SANTA CRUZ BIOTECHNOLOGY, INC.

GADD 34 (C-19): sc-825

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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 the mitotic inhibitory protein p21. GADD 45 has been shown to stimulate DNA excision repair *in vitro* and to inhibit entry of cells into S phase, and it apparently acts in concert with GADD 153 in inducing growth arrest. A related DNA-damage inducible gene, GADD 34 (also designated MyD116) has been shown to synergize with GADD 45 or GADD 153 in suppressing cell growth. PEG-3 (progression elevated gene-3) shares significant homology with GADD 34 and is inducible by DNA damage. PEG-3 expression has been shown to be elevated in cells displaying a progressed-transformed phenotype.

REFERENCES

1. Sherr, C.J. 1994. G_1 phase progression: cycling on cue. Cell 79: 551-555.
2. Hunter, T., et al. 1994. Cyclins and cancer II: cyclin D and CDK inhibitors come of age. Cell 79: 573-582.
3. Ron, D. 1994. Inducible growth arrest: new mechanistic insights. Proc. Natl. Acad. Sci. USA 91: 1985-1986.
4. Smith, M.L., et al. 1994. Interaction of the p53-regulated protein GADD 45 with proliferating cell nuclear antigen. Science 266: 1376-1380.
5. Gujuluva, C.N., et al. 1994. Effect of UV-irradiation on cell cycle, viability and the expression of p53, GADD 153 and GADD 45 genes in normal and HPV-immortalized human oral keratinocytes. Oncogene 9: 1819-1827.

CHROMOSOMAL LOCATION

Genetic locus: Myd116 (mouse) mapping to 7 B4.

SOURCE

GADD 34 (C-19) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the C-terminus of GADD 34 of mouse 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-825 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

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.

APPLICATIONS

GADD 34 (C-19) is recommended for detection of GADD 34 of mouse and rat 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).

Suitable for use as control antibody for GADD 34 siRNA (m): sc-37415, GADD 34 shRNA Plasmid (m): sc-37415-SH and GADD 34 shRNA (m) Lentiviral Particles: sc-37415-V.

Molecular Weight of GADD 34: 73 kDa.

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use goat anti-rabbit IgG-HRP: sc-2004 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible goat anti-rabbit IgG-HRP: sc-2030 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto A Blocking Reagent: sc-2333 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/ 2.0 ml). 3) Immunofluorescence: use goat anti-rabbit IgG-FITC: sc-2012 (dilution range: 1:100-1:400) or goat anti-rabbit IgG-TR: sc-2780 (dilution range: 1:100-1:400) with UltraCruz™ Mounting Medium: sc-24941.

SELECT PRODUCT CITATIONS

1. McCaig, D., et al. 2005. Evolution of GADD 34 expression after focal cerebral ischaemia. Brain Res. 1034: 51-61.
2. Patterson, A.D., et al. 2006. GADD 34 requirement for normal hemoglobin synthesis. Mol. Cell. Biol. 26: 1644-1653.
3. Wei, Y., et al. 2006. Saturated fatty acids induce endoplasmic reticulum stress and apoptosis independently of ceramide in liver cells. Am. J. Physiol. Endocrinol. Metab. 291: E275-E281.
4. Yusta, B., et al. 2006. GLP-1 receptor activation improves β cell function and survival following induction of endoplasmic reticulum stress. Cell Metab. 4: 391-406.
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7. Wang, M., et al. 2010. Essential role of the unfolded protein response regulator GRP78/BiP in protection from neuronal apoptosis. Cell Death Differ. 17: 488-498.
8. de Ridder, G., et al. 2011. Modulation of the unfolded protein response by GRP78 in prostate cancer. Meth. Enzymol. 489: 245-257.