abcam

ab110315

Anti-Cleaved PARP1 antibody [4B5BD2]

Lab Essentials | KO Validated

Key facts

Isotype	IgG1
Host species	Mouse
Storage buffer	pH: 7.5 Preservative: 0.02% Sodium azide Constituents: HEPES buffered saline
Form	Liquid
Clonality	Monoclonal
Immunogen	The exact immunogen used to generate this antibody is proprietary information.
Clone number	4B5BD2
Purification technique	Precipitation Ammonium Sulphate
Specificity	ab110315 reacts with the N-terminal end formed by the cleavage adjacent to Asp214; it thus recognizes the apoptosis-specific 89 kDa catalytic domain fragment, but it does not recognize the full-length PARP1 or the 24 kDa DNA binding domain fragment.
Light chain type	kappa
Concentration	1 mg/mL The concentration of this product may be batch-dependent Batch concentration finder →
Purification notes	The antibody was produced in vitro using hybridomas grown in serum-free medium, and then purified by ammonium sulfate precipiation.

Reactivity data

Flow Cyt

Tested

Species	Human
Dilution info	1 µg/mL
Notes	ab170190 - Mouse monoclonal IgG1, is suitable for use as an isotype control with this antibody.

WB

Tested

Species	Human
Dilution info	0.25-1 µg/mL
Notes	-

In-Cell ELISA

Tested

Species	Human
Dilution info	1 µg/mL
Notes	-

ICC/IF

Tested

Species	Human
Dilution info	1 µg/mL
Notes	-

Target data

See full target information PARP1

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-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). Poly [ADP-ribose] polymerase 1, processed C-terminus. Promotes AIFM1-mediated apoptosis (PubMed:33168626). This form, which translocates into the cytoplasm following cleavage by caspase-3 (CASP3) and caspase-7 (CASP7) in response to apoptosis, is auto-poly-ADP-ribosylated and serves as a poly-ADP-ribose carrier to induce AIFM1-mediated apoptosis (PubMed:33168626). Poly [ADP-ribose] polymerase 1, processed N-terminus. This cleavage form irreversibly binds to DNA breaks and interferes with DNA repair, promoting DNA damage-induced apoptosis.

Storage

Shipped at conditions	Blue Ice
Appropriate short-term storage conditions	+4°C
Appropriate long-term storage conditions	+4°C
Storage information	Do Not Freeze

Notes

This monoclonal antibody to cleaved PARP1 has been knockout validated in Western blot. The expected band for cleaved PARP1 was observed in wild type cells and the band was not seen in knockout cells.

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Supplementary info

This supplementary information is collated from multiple sources and compiled automatically.

Activity summary	Cleaved PARP1 also known as cPARP is a fragment of the PARP1 protein an important DNA repair enzyme. The full PARP1 protein has a molecular weight of approximately 116 kDa but after cleavage during apoptosis the cleaved PARP1 fragments typically have a molecular weight of around 89 kDa and 24 kDa. PARP1 is expressed abundantly in the cell nucleus where it plays important roles in maintaining genomic integrity. The cleavage of PARP1 is a common marker for cell apoptosis pointing towards its breakdown in response to cellular stress.
Biological function summary	The enzymatic function of PARP1 involves the transfer of ADP-ribose units from NAD+ to target proteins a process known as ADP-ribosylation. PARP1 operates as a part of the base excision repair complex essential in DNA repair processes. The cleaved form of PARP1 no longer facilitates DNA repair marking a shift towards apoptosis. When PARP1 is cleaved it indicates caspase activity implying cells are undergoing programmed cell death.
Pathways	Cleaved PARP1 is deeply involved in the apoptosis and DNA damage response pathways. In the apoptosis pathway PARP1 interacts with key proteins like caspase-3 which cleaves PARP during apoptosis. In the DNA damage response PARP1 collaborates with proteins such as XRCC1 facilitating the base excision repair pathway important for fixing single-strand DNA breaks. These pathways highlight the dual role of PARP1 in promoting cell survival through repair and cell death via apoptosis.
Associated diseases and disorders	Cleaved PARP1 serves as an important marker in cancer and neurodegenerative diseases. In cancer research elevated levels of cleaved PARP1 suggest increased rates of apoptosis in response to anti-cancer therapies linking it to tumor suppression efforts. In neurodegenerative diseases excessive activation and cleavage of PARP1 can result in cell death exacerbating conditions like Alzheimer's disease. Through these contexts cleaved PARP1 connects to other therapeutic targets such as caspase proteins in cancer and to potential PARP inhibitors in neurodegenerative disorders.

Product promise

Tested

We have tested this species and application combination and it works. It is covered by our product promise.

Expected

We have not tested this specific species and application combination in-house, but expect it will work. It is covered by our product promise.

Predicted

This species and application combination has not been tested, but we predict it will work based on strong homology. However, this combination is not covered by our product promise.

Not recommended

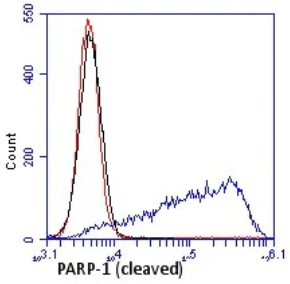
We do not recommend this combination. It is not covered by our product promise.

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In the unlikely event of one of our products not working as expected, you are covered by our product promise.

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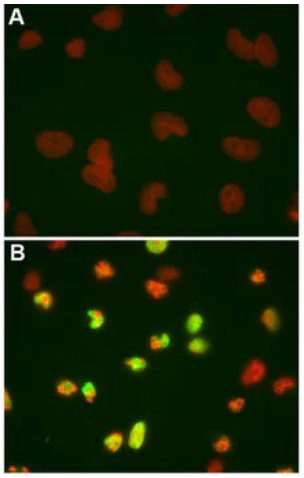
6 product images



Flow Cytometry - Anti-Cleaved PARP1 antibody [4B5BD2] (ab110315)

Cleaved PARP1 Flow Cytometry staining using mouse Anti-Cleaved PARP1 antibody

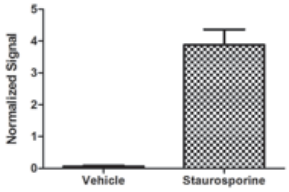
Flow cytometry analysis of apoptosis using ab110315. HL-60 cells were treated with 1 µM Staurosporin for 4 hours (blue) or vehicle control (red). Control cells were also stained with an equal amount of an isotype control antibody (black).



Immunocytochemistry/ Immunofluorescence - Anti-Cleaved PARP1 antibody [4B5BD2] (ab110315)

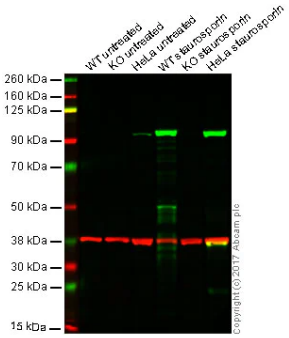
Cleaved PARP1 Immunocytochemistry/ Immunofluorescence staining using mouse Anti-Cleaved PARP1 antibody

Immunocytochemistry images of stained untreated (A) and 4 hours 1 µM Staurosporine-treated (B) Human HeLa cells. The cells were paraformaldehyde fixed (4%, 20 minutes) and Triton X-100 permeabilized (0.1%, 15 minutes). The cells were incubated with 1.0 µg/ml ab110315 for 2 hours at room temperature or over night at 4°C. 10% goat serum was used as the blocking agent for all blocking steps. The secondary antibody was Alexa Fluor® 488 goat anti-mouse IgG (H+L) (in green) used at 2.0 µg/ml for 2 hours. DAPI was used to stain the cell nuclei (in red). Heat induced antigen retrieval (0.1 M Tris-HCl, 5% urea, pH 9.5 for 5 min at 95°C) improves signal. Note that the ab110315 labels only condensed and/or fragmented nuclei of apoptotic Staurosporine-treated cells.



In-Cell ELISA - Anti-Cleaved PARP1 antibody [4B5BD2] (ab110315)

In-Cell ELISA (ICE) using ab110315 on HeLa cells treated with Staurosporine to induce apoptosis. HeLa cells were seeded overnight (50,000 cells/well), treated for 4 hours with 1 µM Staurosporine or with the drug vehicle (DMSO), fixed for Detaching Adherent Cells and analyzed.



Western blot - Anti-Cleaved PARP1 antibody [4B5BD2] (ab110315)

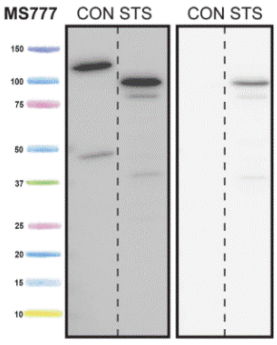
Cleaved PARP1 Western blot staining using mouse Anti-Cleaved PARP1 antibody

Lane 1: Wild type HAP1 (untreated) whole cell lysate (20 µg)
Lane 2: PARP1 (untreated) knockout HAP1 (untreated) whole cell lysate (20 µg)
Lane 3: HeLa (untreated) whole cell lysate (20 µg)
Lane 4: HAP1 (staurosporine treated, 1 uM, 4 hr) whole cell lysate (20 µg)
Lane 5: PARP1 (staurosporine treated, 1 uM, 4 hr) knockout HAP1 whole cell lysate (20 µg)
Lane 6: HeLa (staurosporine treated, 1 uM, 4 hr) whole cell lysate (20 µg)
Lanes 1 - 6: Merged signal (red and green). Green - ab110315 observed at 100 kDa. Red - loading control, ab181602, observed at 37 kDa

ab110315 detected the expected band for cleaved PARP1 in wild type HAP1 cells treated with staurosporine and the band was not seen in PARP1 knockout cells treated with staurosporine. Wild-type and PARP1 knockout samples were subjected to SDS-PAGE. ab110315 and ab181602 (Rabbit anti GAPDH loading control) were incubated overnight at 4°C at 1 ug/ml and 1/10000 dilution respectively. Blots were developed with Goat anti-Mouse IgG H&L (IRDye® 800CW) preabsorbed ab216772 and Goat anti-Rabbit IgG H&L (IRDye® 680RD) preabsorbed ab216777 secondary antibodies at 1/10000 dilution for 1 hour at room temperature before imaging.

All lanes:
Western blot - Anti-Cleaved PARP1 antibody [4B5BD2] (ab110315)

Predicted band size: 113 kDa



Western blot - Anti-Cleaved PARP1 antibody [4B5BD2] (ab110315)

Western Blot analysis using ab110315 antibody and 20 µg of untreated (CON) or 4 hours 1 µM Staurosporine-treated (STS) HeLa cells. Blots were incubated with an antibody that recognizes both the full-length PARP1 and its 89 kDa fragment (left panel), or 1.0 µg/mL PARP1 (cleaved) antibody (ab110315) (right panel). Appropriate HRP-conjugated secondary antibodies followed by ECL detection were used. Note that the MS777 antibody recognizes the apoptosis-specific 89 kDa fragment of PARP1 but it does not recognize the full-length PARP1.
Lanes 1 - 2:
Antibody that recognizes full-length PARP1

Lanes 3 - 4:
Western blot - Anti-Cleaved PARP1 antibody [4B5BD2] (ab110315) at 1 µg/mL

Lanes 1 and 3:
untreated HeLa cells at 20 µg

Lanes 2 and 4:
HeLa cells treated with 1 µM Staurosporinefor 4 hours at 20 µg

Predicted band size: 113 kDa

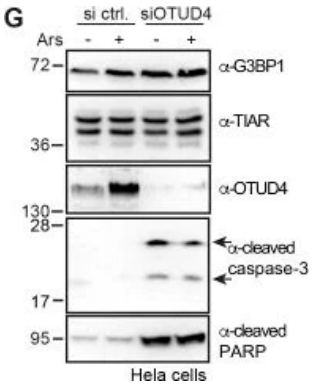


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Western blot - Anti-Cleaved PARP1 antibody [4B5BD2] (ab110315)

Cleaved PARP1 Western blot staining using mouse Anti-Cleaved PARP1 antibody

Cleaved PARP1 western blot using anti-Cleaved PARP1 antibody [4B5BD2] ab110315. Publication image and figure legend from Das, R., Schwintzer, L., et al., 2019, J Cell Sci, PubMed 31138677.

ab110315 was used in this publication in western blot. This may not be the same as the application(s) guaranteed by Abcam. For a full list of applications guaranteed by Abcam for ab110315 please see the product overview.

OTUD4 is required for correct stress granule formation. (A) Knockdown of OTUD4 decreases stress granule size and increases stress granule number. HeLa cells were transfected with two different siRNA oligonucleotides (oligo5 and oligo7) against OTUD4 or with control siRNA. OTUD4 and TIAR staining after 30 min arsenite treatment (0.5 mM) reveals differences in stress granule formation in the absence of OTUD4. White arrows indicate residual OTUD4 protein after knockdown, resulting in larger stress granules, resembling control siRNA-transfected cells. Scale bar: 10 µm. The experiment was repeated three times. (B) Quantification of the average number of stress granules per cell. Stress granules were scored in at least 200 cells per condition using CellProfiler software and the average number of granules per cell was depicted in a box plot. (C) Quantification of the average stress granule area. Granule area was determined with CellProfiler software as in B, and size distribution is presented as a box plot. (D) Re-introduction of siRNA-resistant OTUD4 rescues defects in stress granule formation. HeLa cells were transfected with FLAG–OTUD4 or its catalytic inactive mutant (C45A) 24 h after OTUD4 knockdown. Arsenite-treated cells (as in A) were stained with anti-OTUD4 and anti-TIAR antibodies. Scale bar: 20 µm. (E,F) Quantification of granule number and area in 55–100 FLAG–OTUD4-expressing cells per condition was performed as above. Wild-type and C45A-mutated OTUD4 reverse defects in granule formation. Cells with exogenous OTUD4 expression above endogenous levels were omitted for the analysis to exclude overexpression artifacts. In B,C,E,F, the box represents the 25–75th percentiles, and the median is indicated. The whiskers show the 1–99th percentiles and outliers are indicated. *p<0.05, **p<0.01, ***p<0.001 (B,E, quasi-Poissonian regression analysis; C,F, gamma regression analysis). (G) Analysis of changes in core stress granule proteins and apoptotic markers by western blot. HeLa cells were transfected with OTUD4 siRNA or control siRNA with or without arsenite (30 min, 0.5 mM). Western blot shows that G3BP1 and TIAR levels are unaffected by OTUD4 depletion. Depletion of OTUD4 leads to activation (cleavage) of caspase-3, as detected with anti-cleaved caspase-3 (Asp175) antibody and increased amounts of cleaved PARP, both indicating induction of apoptosis upon loss of OTUD4. The experiment was repeated three times. In D–G, oligo 7 was used for knockdown. (H) Knockdown of OTUD4 enhances the sensitivity of SH-SY5Y cells. SH-SY5Y cells were transfected with siRNA against OTUD4 or control siRNA as indicated. Cells were treated with arsenite (30 min, 0.5 mM) 24 h after siRNA transfection or left untreated. Cells were lysed 6 h later and levels of OTUD4, cleaved caspase-3 and actin were analyzed by western blot. OTUD4-knockdown leads to increased caspase activation in response to arsenite treatment. The experiment was performed three times.

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