

# Product Datasheet

## CtIP Antibody - BSA Free NB100-79810

Unit Size: 100 ul

Store at 4C. Do not freeze.

[www.novusbio.com](http://www.novusbio.com)



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### Publications: 5

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**NB100-79810**

CtIP Antibody - BSA Free

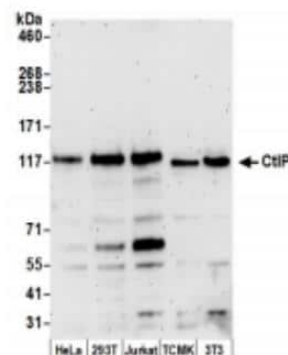
Product Information	
Unit Size	100 ul
Concentration	1.0 mg/ml
Storage	Store at 4C. Do not freeze.
Clonality	Polyclonal
Preservative	0.09% Sodium Azide
Isotype	IgG
Purity	Immunogen affinity purified
Buffer	Tris-Citrate/Phosphate (pH 7.0 - 8.0)

Product Description	
Description	Novus Biologicals Rabbit CtIP Antibody - BSA Free (NB100-79810) is a polyclonal antibody validated for use in WB, ICC/IF and IP. Anti-CtIP Antibody: Cited in 5 publications. All Novus Biologicals antibodies are covered by our 100% guarantee.
Host	Rabbit
Gene ID	5932
Gene Symbol	RBBP8
Species	Human, Mouse
Immunogen	The immunogen recognized by this antibody maps to a region between residue 850 and the C-terminus (residue 897) of human CtBP Interacting Protein (Retinoblastoma Binding Protein 8) using the numbering given in entry NP_002885.1 (GeneID 5932).

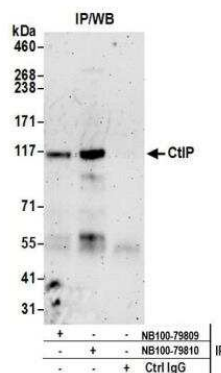
Product Application Details	
Applications	Western Blot, Immunocytochemistry/ Immunofluorescence, Immunoprecipitation
Recommended Dilutions	Western Blot 1:2000-1:10000, Immunocytochemistry/ Immunofluorescence, Immunoprecipitation 2 - 10 ug/mg lysate
Application Notes	Use in ICC/IF reported in scientific literature (PMID 26215093).

**Images**

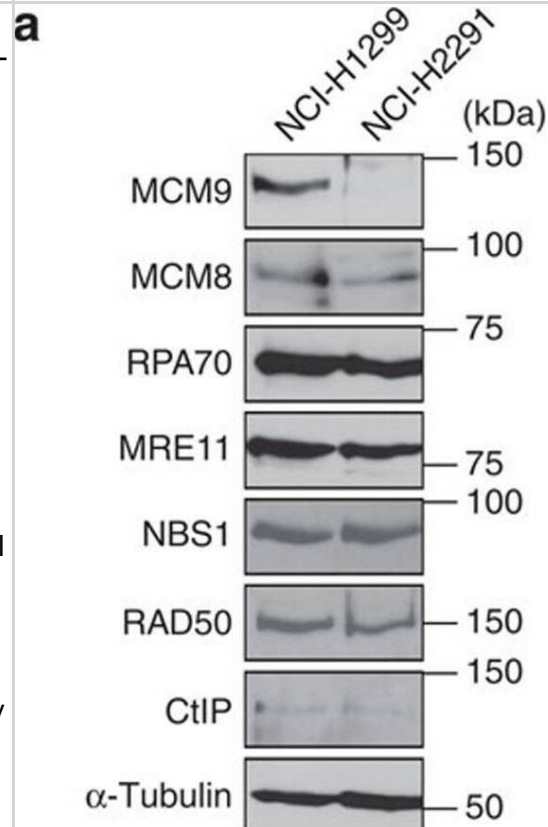
Western Blot: CtIP Antibody [NB100-79810] - Whole cell lysate (15 ug) from HeLa, HEK293T, Jurkat, mouse TCMK-1, and mouse NIH 3T3 cells prepared using NETN lysis buffer. Antibody: Affinity purified rabbit anti-CtIP antibody used for WB at 0.1 ug/ml. Detection: Chemiluminescence with an exposure time of 3 minutes.



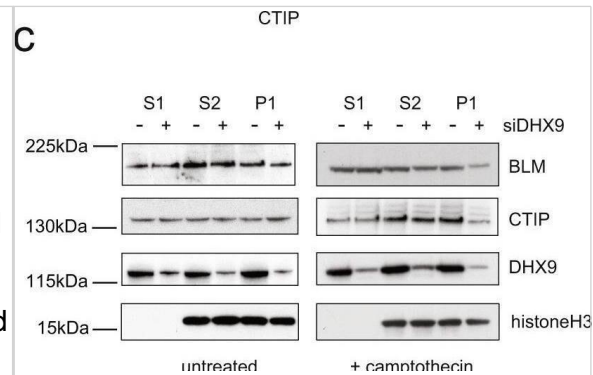
Immunoprecipitation: CtIP Antibody [NB100-79810] - Detection of human CtIP by western blot of immunoprecipitates. Samples: Whole cell lysate (1 mg for IP; 20% of IP loaded) from HEK293T cells prepared using NETN lysis buffer. Antibodies: Affinity purified rabbit anti-CtIP antibody NB100-79810 used for IP at 3 ug per reaction. CtIP was also immunoprecipitated by rabbit anti-CtIP antibody NB100-79809. For blotting immunoprecipitated CtIP, NB100-79810 was used at 1 ug/ml. Detection: Chemiluminescence with an exposure time of 3 minutes.



Functional inactivation of MCM9 in cancers. (a) Absence of MCM9 protein in NCI-H2291. Immunoblot of indicated proteins in lysates of NCI-H2291 and NCI-H1299 (control cell line). (b) Decrease of cisplatin-induced RPA70- or Mre11 foci-positive cells in NCI-H2291. \*\*\* $P < 0.005$ ; Student's t-test. (c) Decrease of HR efficiency in NCI-H2291. HR assay was performed by transient transfection of DSB recombination reporter and I-SceI expression plasmids as described in Methods section. HR efficiency was calculated by normalizing the percentage of GFP-positive cells to transfection efficiency in each cell line. \*\*\* $P < 0.005$ ; Student's t-test. (d) Transient expression of ectopic HA-MCM9 (WT) in NCI-H2291. (e) Restoration of relative resistance of NCI-H2291 to cisplatin by overexpression of MCM9. Cell viability was measured by clonogenic assay as described in Methods section. Top: representative wells. Bottom: quantification of viable cells. \*\* $P < 0.01$ ; Student's t-test. (f) Protein expression of MCM9 in prostate cancer cells. Top: amount of MCM9 protein in each cancer cell line measured by immunoblotting. All lanes were in the same blot and exposed similarly. Bottom: MCM9 signal quantified with ImageJ software, normalized to  $\alpha$ -tubulin and expressed relative to 293T. (g) Correlation of MCM9 levels to IC75 to cisplatin in indicated cancer cell lines (also see viability curves measured by MTT assay in Supplementary Fig. 12). All error bars represent s.d. of the mean from triplicates. EV, empty vector. Image collected and cropped by CiteAb from the following open publication (<https://pubmed.ncbi.nlm.nih.gov/26215093>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



DHX9 promotes the recruitment of CTIP and BLM to DNA damage. a Fluorescence images (left panel) and graph (right panel) showing that localization of BLM to camptothecin-induced DNA damage foci is impaired in cells knocked down for DHX9. b Fluorescence images (left panel) and graph (right panel) showing that localization of CTIP to camptothecin-induced DNA damage foci is impaired in cells knocked down for DHX9. Quantification of  $n$  cells (as indicated) from three pooled biologically independent experiments were performed in (a) and (b). Means were shown to be significantly different using one-way ANOVA with post hoc Tukey's test ( $****p < 0.0001$ ). c Western blot of fractionated cell extracts showing that localization of BLM and CTIP to chromatin (P1 fraction) in response to camptothecin-induced DNA damage is reduced in cells knocked down for DHX9. Localization of BLM and CTIP in cytoplasmic (S1) and nuclear fractions (S2) is not decreased. Histone H3 is shown as a marker of S2 and P1 fractions. d DNA synthesis is impaired in DHX9 and BRCA1 deficient cells treated with camptothecin ( $5 \mu\text{M}$  for 2 h). This defect is not suppressed by knockdown of 53BP1. Right panel shows representative images for the incorporation of CldU and IdU nucleotide analogs as well as merged images. The left panel shows graphical data of cells stained with both CldU and IdU as a percentage of total cells stained with CldU. Graphs include data from three biologically independent experiments. Mean and error bars indicating one standard deviation are also indicated. Statistical significance for all experiments was demonstrated using one-way ANOVA with post hoc Tukey's test ( $****p < 0.0001$ ,  $*p < 0.1$ , ns not significant). Source data are provided as a Source Data file. Image collected and cropped by CiteAb from the following open publication (<https://pubmed.ncbi.nlm.nih.gov/34226554>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



## Publications

Chakraborty P, Hiom K DHX9-dependent recruitment of BRCA1 to RNA promotes DNA end resection in homologous recombination *Nature communications* 2021-07-05 [PMID: 34226554] (WB, ICC/IF)

Quennet V, Beucher A, Barton O et al CtIP and MRN promote non-homologous end-joining of EPE-induced DNA double-strand breaks in G1 *Nucleic Acids Res* 2011-03-01 [PMID: 21087997]

Lee K Y, Im J. S, et al. ASF1a Promotes Non-homologous End Joining Repair by Facilitating Phosphorylation of MDC1 by ATM at Double-Strand Breaks. *Mol Cell* 2017-10-05 [PMID: 28943310] (WB)

Lee KY, Im JS, Shibata E et al. MCM8-9 complex promotes resection of double-strand break ends by MRE11-RAD50-NBS1 complex. *Nat Commun.* 2015-07-28 [PMID: 26215093] (WB, ICC/IF, Human)

Hu Y, Scully R, Sobhian B et al. RAP80-directed tuning of BRCA1 homologous recombination function at ionizing radiation-induced nuclear foci. *Genes Dev* 2011-04-01 [PMID: 21406551]



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### **Products Related to NB100-79810**

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NBP2-33376H	Blue Marker Antibody (6F4-F6) [HRP]
HAF008	Goat anti-Rabbit IgG Secondary Antibody [HRP]
NB7160	Goat anti-Rabbit IgG (H+L) Secondary Antibody [HRP]
NBP2-24891	Rabbit IgG Isotype Control

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### **Limitations**

This product is for research use only and is not approved for use in humans or in clinical diagnosis. Primary Antibodies are guaranteed for 1 year from date of receipt.

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