

# Phospho-PARP1(S864) Antibody

Affinity Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP3786f

# **Specification**

# Phospho-PARP1(S864) Antibody - Product Information

Application DB,E
Primary Accession P09874

Other Accession <u>P27008</u>, <u>P11103</u>, <u>Q9R152</u>, <u>P18493</u>,

NP\_001609.2

Reactivity Human

Predicted Bovine, Hamster, Mouse, Rat

Host Rabbit
Clonality Polyclonal
Isotype Rabbit IgG
Calculated MW 113084

# Phospho-PARP1(S864) Antibody - Additional Information

### Gene ID 142

### **Other Names**

Poly [ADP-ribose] polymerase 1, PARP-1, ADP-ribosyltransferase diphtheria toxin-like 1, ARTD1, NAD(+) ADP-ribosyltransferase 1, ADPRT 1, Poly[ADP-ribose] synthase 1, PARP1, ADPRT, PPOL

# Target/Specificity

This PARP1 Antibody is generated from rabbits immunized with a KLH conjugated synthetic phosphopeptide corresponding to amino acid residues surrounding S864 of human PARP1.

#### **Dilution**

DB~~1:500

#### **Format**

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

### **Storage**

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

#### **Precautions**

Phospho-PARP1(S864) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

# Phospho-PARP1(S864) Antibody - Protein Information

Name PARP1 {ECO:0000303|PubMed:21680843, ECO:0000312|HGNC:HGNC:270}



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Function Poly-ADP-ribosyltransferase that mediates poly-ADP- ribosylation of proteins and plays a
key role in DNA repair (PubMed: 17177976, PubMed: 18055453, PubMed: 18172500,
PubMed: 19344625, PubMed: 19661379, PubMed: 20388712, PubMed: 21680843,
PubMed: <u>22582261</u>, PubMed: <u>23230272</u>, PubMed: <u>25043379</u>, PubMed: <u>26344098</u>,
PubMed: <u>26626479</u>, PubMed: <u>26626480</u>, PubMed: <u>30104678</u>, PubMed: <u>31796734</u>,
PubMed: 32028527, PubMed: 32241924, PubMed: 32358582, PubMed: 33186521,
PubMed: 34465625, PubMed: 34737271). Mediates glutamate, aspartate, serine, histidine or
tyrosine ADP-ribosylation of proteins: the ADP-D-ribosyl group of NAD(+) is transferred to the
acceptor carboxyl group of target residues and further ADP-ribosyl groups are transferred to the
2'-position of the terminal adenosine moiety, building up a polymer with an average chain length
of 20-30 units (PubMed: 19764761, PubMed: 25043379, PubMed: 28190768, PubMed: 29954836,
PubMed:35393539, PubMed:7852410, PubMed:9315851). Serine ADP-ribosylation of proteins
constitutes the primary form of ADP-ribosylation of proteins in response to DNA damage
(PubMed:33186521, PubMed:34874266). Specificity for the different amino acids is conferred by
interacting factors, such as HPF1 and NMNAT1 (PubMed: 28190768, PubMed: 29954836,
PubMed:32028527, PubMed:33186521, PubMed:33589610, PubMed:34625544,
PubMed:34874266). Following interaction with HPF1, catalyzes serine ADP-ribosylation of target
proteins; HPF1 confers serine specificity by completing the PARP1 active site (PubMed: 28190768,
PubMed: <u>32028527</u>, PubMed: <u>33186521</u>, PubMed: <u>33589610</u>,
PubMed: 34625544, PubMed: 34874266). Also catalyzes tyrosine ADP-ribosylation of target proteins
following interaction with HPF1 (PubMed: 29954836, PubMed: 30257210). Following interaction with
NMNAT1, catalyzes glutamate and aspartate ADP- ribosylation of target proteins; NMNAT1 confers
glutamate and aspartate specificity (By similarity). PARP1 initiates the repair of DNA breaks:
recognizes and binds DNA breaks within chromatin and recruits HPF1, licensing serine
ADP-ribosylation of target proteins, such as histones (H2BS6ADPr and H3S10ADPr), thereby
promoting decompaction of chromatin and the recruitment of repair factors leading to the
reparation of DNA strand breaks (PubMed: 17177976, PubMed: 18172500, PubMed: 19344625,
PubMed: 19661379, PubMed: 23230272, PubMed: 27067600, PubMed: 34465625,
PubMed: 34874266). HPF1 initiates serine ADP-ribosylation but restricts the polymerase activity of
PARP1 in order to limit the length of poly- ADP-ribose chains (PubMed: 33683197,
PubMed: 34732825, PubMed: 34795260). In addition to base excision repair (BER) pathway, also
involved in double-strand breaks (DSBs) repair: together with TIMELESS, accumulates at DNA
damage sites and promotes homologous recombination repair by mediating poly-ADP-ribosylation
(PubMed: 26344098, PubMed: 30356214). Mediates the poly-ADP-ribosylation of a number of
proteins, including itself, APLF, CHFR, RPA1 and NFAT5 (PubMed: 17396150, PubMed: 19764761,
PubMed: 24906880, PubMed: 34049076). In addition to proteins, also able to ADP-ribosylate DNA:
catalyzes ADP-ribosylation of DNA strand break termini containing terminal phosphates and a
2'-OH group in single- and double-stranded DNA, respectively (PubMed: 27471034). Required for
PARP9 and DTX3L recruitment to DNA damage sites (PubMed: 23230272). PARP1- dependent
PARP9-DTX3L-mediated ubiquitination promotes the rapid and specific recruitment of
53BP1/TP53BP1, UIMC1/RAP80, and BRCA1 to DNA damage sites (PubMed:23230272).
PARP1-mediated DNA repair in neurons plays a role in sleep: senses DNA damage in neurons and
promotes sleep, facilitating efficient DNA repair (By similarity). In addition to DNA repair, also
involved in other processes, such as transcription regulation, programmed cell death, membrane
repair, adipogenesis and innate immunity (PubMed: 15607977, PubMed: 17177976,
PubMed: <u>19344625</u>, PubMed: <u>27256882</u>, PubMed: <u>32315358</u>, PubMed: <u>32844745</u>,
PubMed:35124853, PubMed:35393539, PubMed:35460603). Acts as a repressor of transcription:
binds to nucleosomes and modulates chromatin structure in a manner similar to histone H1,
thereby altering RNA polymerase II (PubMed: 15607977, PubMed: 22464733). Acts both as a
positive and negative regulator of transcription elongation, depending on the context
(PubMed: <u>27256882</u>, PubMed: <u>35393539</u>). Acts as a positive regulator of transcription elongation by
mediating poly-ADP- ribosylation of NELFE, preventing RNA-binding activity of NELFE and relieving
transcription pausing (PubMed: 27256882). Acts as a negative regulator of transcription elongation
in response to DNA damage by catalyzing poly-ADP-ribosylation of CCNT1, disrupting the phase
separation activity of CCNT1 and subsequent activation of CDK9 (PubMed: 35393539). Involved in
replication fork progression following interaction with CARM1: mediates poly-ADP-ribosylation at
replication forks, slowing fork progression (PubMed: 33412112). Poly-ADP-ribose chains generated
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by PARP1 also play a role in poly-ADP-ribose-dependent cell death, a process named parthanatos (By similarity). Also acts as a negative regulator of the cGAS-STING pathway (PubMed:32315358, PubMed:32844745, PubMed:35460603). Acts by mediating poly-ADP- ribosylation of CGAS: PARP1 translocates into the cytosol following phosphorylation by PRKDC and catalyzes poly-ADP-ribosylation and inactivation of CGAS (PubMed:35460603). Acts as a negative regulator of adipogenesis: catalyzes poly-ADP-ribosylation of histone H2B on 'Glu- 35' (H2BE35ADPr) following interaction with NMNAT1, inhibiting phosphorylation of H2B at 'Ser-36' (H2BS36ph), thereby blocking expression of pro-adipogenetic genes (By similarity). Involved in the synthesis of ATP in the nucleus, together with NMNAT1, PARG and NUDT5 (PubMed:27257257). Nuclear ATP generation is required for extensive chromatin remodeling events that are energy-consuming (PubMed:27257257).

### **Cellular Location**

Chromosome. Nucleus. Nucleus, nucleolus. Cytoplasm, cytosol. Note=Localizes to sites of DNA damage (PubMed:22683995, PubMed:23230272, PubMed:26344098, PubMed:27568560, PubMed:30675909, PubMed:32241924, PubMed:32358582, PubMed:34625544, PubMed:34795260). Recognizes (via PARP-type zinc-fingers) and binds DNA strand breaks (PubMed:22683995). Also binds normal/undamaged chromatin (PubMed:15607977). Auto poly-ADP-ribosylation promotes dissociation from chromatin (PubMed:15607977, PubMed:30675909, PubMed:32358582, PubMed:34625544). Extracted from chromatin by VCP/p97 following sumoylation and ubiquitination (PubMed:35013556). Translocates from the nucleus to the cytosol following phosphorylation by PRKDC (PubMed:35460603). Recruited to replication forks following interaction with CARM1 (PubMed:33412112). [Poly [ADP-ribose] polymerase 1, processed C- terminus]: Cytoplasm. Note=Following cleavage by caspase-3 (CASP3) and caspase-7 (CASP7) in response to apoptosis, translocates into the cytoplasm, where the auto-poly-ADP- ribosylated form serves as a poly-ADP-ribose carrier to induce AIFM1- mediated apoptosis.

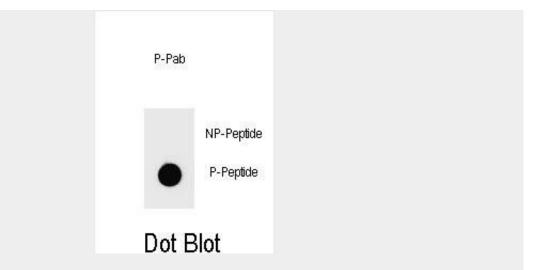
# Phospho-PARP1(S864) Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- <u>Immunoprecipitation</u>
- Flow Cytomety
- Cell Culture

# Phospho-PARP1(S864) Antibody - Images





Dot blot analysis of Phospho-PARP1-S864 Antibody Phospho-specific Pab (Cat. #AP3786f) on nitrocellulose membrane. 50ng of Phospho-peptide or Non Phospho-peptide per dot were adsorbed. Antibody working concentrations are 0.6ug per ml.

# Phospho-PARP1(S864) Antibody - Background

This gene encodes a chromatin-associated enzyme, poly(ADP-ribosyl)transferase, which modifies various nuclear proteins by poly(ADP-ribosyl)ation. The modification is dependent on DNA and is involved in the regulation of various important cellular processes such as differentiation, proliferation, and tumor transformation and also in the regulation of the molecular events involved in the recovery of cell from DNA damage. In addition, this enzyme may be the site of mutation in Fanconi anemia, and may participate in the pathophysiology of type I diabetes.

# Phospho-PARP1(S864) Antibody - References

Majewski, P.M., et al. J. Biol. Chem. 285(45):34828-34838(2010) Kim, M., et al. Cancer Sci. 101(11):2436-2442(2010) Dong, Y., et al. Cancer Res. 70(20):8088-8096(2010) Krishnakumar, R., et al. Mol. Cell 39(5):736-749(2010) Lee, K.A., et al. Rheumatol. Int. (2010) In press: