

Phospho-p95/NBS1 (S343) Antibody

Rabbit mAb Catalog # AP90300

Product Information

Application Primary Accession Reactivity Clonality Other Names	WB, IF, ICC, IP <u>O60934</u> Human Monoclonal Cell cycle regulatory protein P95, NBN, NBS, NIBRIN, NIJMEGEN BREAKAGE syndrome protein 1, p95-NBS1
lsotype	Rabbit IgG
Host	Rabbit
Calculated MW	84959

Additional Information

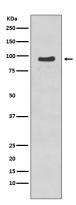
Dilution Purification Immunogen	WB 1:500~1:2000 ICC/IF 1:50~1:200 IP 1:50 Affinity-chromatography A synthesized peptide derived from human Phospho-p95/NBS1 (S343)
Description	NBS1 is a member of the MRE11/RAD50 double-strand break repair complex. Involved in DNA double-strand break repair and DNA damage-induced checkpoint activation. Mutation results in the Nijmegen breakage syndrome (NBS), an autosomal recessive chromosomal instability syndrome.
Storage Condition and Buffer	

Protein Information

Name	NBN (<u>HGNC:7652</u>)
Function	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: <u>10888888</u> , PubMed: <u>15616588</u> , PubMed: <u>18411307</u> , PubMed: <u>18583988</u> , PubMed: <u>18678890</u> , PubMed: <u>19759395</u> , PubMed: <u>23115235</u> , PubMed: <u>28216226</u> , PubMed: <u>28867292</u> , PubMed: <u>9705271</u>). 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: <u>19759395</u> , PubMed: <u>28867292</u> , PubMed: <u>9705271</u>). 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: <u>19759395</u> , PubMed: <u>9705271</u>). The MRN complex possesses single-strand endonuclease activity and double-strand-specific 3'-5'

	 exonuclease activity, which are provided by MRE11, to initiate end resection, which is required for single-strand invasion and recombination (PubMed:<u>19759395</u>, PubMed:<u>28867292</u>, PubMed:<u>9705271</u>). Within the MRN complex, NBN acts as a protein-protein adapter, which specifically recognizes and binds phosphorylated proteins, promoting their recruitment to DNA damage sites (PubMed:<u>12419185</u>, PubMed:<u>15616588</u>, PubMed:<u>18411307</u>, PubMed:<u>18582474</u>, PubMed:<u>18583988</u>, PubMed:<u>18678890</u>, PubMed:<u>19759395</u>, PubMed:<u>19804756</u>, PubMed:<u>23762398</u>, PubMed:<u>24534091</u>, PubMed:<u>27814491</u>, PubMed:<u>27889449</u>, PubMed:<u>33836577</u>). Recruits MRE11 and RAD50 components of the MRN complex to DSBs in response to DNA damage (PubMed:<u>12419185</u>, PubMed:<u>18411307</u>, PubMed:<u>18583988</u>, PubMed:<u>18678890</u>, PubMed:<u>24534091</u>, PubMed:<u>26438602</u>). Promotes the recruitment of PI3/PI4-kinase family members ATM, ATR, and probably DNA-PKcs to the DNA damage sites, activating their functions (PubMed:<u>15064416</u>, PubMed:<u>15616588</u>, PubMed:<u>15790808</u>, PubMed:<u>16622404</u>, PubMed:<u>22464731</u>, PubMed:<u>30952868</u>, PubMed:<u>35076389</u>). Mediates the recruitment of phosphorylated RBBP8/CtIP to DSBs, leading to cooperation
	between the MRN complex and RBBP8/CtIP to initiate end resection (PubMed: <u>19759395</u> , PubMed: <u>27814491</u> , PubMed: <u>27889449</u> , PubMed: <u>33836577</u>). RBBP8/CtIP specifically promotes the endonuclease activity of the MRN complex to clear DNA ends containing protein adducts (PubMed: <u>27814491</u> , PubMed: <u>27889449</u> , PubMed: <u>30787182</u> , PubMed: <u>33836577</u>). The MRN complex is also required for the processing of R-loops (PubMed: <u>31537797</u>). NBN also functions in telomere length maintenance via its interaction with TERF2: interaction with TERF2 during G1 phase preventing recruitment of DCLRE1B/Apollo to telomeres (PubMed: <u>10888888</u> , PubMed: <u>28216226</u>). NBN also promotes DNA repair choice at dysfunctional telomeres: NBN phosphorylation by CDK2 promotes non- homologous end joining repair at telomeres, while unphosphorylated NBN promotes microhomology-mediated end-joining (MMEJ) repair (PubMed: <u>28216226</u>). Enhances AKT1 phosphorylation possibly by association with the mTORC2 complex (PubMed: <u>23762398</u>).
Cellular Location	Nucleus. Chromosome. Nucleus, PML body. Chromosome, telomere Note=Localizes to discrete nuclear foci after treatment with genotoxic agents (PubMed:10783165, PubMed:26215093, PubMed:26438602). Localizes to DNA double-strand breaks (DSBs); recruited to DNA damage sites via association with phosphorylated proteins, such as phosphorylated H2AX, phosphorylated MDC1 and phosphorylated RAD17 (PubMed:12419185, PubMed:18411307, PubMed:18582474, PubMed:18583988, PubMed:18678890, PubMed:19338747, PubMed:23115235, PubMed:24534091, PubMed:26438602) Acetylation of 'Lys-5' of histone H2AX (H2AXK5ac) promotes NBN/NBS1 assembly at the sites of DNA damage (PubMed:26438602)
Tissue Location	Ubiquitous (PubMed:9590180). Expressed at high levels in testis (PubMed:9590180).
Images	

Western blot analysis of p95/NBS1 phosphorylation expression in Jurkat cell lysate treated with Etopside.



Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.