MONOCLONAL ANTI-RAD1 DNA REPAIR PROTEIN CLONE 33
Purified Mouse Immunoglobulin

Product Number **R 2402**

*Product*Information

Product Description

Monoclonal Anti-RAD1 DNA Repair Protein (mouse IgG1 isotype) is derived from the hybridoma produced by the fusion of mouse myeloma Sp2/0 cells and splenocytes from BALB/c mice immunized with recombinant full length human RAD1 protein.

Monoclonal Anti- RAD1 DNA Repair Protein recognizes human RAD1 (approximately 31 kDa). It has been used in immunoblotting and immunoprecipitation applications. The antibody is purified by protein A chromatography.

Eukaryotic cells actively block entry into mitosis in the presence of DNA damage or incompletely replicated DNA. Signal transduction cascades called cell cycle checkpoints mediate this response. Six checkpoint RAD proteins are required for all *Schizosaccharomyces pombe* DNA integrity checkpoints. They include HUS1, RAD1, RAD3, RAD9, RAD17 and RAD26. These genes regulate checkpoint protein kinases Chk1 and Cds1. Chk1 is required to prevent mitosis when DNA is damaged by ionizing radiation (IR), whereas either kinase is sufficient to prevent mitosis when DNA replication is inhibited by hydroxyurea (HU). Checkpoint RAD proteins are required for IR-induced phosphorylation of Chk1 and HU-induced activation of Cds1.[1]

Human RAD1 mRNA (5, 3, and 1.3 Kb) is expressed in a variety of human tissues, with higher levels present in some cancer cell lines. The cDNAs encode the 282-amino acid RAD1 isoform, which is 90% and 27% identical to mouse RAD1 and *S. pombe* RAD1+, respectively. The human RAD1 locus has been mapped to human chromosome 5p13.2, a locus frequently altered in non-small-cell lung cancer and bladder cancer.[2,3]

The human checkpoint control protein hRAD9 physically associates with two other checkpoint control proteins, hRAD1 and hHUS1. *In vivo*, the human RAD9 protein is phosphorylated in response to DNA damage, suggesting that it participates in a DNA damage-inducible signaling pathway. Furthermore, hRAD1 and hHUS1 interact analogously to their fission yeast homologues RAD1 and HUS1. These three proteins are central components of a DNA damage-responsive protein complex in human cells.
To maintain genomic stability following DNA damage, multicellular organisms activate checkpoints that induce cell cycle arrest or apoptosis. HUS1p interacts with itself and with the N-terminal region of RAD1p. In contrast, the C-terminal portion of the checkpoint protein RAD9p is essential for interacting with HUS1p and the C-terminal region of RAD1p. Since the N-terminal portion of RAD9p has been demonstrated to participate in apoptosis, this protein likely has at least two functional domains, one that regulates programmed cell death and another that regulates cell cycle checkpoint control. [4-6]

Reagent

Monoclonal Anti-RAD1 DNA Repair Protein is supplied as a solution in phosphate buffered saline, pH 7.4, with 0.08% sodium azide as a preservative.

Precautions and Disclaimer

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

Storage/Stability

Store at –20 °C. Upon initial thawing freeze the solution in working aliquots for extended storage. Avoid repeated freezing and thawing to prevent denaturing the antibody. Do not store in a frost-free freezer. The antibody is stable for at least 12 months when stored appropriately. Working dilutions should be discarded if not used within 12 hours.

Product Profile

A recommended working concentration of 1 µg/ml is determined by immunoblotting and immunoprecipitation using MCF–7 breast cancer cells or human tonsil tissue. The data demonstrate that only tissues containing RAD1 protein stain positively, which confirms the specificity of Anti-RAD1 DNA Repair antibody for RAD1 protein.

Note: In order to obtain best results using different techniques and preparations we recommend determining optimal working concentration by titration.

References

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