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Performance guaranteed

ATM Polyclonal Antibody

Catalog Number **PA1-33020**

Product data sheet

Details

Size	50 µL
Host/Isotope	Rabbit / IgG
Class	Polyclonal
Type	Antibody
Immunogen	Recombinant human ATM fragment (amino acids 1-308).
Conjugate	Unconjugated
Form	Liquid
Concentration	Conc. Not Determined
Storage Conditions	Store at 4°C short term. For long term storage, store at -20°C, avoiding freeze/thaw cycles.

Species Reactivity

Species reactivity	Hamster, Human, Mouse, Non-human primate

Tested Applications	Dilution *
Western Blot (WB)	1/100-1/200

* Suggested working dilutions are given as a guide only. It is recommended that the user titrate the product for use in their own experiment using appropriate negative and positive controls.

Product specific information

Store product as a concentrated solution. Centrifuge briefly prior to opening the vial.

Background/Target Information

Ataxia-telangiectasia Mutated (ATM) is a protein that belongs to the PI3/PI4 kinase family. Ataxia-telangiectasia is a rare autosomal recessive disorder characterized by progressive neurologic degeneration, immunologic deficiency, and an increased risk of lymphoid cancer. The ATM gene codes for a protein belonging to the phosphoinositide 3-kinase (PI3K) superfamily. ATM phosphorylates proteins instead of lipid and has many downstream targets that act as cell-cycle regulators including: P53, Mdm2, BRCA1, and SMC1. The ATM protein is responsible for repairing double-stranded DNA breaks that occur because of ionizing radiation and other mutagens. The ATM's C-terminal region has extensive homology to the catalytic domains of phosphatidylinositol 3-kinases (PI3 kinases). Studies have shown that ATM becomes autophosphorylated and upregulated by exposure to ionizing radiation. AT cells are hypersensitive to ionizing radiation, impaired in mediating the inhibition of DNA synthesis and display delays in p53 induction. Further, DNA damage caused by ionizing irradiation activates ATM-kinase, leading to a cascade of kinase reactions that regulate cell cycle, apoptosis, and DNA damage repair. Studies have linked ATM to apoptosis along with Nbs1 and Chk2 in the E2F1 pathway.

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