclease activity of MRE11 is not required to activate ATM and ATR (PubMed:14657032, PubMed:15064416, PubMed:15790808, PubMed:16622404). The MRN complex is also required for the processing of R-loops (PubMed:31537797). The MRN complex is involved in the activation of the cGAS-STING pathway induced by DNA damage during tumorigenesis: the MRN complex acts by displacing CGAS from nucleosome sequestration, thereby activating it (By similarity). In telomeres the MRN complex may modulate t-loop formation (PubMed:10888888). MRE11 contains two DNA-binding domains (DBDs), enabling it to bind both single-stranded DNA (ssDNA) and double-stranded DNA (dsDNA).

Storage

Shipped at conditions	Blue Ice
Appropriate short-term storage duration	1-2 weeks
Appropriate short-term storage conditions	+4°C
Appropriate long-term storage conditions	-20°C
Aliquoting information	Upon delivery aliquot
Storage information	Avoid freeze / thaw cycle

Notes

This product was changed from ascites to tissue culture supernatant on 10th April 2019. Please note that the dilutions may need to be adjusted accordingly. If you have any questions, please do not hesitate to contact our scientific support team.

Supplementary info

This supplementary information is collated from multiple sources and compiled automatically.

Activity summary	The Mre11 protein also known as MRE11A serves as an integral part of the DNA repair machinery in cells. It forms a component of the MRE11/RAD50/NBS1 (MRN) complex which is essential for the detection and repair of DNA double-strand breaks (DSBs). The molecular weight of the Mre11 protein is approximately 81 kDa. It is broadly expressed in various human tissues highlighting its extensive role in maintaining genomic stability.
Biological function summary	This protein acts in the repair of DSBs by initiating homologous recombination and non-homologous end joining pathways. Together with the RAD50 and NBS1 proteins Mre11 forms the MRN complex which processes DNA ends and signals to other repair mechanisms. Additionally Mre11's exonuclease and endonuclease activities are important for the resection of DNA at break sites facilitating subsequent repair synthesis.
Pathways	Mre11 is instrumental in the DNA damage response and maintenance of genomic integrity. It operates within the ATM (ataxia-telangiectasia mutated) signaling pathway which activates upon DNA damage and regulates cell cycle checkpoints. Mre11 interacts with the ATM protein modifying the cellular response to DNA damage. The complex also collaborates closely with BRCA1 an important regulator of the repair process and associated with preventing breast cancer development.
Associated diseases and disorders	Mutations or dysfunction in the MRE11A gene can be linked to several conditions including ataxia-telangiectasia-like disorder (ATLD) and Nijmegen breakage syndrome (NBS). These disorders result from impaired DNA repair mechanisms leading to increased sensitivity to radiation and predisposition to cancer. The NBS1 protein as part of the MRN complex works closely with Mre11 in these conditions. Both disorders highlight the critical role of Mre11 in safeguarding genomic stability and preventing disease.

Product promise

Tested

We have tested this species and application combination and it works. It is covered by our product promise.

Expected

We have not tested this specific species and application combination in-house, but expect it will work. It is covered by our product promise.

Predicted

This species and application combination has not been tested, but we predict it will work based on strong homology. However, this combination is not covered by our product promise.

Not recommended

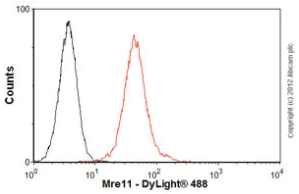
We do not recommend this combination. It is not covered by our product promise.

We are dedicated to supporting your work with high quality reagents and we are here for you every step of the way should you need us.

In the unlikely event of one of our products not working as expected, you are covered by our product promise.

Full details and terms and conditions can be found here:
Terms & Conditions.

4 product images

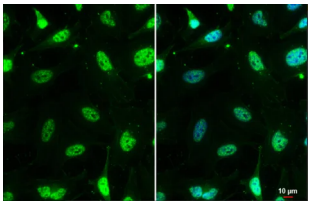


Flow Cytometry - Anti-Mre11 antibody [12D7] - BSA and Azide free (ab214)

Mre11 Flow Cytometry staining using mouse Anti-Mre11 antibody

Overlay histogram showing HeLa cells stained with ab214 (red line). The cells were fixed with 80% methanol (5 min) and then permeabilized with 0.1% PBS-Tween for 20 min. The cells were then incubated in 1x PBS / 10% normal goat serum / 0.3M glycine to block non-specific protein-protein interactions followed by the antibody (ab214, 1µg/1x10^6 cells) for 30 min at 22°C. The secondary antibody used was a goat anti-mouse DyLight® 488 (IgG, H+L) (ab96879) at 1/500 dilution for 30 min at 22°C. Isotype control antibody (black line) was mouse IgG1 [ICIGG1] (ab91353, 2µg/1x10^6 cells) used under the same conditions. Acquisition of >5,000 events was performed. This antibody gave a positive signal in HeLa cells fixed with 4% paraformaldehyde (10 min)/permeabilized with 0.1% PBS-Tween for 20 min used under the same conditions.

This image was generated using the ascites version of the product.



Immunocytochemistry/ Immunofluorescence - Anti-Mre11 antibody [12D7] - BSA and Azide free (ab214)

Mre11 Immunocytochemistry/ Immunofluorescence staining using mouse Anti-Mre11 antibody

Immunocytochemical analysis of, 4% paraformaldehyde-fixed at RT for 15 min, HeLa (Human epithelial cell line from cervix adenocarcinoma) cells labeling Mre-11 (green) with ab214 at 1/200 dilution. Blue: Hoechst 33342 staining. Scale bar= 10 µm.

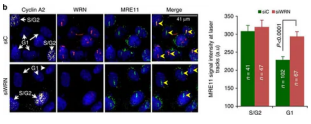


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Immunocytochemistry/ Immunofluorescence - Anti-Mre11 antibody [12D7] - BSA and Azide free (ab214)

Mre11 Immunocytochemistry/ Immunofluorescence staining using mouse Anti-Mre11 antibody

Immunocytochemistry-immunofluorescence using Anti-Mre11 antibody [12D7] - BSA and Azide free, ab214. Publication image from Bohr, V. A. et al., 2016, Nat Commun, 27922005. Legend direct from paper.

WRN inhibits the recruitment of MRE11 and CtIP to DSBs.(a) Confocal microphotographs showing cell cycle independent recruitment of WRN to laser-induced DSBs in U2OS cells. Graph represents data from >200 cells. gH2AX; gamma H2AX. (b) WRN depletion increases MRE11 recruitment to

DSBs in G1 cells. siRNA transfected U2OS cells were microirradiated and immunostained for cyclin A2, WRN and MRE11. Graph indicates MRE11 recruitment to laser-induced DSBs in control and WRN siRNA transfected S/G2 and G1 cells. n; number of cells. (c) WRN knockdown promotes CtIP recruitment to laser-induced DSBs. Control (siC) and WRN knockdown U2OS cells were microirradiated to generate DSBs and immunostained with WRN, CtIP and cyclin A2 antibodies. Cyclin A2 positive cells indicate cells in S/G2 phase. (d) Quantitation of CtIP and WRN signals at DSBs as seen in panel E. n=26 cells per set (e) Recruitment kinetics of mCherry-WRN and YFP-MRE11 to laser-induced DSBs in U2OS cells. n=14 cells (f) Real-time recruitment of YFP-MRE11 to laser-induced DSBs in WS cells, AG11395, expressing no WRN (vector control) and mCherry-WRN. n=28 cells (g) Recruitment of GFP-CtIP to laser-induced DSBs in AG11395 WS cells transfected with or without mCherry WRN. n=22 cells. Yellow arrow heads in panel A, B and C indicate laser-induced DSB tracks. a.u., arbitrary unit.

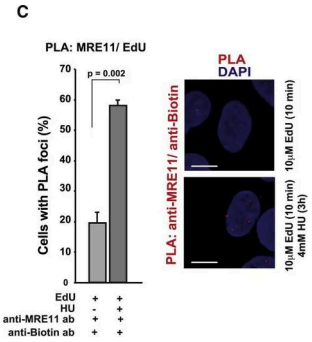


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Other - Anti-Mre11 antibody [12D7] - BSA and Azide free (ab214)

Proximity Ligation Assay using Anti-Mre11 antibody [12D7] - BSA and Azide free, ab214. Publication image from Nieminuszczy, J. et al., 2019, Mol Cell, 31255466. Legend direct from paper.

EXD2 Is Recruited to Stressed Replication Forks(A) Western blot of iPOND samples. Thymidine chase analysis illustrates that EXD2 specifically associates with the replisome. PCNA acts as a control.(B) Schematic of the proximity ligation assay (PLA) employed to detect colocalization of target proteins with nascent DNA.(C) Percentage of cells with MRE11/biotin PLA foci (mean ± SEM, n = 3 independent experiments, t test). Right: representative images of PLA foci (red), DAPI acts as a nuclear counterstain. Scale bar, 10 μm.(D) Percentage of cells with GFP/biotin PLA foci (mean ± SEM, n = 3 independent experiments, t test) in U2OS control cells and U2OS cells expressing GFP-EXD2. Right: representative images of PLA foci (red), DAPI acts as a nuclear counterstain. Scale bar, 10 μm.(E) Laser microirradiation induces rapid redistribution of GFP-EXD2 to damaged chromatin; representative images showing GFP-EXD2 accumulation at laser-generated DNA lesions. GFP-CtIP was used as a positive control. Scale bar, 10 μm.(F) Quantification of GFP-EXD2 (left panel) and GFP-CtIP (right panel) recruitment kinetics (intensity versus time) to laser-generated DNA lesions (mean ± SE, n ≥ 10 cells from 2 independent experiments).

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