SANTA CRUZ BIOTECHNOLOGY, INC.

Atm (2830-3056): sc-4456 WB

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The phosphatidylinositol kinase (PIK) family members fall into two distinct subgroups. The first subgroup contains proteins such as the PI 3- and PI 4-kinases and the second group comprises the PIK-related kinases. The PIK-related kinases include Atm, PKcs and FRAP. These proteins have in common a region of homology at their carboxy termini that is not present in the PI 3- and PI 4-kinases. All of the members of the PIK-related kinases are also more than 270 kDa. The Atm gene is mutated in the autosomal recessive disorder ataxia telangiectasia (AT), that is characterized by cerebellar degeneration (ataxia) and the appearance of dilated blood vessels (telangiectases) in the conjunctivae of the eyes. AT cells are hypersensitive to ionizing radiation, impaired in mediating the inhibition of DNA synthesis and display delays in p53 induction. DNA-PK is a heterotrimeric DNA binding enzyme that is composed of a large subunit, , and two smaller subunits collectively known as Ku. The loss of DNA-PK leads to defects in DSB repair and V(D)J recombination. FRAP can autophosphorylate on serine and bind to rapamycin/FKBP. FRAP is also an upstream regulator of S6 kinase and has been implicated in the regulation of p27 and p21 expression.

REFERENCES

1. Nowak, R. 1995. Discovery of AT gene sparks biomedical research bonanza. Science 268: 1700-1701.
2. Savitsky, K., Bar-Shira, A., Gilad, S., Rotman, G., Ziv, Y., Vanagaite, L., Tagle, D.A., Smith, S., Uziel, T., Sfez, S., Ashkenazi, M., Pecker, I., Frydman, M., Harnik, R., Patanjali, S.R., Simmons, A., Clines, G.A., Sartiel, A., Gatti, R.A., et al. 1995. A single ataxia telangiectasia gene with a product similar to PI-3 kinase. Science 268: 1749-1753.
3. Keith, C.T. and Schreiber, S.L. 1995. PIK-related kinases: DNA repair, recombination, and cell cycle checkpoints. Science 270: 50-51.
4. Hartley, K.O., Gell, D., Smith, G.C.M., Zhang, H., Divecha, N.Connelly, M.A., Admon, A., Lees-Miller, S.P., Anderson, C.W., and Jackson, S.P. 1995. DNA-dependent protein kinase catalytic subunit: a relative of phosphatidylinositol 3-kinase and the ataxia telangiectasia gene product. Cell 82: 849-856.
5. Hunter, T. 1995. When is a lipid kinase not a lipid kinase? When it is a protein kinase. Cell 83: 1-4.
6. Hickson, I., et al. 2004. Identification and characterization of a novel and specific inhibitor of the ataxia-telangiectasia mutated kinase Atm. Cancer Res. 64:9152-9159.
7. Chen, L., et al. 2005. Ataxia-telangiectasia-mutated dependent phosphorylation of Artemis in response to DNA damage. Cancer Sci. 96:134-141.
8. Liu, A., et al. 2005. Alterations of DNA damage-response genes Atm and ATR in pyothorax-associated lymphoma. Lab Invest. 85:436-446.
9. Lavin, M.F., et al. 2005. Atm signaling and genomic stability in response to DNA damage. Mutat Res. 569: 123-132.

SOURCE

Atm (2830-3056) is expressed in *E. coli* as a 52 kDa tagged fusion protein corresponding to amino acids 2830-3056 of Atm of human origin.

PRODUCT

Atm (2830-3056) is purified from bacterial lysates (>98%) by glutathione agarose affinity chromatography; supplied as 10 µg in 0.1 ml SDS-PAGE loading buffer.

APPLICATIONS

Atm (2830-3056) is suitable as a Western blotting control for sc-1212, sc-1214, sc-7128, sc-7230 and sc-8434.

STORAGE

Store at -20° C; stable for one year from the date of shipment.

RESEARCH USE

For research use only, not for use in diagnostic procedures.