SANTA CRUZ BIOTECHNOLOGY, INC.

FRAP (N-19): sc-1549

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The PIK-related kinases include Atm, DNA-PK_{CS} and mTOR. The Atm gene is mutated in the autosomal recessive disorder ataxia telangiectasia (AT) that is characterized by cerebellar degeneration and the appearance of dilated blood vessels 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. mTOR, also known as FRAP, can autophosphorylate on serine and bind to rapamycin/FKBP. mTOR is also an upstream regulator of S6 kinase and has been implicated in the regulation of p27 and p21 expression. mTOR autophosphorylates at Ser2481 under translationally repressive conditions. Phosphorylation of mTOR at Ser2448 is mediated by p70S6 kinase.

CHROMOSOMAL LOCATION

Genetic locus: MTOR (human) mapping to 1p36.22.

SOURCE

FRAP (N-19) is available as either goat (sc-1549) or rabbit (sc-1549-R) polyclonal affinity purified antibody raised against a peptide mapping at the N-terminus of FRAP of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-1549 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

FRAP (N-19) is recommended for detection of FRAP of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500).

Suitable for use as control antibody for FRAP siRNA (h): sc-35409, FRAP shRNA Plasmid (h): sc-35409-SH and FRAP shRNA (h) Lentiviral Particles: sc-35409-V.

Molecular Weight (predicted) of FRAP: 289 kDa.

Molecular Weight (observed) of FRAP: 211-245 kDa.

Positive Controls: K-562 whole cell lysate: sc-2203, HeLa whole cell lysate: sc-2200 or MOLT-4 cell lysate: sc-2233.

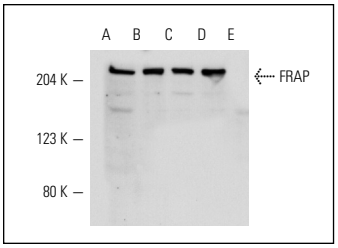
STORAGE

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

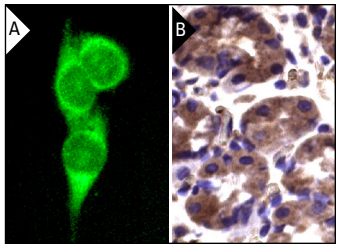
RESEARCH USE

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

DATA



FRAP (N-19): sc-1549. Western blot analysis of FRAP expression in HeLa (**A**), K-562 (**B**), Jurkat (**C**), MOLT-4 (**D**) and CTLL-2 (**E**) whole cell lysates. Note lack of reactivity with mouse FRAP in CTLL-2 cells.



FRAP (N-19): sc-1549. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic staining (**A**). Immunoperoxidase staining of formalin fixed, paraffin-embedded human stomach tissue showing cytoplasmic staining of glandular cells (**B**).

SELECT PRODUCT CITATIONS

1. Kim, D.H., et al. 2002. mTOR interacts with raptor to form a nutrient-sensitive complex that signals to the cell growth machinery. Cell 110: 163-175.
2. Beevers, C.S., et al. 2009. Curcumin disrupts the mammalian target of Rapamycin-Raptor complex. Cancer Res. 69: 1000-1008.
3. Yu, K., et al. 2009. Biochemical, cellular, and *in vivo* activity of novel ATP-competitive and selective inhibitors of the mammalian target of rapamycin. Cancer Res. 69: 6232-6240.
4. Dibble, C.C., et al. 2009. Characterization of Rictor phosphorylation sites reveals direct regulation of mTOR complex 2 by S6K1. Mol. Cell. Biol. 29: 5657-5670.
5. Pearce, L.R., et al. 2010. Characterization of PF-4708671, a novel and highly specific inhibitor of p70 ribosomal S6 kinase (S6K1). Biochem. J. 431: 245-255.
6. Bandhakavi, S., et al. 2010. Quantitative nuclear proteomics identifies mTOR regulation of DNA damage response. Mol. Cell Proteomics 9: 403-414.
7. Shor, B., et al. 2010. Requirement of the mTOR kinase for the regulation of Maf1 phosphorylation and control of RNA polymerase III-dependent transcription in cancer cells. J. Biol. Chem. 285: 15380-15392.
8. Javle, M.M., et al. 2010. Inhibition of the mammalian target of rapamycin (mTOR) in advanced pancreatic cancer: results of two phase II studies. BMC Cancer 10: 368.