SANTA CRUZ BIOTECHNOLOGY, INC.

p-DNA-PK$_{CS}$ (Thr 2609): sc-101664

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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, DNA-PK$_{CS}$ 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. 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 they display delays in p53 induction. DNA-PK is a heterotrimeric DNA binding enzyme that is composed of a large subunit, DNA-PK$_{CS}$, 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., et al. 1995. A single ataxia telangiectasia gene with a product similar to PI-3 kinase. Science 268: 1749-1753.
3. Keith, C.T., et al. 1995. PIK-related kinases: DNA repair, recombination, and cell cycle checkpoints. Science 270: 50-51.
4. Hartley, K.O., et al. 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. Yan, Y.Q., et al. 2007. Induction of apoptosis and autophagic cell death by the vanillin derivative 6-bromine-5-hydroxy-4-methoxybenzaldehyde is accompanied by the cleavage of DNA-PK$_{CS}$ and rapid destruction of c-Myc oncoprotein in HepG2 cells. Cancer Lett. 252: 280-289.
7. Kuhfittig-Kulle, S., et al. 2007. The mutagenic potential of non-homologous end joining in the absence of the NHEJ core factors Ku-70/80, DNA-PK$_{CS}$ and XRCC4-LigIV. Mutagenesis 22: 217-233.
8. Chai, Y.H., et al. 2007. Expression of PTEN mRNA, hTERT and DNA-PK$_{CS}$ in bone marrow cells of children with acute leukemia. Zhonghua Er Ke Za Zhi 45: 74-75.
9. Kanasugi, Y., et al. 2007. Role of DNA-PK$_{CS}$ in the bystander effect after low- or high-LET irradiation. Int. J. Radiat. Biol. 83: 73-80.

CHROMOSOMAL LOCATION

Genetic locus: PRKDC (human) mapping to 8q11.21.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

SOURCE

p-DNA-PK$_{CS}$ (Thr 2609) is a rabbit polyclonal antibody raised against a short amino acid sequence containing Thr 2609 phosphorylated DNA-PK$_{CS}$ of human origin.

PRODUCT

Each vial contains 100 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

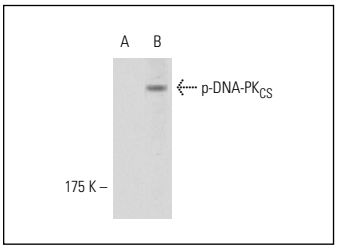
p-DNA-PK$_{CS}$ (Thr 2609) is recommended for detection of Thr 2609 phosphorylated DNA-PK$_{CS}$ of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) and immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)].

Suitable for use as control antibody for DNA-PK$_{CS}$ siRNA (h): sc-35200, DNA-PK$_{CS}$ shRNA Plasmid (h): sc-35200-SH and DNA-PK$_{CS}$ shRNA (h) Lentiviral Particles: sc-35200-V.

Molecular Weight of p-DNA-PK$_{CS}$: 460 kDa.

Positive Controls: K-562 + hydroxyurea whole cell lysate.

DATA



p-DNA-PK$_{CS}$ (Thr 2609): sc-101664. Western blot analysis of phosphorylated DNA-PK$_{CS}$ expression in untreated (**A**) and hydroxyurea-treated (**B**) K-562 whole cell lysates.

SELECT PRODUCT CITATIONS

1. Huwiler, A., et al. 2011. Loss of sphingosine kinase-1 in carcinoma cells increases formation of reactive oxygen species and sensitivity to doxorubicin-induced DNA damage. Br. J. Pharmacol. 162: 532-543.
2. Gravina, G.L., et al. 2014. Torc1/Torc2 inhibitor, Palomid 529, enhances radiation response modulating CRM1-mediated survivin function and delaying DNA repair in prostate cancer models. Prostate 74: 852-868.

RESEARCH USE

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

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.