

PARP1 Antibody (N-term)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP13308A

Specification

PARP1 Antibody (N-term) - Product Information

Application	WB,E
Primary Accession	P09874
Other Accession	P27008 , P11103 , O9R152 , NP_001609.2
Reactivity	Human
Predicted	Hamster, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	113084
Antigen Region	58-87

PARP1 Antibody (N-term) - 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 peptide between 58-87 amino acids from the N-terminal region of human PARP1.

Dilution

WB~~1:1000

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

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

PARP1 Antibody (N-term) - Protein Information

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

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:[22582261](#), PubMed:[23230272](#), PubMed:[25043379](#), PubMed:[26344098](#), PubMed:[26626479](#), PubMed:[26626480](#), PubMed:[30104678](#), PubMed:[31796734](#), 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:[29954836](#), PubMed:[32028527](#), PubMed:[33186521](#), PubMed:[33589610](#), 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:[19344625](#), PubMed:[27256882](#), PubMed:[32315358](#), PubMed:[32844745](#), 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:[27256882](#), PubMed:[35393539](#)). 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

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-adipogenic 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

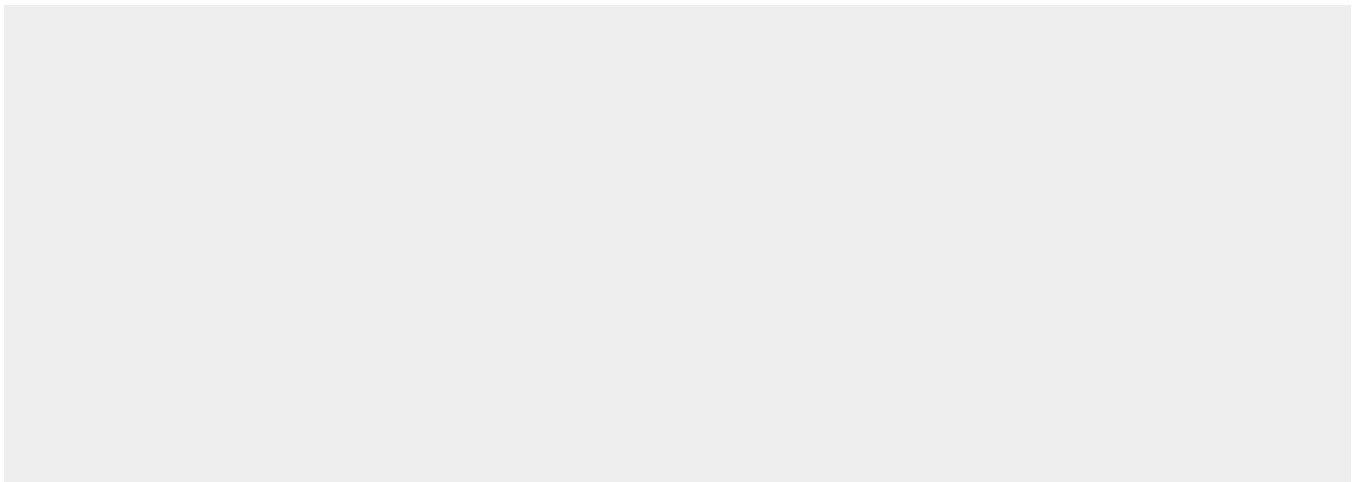
Chromosome. Nucleus. Nucleus, nucleolus. Cytoplasm, cytosol. Note=Localizes to sites of DNA damage (PubMed:22683995, PubMed:23230272, PubMed:26344098, PubMed:27568560, PubMed:30675909, PubMed:32241924, PubMed:32358582, PubMed:34625544, PubMed:34795260). Recognizes (via PARP-type zinc-fingers) and binds 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.

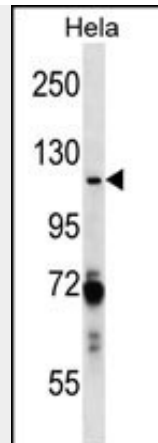
PARP1 Antibody (N-term) - Protocols

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](#)

PARP1 Antibody (N-term) - Images





PARP1 Antibody (N-term) (Cat. #AP13308a) western blot analysis in HeLa cell line lysates (35ug/lane). This demonstrates the PARP1 antibody detected the PARP1 protein (arrow).

PARP1 Antibody (N-term) - Background

This gene encodes a chromatin-associated enzyme, poly(ADP-ribosyl)transferase, which modifies various nuclear proteins by poly(ADP-ribosylation). 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.

PARP1 Antibody (N-term) - 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 :