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ProductInformation

ANTI-PARP [Poly (ADP-ribose) Polymerase]
Developed in Rabbit,
Affinity Isolated Antibody

Product Number **P7605**

Product Description

Anti-PARP is developed in rabbit using a synthetic PARP N-terminal peptide (Ala-Glu-Ser-Ser-Asp-Lys-Leu-Tyr-Arg-Val-Glu-Tyr-Ala-Lys-Ser-Gly-Arg-Ala-Ser-Lys) conjugated to KLH with glutaraldehyde as immunogen. The peptide corresponds to amino acid residues 2-20 of human PARP with C-terminal added lysine. The antibody is affinity-purified using the immunizing peptide immobilized on agarose.

Anti-PARP recognizes an epitope located on the N-terminal part of PARP. This epitope is highly conserved in PARP proteins of bovine, mouse and human. The antibody reacts specifically with PARP by immunoblotting (band at 116 kD). An additional 85 kD band presumably due to a non-apoptotic cleavage product may be detected in some preparations. Staining of the PARP band is inhibited by the PARP peptide (amino acid residues 2-20 with C-terminal added lysine). The antibody reacts by immunofluorescent staining with nuclei of fixed cultured cells. Staining is preferentially localized at the nuclear periphery.

Poly (ADP-ribose) Polymerase (PARP, EC 2.4.2.30) is an abundant, zinc-dependent eukaryotic nuclear enzyme that specifically recognizes single or double strand DNA breaks produced by various genotoxic agents.[1-3] PARP is thus, a molecular nick sensor, that following binding to damaged DNA converts NAD to nicotinamide and branched polymers of various poly (ADP-ribose) on glutamate residues of a limited number of nuclear acceptor proteins, including PARP itself. The increased negative charge of modified PARP results in loss of interaction with DNA due to electrostatic repulsion. Access of DNA repair proteins to the damaged DNA is thereby facilitated. The poly (ADP-ribose) moiety is quickly degraded by a PARP-associated Poly (ADP-ribose) glycohydrolase. PARP is composed of an N-terminal DNA binding domain, a central regulatory automodification domain that accepts poly (ADP-ribose) and a C-terminal catalytic domain.

Also, PARP modification of nuclear proteins is involved in chromatin structure formation, the regulation of differentiation, proliferation, development, apoptosis, gene expression, response to heart and brain ischemia/reperfusion, and malignant transformation.[4]

Rapid activation of PARP may deplete NAD, slow glycolysis, electron transport and ATP formation and cause cell dysfunction and cell death. PARP contains a conserved proteinase recognition site (DEVD) a target for several caspases (e.g. Caspase 2, 3, 6, 7 and 9). Cleavage of PARP into fragments of 29kD and 85kD by caspase-3 is an early marker of apoptosis. Necrotic cleavage of PARP generates different fragments.[5]

Reagents

Anti-PARP is provided as a solution of affinity isolated antibody in 10 mM phosphate buffered saline, pH 7.4 containing 1% bovine serum albumin and 15 mM sodium azide (see MSDS)* as a preservative.

Precautions and Disclaimer

* Due to the sodium azide content a material safety sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazardous and safe handling practices.

Storage/Stability

For continuous use, store at 2-8°C for up to one month. For extended storage, freeze in working aliquots. Repeated freezing and thawing is not recommended. Storage in "frost-free" freezers is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discarded if not used within 12 hours.

Product Profile
A minimum working dilution of 1:200 is determined by immunoblotting using a whole cell extract of MCF-7 human mammary adenocarcinoma cells.

A minimum working dilution of 1:100 is determined by indirect immunofluorescent staining of cultured MCF-7 cells.

Note: In order to obtain best results and assay sensitivity in different techniques and preparations we recommend determining optimal working dilutions by titration test.

References

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