

Anti-Phospho-NBN (Ser343) Antibody (5K712)

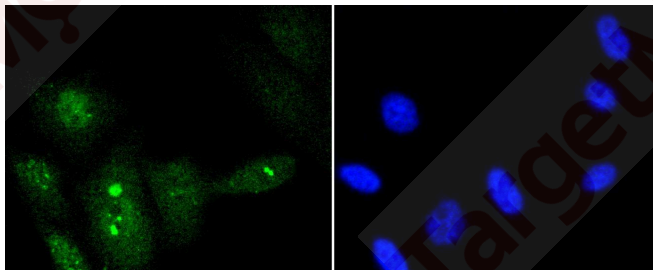
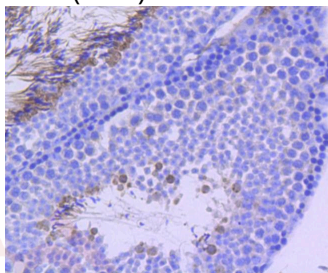
Product Details

Ig Type:	IgG
Reactivity:	Human
Conjugation:	Unconjugated
Molecular Weight:	Theoretical: 95 kDa.
Clone:	5K712
Purification:	ProA affinity purified

Applications

Verified Activity:

1. Immunohistochemical analysis of paraffin-embedded mouse testis tissue using anti-Phospho-p95/NBS1 (S343) antibody. Counter stained with hematoxylin.
2. ICC staining Phospho-p95/NBS1 (S343) in PC-3M cells (green). The nuclear counter stain is DAPI (blue). Cells were fixed in paraformaldehyde, permeabilised with 0.25% Triton X100/PBS.



Application: ICC/IF,IHC,IP,WB

Recommended WB: 1:1000-2000; IHC: 1:50-200; ICC/IF: 1:50-200

Properties

Stability & Storage: Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles.

Shipping: Shipping with blue ice.

Antigen Details

Immunogen:	A synthesized phosphopeptide: human p95/NBS1 around the phosphorylation site of Ser343
Antigen Species:	human
Uniprot ID:	O60934
Synonyms:	NBS;MGC87362;Nijmegen breakage syndrome 1;p95;NBS1;AT V2;ATV;nibrin;Nijmegen breakage syndrome protein 1;NBN (p-Ser343);FLJ10155;p-NBN (S343);NBN (p-S343);NBS 1;p-NBN (Ser343);Nijmegen breakage syndrome;p95 protein of the MRE11/RAD50 complex;Cell cycle regulatory protein p95;AT V1;Phospho-NBN (S343)

Research Background

Nijmegen breakage syndrome (NBS) is characterized by extreme radiation sensitivity, chromosomal instability and cancer. These phenotypes are similar to those of ataxia telangiectasia mutated (ATM) disease, where there is a deficiency in a protein kinase that is activated by DNA damage, indicating that the NBS1 (Nibrin) and ATM proteins may participate in common pathways. Nibrin is specifically phosphorylated in response to gamma-radiation, ultraviolet light and exposure to hydroxyurea. The phosphorylation of Nibrin requires catalytically active ATM. ATM physically interacts with and phosphorylates Nibrin on Serine 343 both in vitro and in vivo. Serine 343 is phosphorylated in vitro by ATM and the modification of this residue in vivo is essential for the cellular response to DNA damage. This response includes S-phase checkpoint activation, formation of the NBS1/Mre11/Rad50 nuclear foci and rescue of hypersensitivity to ionizing radiation.

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