

**Anti-CtIP (Ser326) Antibody**

**Our Anti-CtIP (Ser326) rabbit polyclonal phosphospecific primary antibody from PhosphoSolutions is p  
Catalog # AN1350**

**Specification**

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**Anti-CtIP (Ser326) Antibody - Product Information**

|                   |                        |
|-------------------|------------------------|
| Primary Accession | <a href="#">O99708</a> |
| Host              | <b>Rabbit</b>          |
| Clonality         | <b>Polyclonal</b>      |
| Isotype           | <b>IgG</b>             |
| Calculated MW     | <b>101942</b>          |

**Anti-CtIP (Ser326) Antibody - Additional Information**

Gene ID **5932**

**Other Names**

COM1 antibody, COM1\_HUMAN antibody, CtBP interacting protein antibody, CtBP-interacting protein antibody, CtIP antibody, DNA endonuclease RBBP8 antibody, JWDS antibody, RB binding protein 8 endonuclease antibody, RBBP-8 antibody, RBBP8 antibody, Retinoblastoma-binding protein 8 antibody, Retinoblastoma-interacting protein and myosin-like antibody, Rim antibody, SAE2 antibody, SCKL2 antibody, Sporulation in the absence of SPO11 protein 2 homolog antibody

**Target/Specificity**

CtIP, C-terminal binding protein-interacting protein, is a DNA endonuclease activated by double stranded breaks (DSBs). DSB repairs can be performed by either one of two mechanisms; non-homologous end joining (NHEJ) or homologous recombination (HR). NHEJ is the predominant DSB repair pathway throughout the entire cell cycle, most importantly in the G1 phase (Rothkamm et al, 2003); while HR is important for repairing DSBs in S and G2 phases (Beucher et al, 2009). CtIP controls DSB resection; an event that only occurs in HR during G2-phase. Phosphorylation of Thr-847 dictates the resection efficiency (Huertas et al, 2008). Furthermore, it has been found that DSBs undergo resection and repair in G1-phase cells via a process requiring Plk3 phosphorylation of CtIP at Ser-327 and Thr-847 (Barton et al, 2014). Several additional phosphorylation sites within CtIP have been identified, but their significance in the repair of DNA have yet to be determined.

**Format**

Antigen Affinity Purified from Pooled Serum

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

**Precautions**

Anti-CtIP (Ser326) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**Shipping**

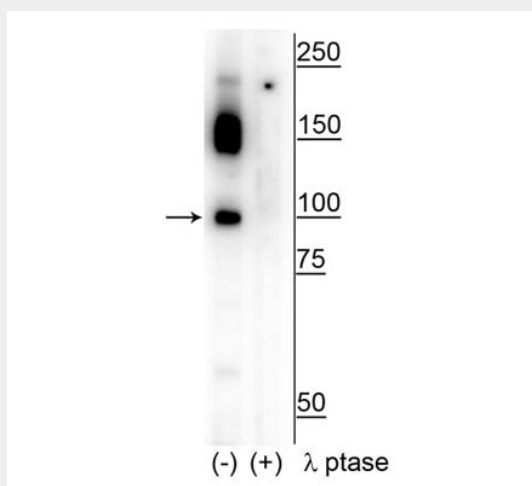
Blue Ice

## Anti-CtIP (Ser326) 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](#)

## Anti-CtIP (Ser326) Antibody - Images



Western blot of human T47D cell lysate showing specific immunolabeling of the ~100 kDa CtIP phosphorylated at Ser326 in the first lane (-). Phosphospecificity is shown in the second lane (+) where immunolabeling is completely eliminated by blot treatment with lambda phosphatase ( $\lambda$ -Ptase, 1200 units for 30 min).

## Anti-CtIP (Ser326) Antibody - Background

CtIP, C-terminal binding protein-interacting protein, is a DNA endonuclease activated by double stranded breaks (DSBs). DSB repairs can be performed by either one of two mechanisms; non-homologous end joining (NHEJ) or homologous recombination (HR). NHEJ is the predominant DSB repair pathway throughout the entire cell cycle, most importantly in the G1 phase (Rothkamm et al, 2003); while HR is important for repairing DSBs in S and G2 phases (Beucher et al, 2009). CtIP controls DSB resection; an event that only occurs in HR during G2-phase. Phosphorylation of Thr-847 dictates the resection efficiency (Huertas et al, 2008). Furthermore, it has been found that DSBs undergo resection and repair in G1-phase cells via a process requiring Plk3 phosphorylation of CtIP at Ser-327 and Thr-847 (Barton et al, 2014). Several additional phosphorylation sites within CtIP have been identified, but their significance in the repair of DNA have yet to be determined.