SANTA CRUZ BIOTECHNOLOGY, INC.

GADD 45α (H-165): sc-797

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

It is well established that cell cycle progression is subject to arrest at G_1 and G_2 checkpoints in response to DNA damage, presumably to allow time for DNA repair prior to entry into S and M phase, respectively. The p53 tumor suppressor is required for one such G_1 checkpoint and functions to upregulate expression of GADD 45 and p21. p21 functions to inhibit the kinase activity of multiple Cdk complexes, which may account for its suppression of cell growth. GADD 45 binds both Cdks and PCNA, a protein involved in DNA replication and repair. GADD 45 has been shown to stimulate DNA excision repair *in vitro* and to inhibit entry of cells into S phase. Thus, it has been suggested that GADD 45 may serve as a link between p53-dependent cell cycle checkpoint and DNA repair.

REFERENCES

1. Murray, A.W. 1992. Creative blocks: cell-cycle checkpoints and feedback controls. Nature 359: 599-604.
2. Kuerbitz, S.J., et al. 1992. Wild-type p53 is a cell cycle checkpoint determinant following irradiation. Proc. Natl. Acad. Sci. USA 89: 7491-7495.

SOURCE

GADD 45α (H-165) is a rabbit polyclonal antibody raised against amino acids 1-165 representing full length GADD 45α 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.

Available as TransCruz reagent for ChIP application, sc-797 X, 200 µg/0.1 ml.

APPLICATIONS

GADD 45α (H-165) is recommended for detection of GADD 45α and, to a lesser extent, GADD 45β and GADD 45γ of mouse, rat and 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 solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

GADD 45α (H-165) is also recommended for detection of GADD 45α and, to a lesser extent, GADD 45β and GADD 45γ in additional species, including bovine and porcine.

GADD 45α (H-165) X TransCruz antibody is recommended for ChIP assays.

Molecular Weight of GADD 45α: 18 kDa.

Positive Controls: K-562 whole cell lysate: sc-2203 or GADD 45α (h2): 293 Lysate: sc-175023.

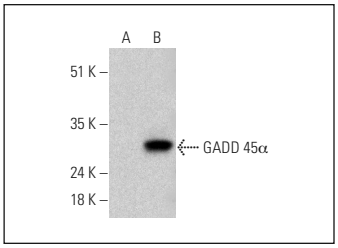
STORAGE

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

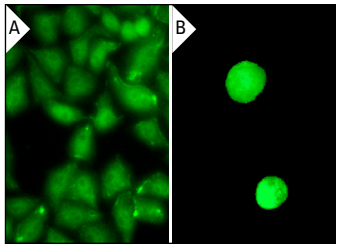
RESEARCH USE

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

DATA



GADD 45α (H-165): sc-797. Western blot analysis of GADD 45α expression in non-transfected: sc-110760 (**A**) and human GADD 45α transfected: sc-175023 (**B**) 293 whole cell lysates.



GADD 45α (H-165): sc-797. Immunofluorescence staining of formalin-fixed HeLa cells showing nuclear and cytoplasmic localization. Kindly provided by Yang Xiang, Ph.D., Division of Newborn Medicine, Boston Children's Hospital, Cell Biology Department, Harvard Medical School (**A**). Immunofluorescence staining of methanol-fixed GADD 45α-transfected COS cells showing nuclear localization (**B**).

SELECT PRODUCT CITATIONS

1. Vairapandi, M., et al. 2000. Characterization of MyD118, GADD 45, and proliferating cell nuclear antigen (PCNA) interacting domains. J. Biol. Chem. 275: 16810-16819.
2. March, M.E., et al. 2000. p135 src homology 2 domain-containing inositol 5'-phosphatase (SHIPβ) isoform can substitute for p145 SHIP in fcγ RIIB1-mediated inhibitory signaling in B cells. J. Biol. Chem. 275: 29960-29967.
3. Wagner, M.W., et al. 2008. Role of c-Abl kinase in DNA mismatch repair-dependent G_2 cell cycle checkpoint arrest responses. J. Biol. Chem. 283: 21382-21393.
4. Shang, L., et al. 2000. Functional link of BRCA1 and ataxia telangiectasia gene product in DNA damage response. Nature 6792: 210-215.
5. Son, Y.O., et al. 2010. Cadmium induces intracellular Ca^{2+}- and H_2O_2-dependent apoptosis through JNK- and p53-mediated pathways in skin epidermal cell line. Toxicol. Sci. 113: 127-137.
6. Watt, H.L., et al. 2010. Receptor activation and inhibition in cellular response to chemotherapeutic combinational mimicries: the concept of divergent targeting. J. Neurooncol. 100: 345-361.
7. Wheaton, K., et al. 2010. BTG2 antagonizes Pin1 in response to mitogens and telomere disruption during replicative senescence. Aging Cell 9: 747-760.
8. Saletta, F., et al. 2011. Cellular iron depletion and the mechanisms involved in the iron-dependent regulation of the growth arrest and DNA damage family of genes. J. Biol. Chem. 286: 35396-35406.
9. Song, L., et al. 2011. p85α mediates p53 K370 acetylation by p300 and regulates its promoter-specific transactivity in the cellular UVB response. Oncogene 30: 1360-1371.
10. Xia, Y., et al. 2011. Dose-dependent mutual regulation between Wip1 and p53 following UVC irradiation. Int. J. Biochem. Cell Biol. 43: 535-544.