

## Product Datasheet

# Anti-CtIP (Ser327)

 **Pooled Serum**

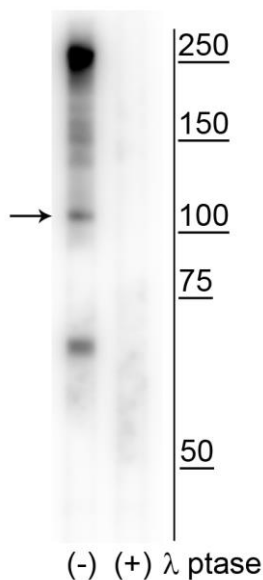
### Overview

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<b>Catalog #</b>	p1012-327
<b>Host Species</b>	Rabbit Polyclonal
<b>Format</b>	Antigen Affinity Purified from Pooled Serum
<b>Applications</b>	WB 1:1000
<b>Species Tested</b>	Human
<b>Expected Reactivity</b>	Non-Human Primate
<b>Immunogen</b>	Synthetic phospho-peptide corresponding to amino acid residues surrounding Ser327 of human CtIP, conjugated to keyhole limpet hemocyanin (KLH).
<b>Molecular Weight</b>	100 kDa
<b>Cite this Antibody</b>	PhosphoSolutions Cat# p1012-327, RRID:AB_2651148

### Images

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Western blot of human T47D cell lysate showing specific immunolabeling of the ~100 kDa CtIP phosphorylated at Ser<sup>327</sup> 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).

## Details

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<b>Target Description</b>	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.
<b>Specificity</b>	Specific for endogenous levels of the ~100 kDa CtIP protein phosphorylated at Ser327. Immunolabeling is completely eliminated by treatment with $\lambda$ -phosphatase.
<b>Production/Purification</b>	Prepared from pooled rabbit serum by affinity purification via sequential chromatography on phospho and non-phosphopeptide affinity columns.
<b>Quality Control</b>	Western blots performed on each lot.
<b>Buffer</b>	10 mM HEPES (pH 7.5), 150 mM NaCl, 100 $\mu$ g per ml BSA and 50% glycerol.
<b>Storage</b>	Storage at -20°C is recommended, as aliquots may be taken without freeze/thawing due to presence of 50% glycerol.
<b>Stability</b>	After date of receipt, stable for at least 1 year at -20°C.

## Significant Citations

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Zhou, Q., Tu, X., Hou, X., Yu, J., Zhao, F., Huang, J., Kloeber, J., Olson, A., Gao, M., Luo, K., Zhu, S., Wu, Z., Zhang, Y., Sun, C., Zeng, X., Schoolmeester, K.J., Weroha, J.S., Hu, X., Jiang, Y. and Wang, L. (2024). Syk-dependent homologous recombination activation promotes cancer resistance to DNA targeted therapy. *Drug Resistance Updates: Reviews and Commentaries in Antimicrobial and Anticancer Chemotherapy*, [online] 74, p.101085.

Gao, M., Guo, G., Huang, J., Kloeber, J.A., Zhao, F., Deng, M., Tu, X., Kim, W., Zhou, Q., Zhang, C. and Yin, P., 2020. USP52 regulates DNA end resection and chemosensitivity through removing inhibitory ubiquitination from CtIP. *Nature Communications*, 11(1), pp.1-13.

Barton, O., Naumann, S.C., Diemer-Biehs, R., Künzel, J., Steinlage, M., Conrad, S., Makharashvili, N., Wang, J., Feng, L., Lopez, B.S. and Paull, T.T., 2014. Polo-like kinase 3 regulates CtIP during DNA double-strand break repair in G1. *J Cell Biol*, 206(7), pp.877-894.

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