MONOCULTURAL ANTI-ATM
CLONE ATX08
Purified Mouse Immunoglobulin

Product Number A 4225

Product Description
Monoclonal Anti-ATM (Ataxia-Telengiectasia Mutated), is derived from the hybridoma produced by the fusion of splenocytes from BALB/c mice immunized with recombinant human ATM protein and myeloma NS1 cells. Monoclonal Anti-ATM, a mouse IgG1 isotype, recognizes human and mouse ATM protein (350 kDa), epitope amino acids 5055 – 6585. It has been used in immunohistology with formalin-fixed paraffin-embedded tissue sections. It is not suitable for western blotting.

Ataxia-telangiectasia (A-T) is an autosomal recessive disorder characterized by cerebellar ataxia (loss of coordination), telangiectases (abnormal dilatations of capillary vessels and arterioles often forming angiomas), immune defects, and a predisposition to malignancy. The responsible gene, ATM, encodes a 370 kDa protein kinase that belongs to a family of protein kinases sharing similarities at their C-terminal region with the catalytic domain of phosphatidylinositol 3-kinases. These enzymes function in maintenance of genome stability, in cell cycle control, and in cellular responses to DNA damage.¹ ³

Studies with Ataxia-Telangiectasia (A-T) cells and ATM-deficient mice have shown that ATM is a key regulator of multiple signaling cascades which respond to DNA strand breaks induced by damaging agents or by normal processes. The signaling involves the activation of cell cycle checkpoints, DNA repair and apoptosis. Other roles outside the cell nucleus might be carried out by the cytoplasmic fraction of ATM. In addition, ATM appears to function as a 'caretaker', suppressing tumorigenesis in specific T cell lineages. In eukaryotes, the checkpoint pathway initiated by DNA damage consists of several protein kinases, including the phosphoinositide kinase (PIK) homologs ATM, ATR, Mec1, and Rad3 and the protein kinases Rad53, Cds1, Chk1, and Dun1. In mammals, in response to DNA damage and possibly to replication blocks, ATM activates p53 to control G₁ arrest and activates Chk2, and possibly Chk1, which in turn phosphorylate Cdc25C on Ser 216, leading to inhibition of Cdc2/cyclin B complexes.⁴ ⁵

Recombinant ATM protein phosphorylates p53 on serine 15 near the N-terminus. Ectopic expression of ATM in A-T cells restores normal ionizing radiation (IR)-induced phosphorylation of p53 on serine 15. Thus, ATM can bind p53 directly and is responsible for its serine 15 phosphorylation, contributing to the activation and stabilization of p53 during the IR-induced DNA damage response.⁶

Reagent
Monoclonal Anti-ATM is supplied as a solution in phosphate buffered saline, pH 7.4, with 0.08% sodium azide as a preservative. Each vial contains approximately 100 µg of antibody in 100 µl.

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 2 to 4 µg/ml is determined by immunohistochemical staining using ATM antibody on formalin-fixed, paraffin-embedded human breast carcinoma tissue. The data demonstrate that only tissues containing ATM protein stain positively, confirming the specificity of Anti-ATM for this protein. CoLo32 melanoma cells or breast carcinoma cells may be used as a positive control.

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


