SANTA CRUZ BIOTECHNOLOGY, INC.

GLTPD2 (V-18): sc-102011

BACKGROUND

Chromosome 17 makes up over 2.5% of the human genome with about 81 million bases encoding over 1,200 genes. Two key tumor suppressor genes are associated with chromosome 17, namely, p53 and BRCA1. Tumor suppressor p53 is necessary for maintenance of cellular genetic integrity by moderating cell fate through DNA repair versus cell death. Malfunction or loss of p53 expression is associated with malignant cell growth and Li-Fraumeni syndrome. Like p53, BRCA1 is directly involved in DNA repair, though specifically it is recognized as a genetic determinant of early onset breast cancer and predisposition to cancers of the ovary, colon, prostate gland and fallopian tubes. Chromosome 17 is also linked to neurofibromatosis, a condition characterized by neural and epidermal lesions, and dysregulated Schwann cell growth. Alexander disease, Birt-Hogg-Dubé syndrome and Canavan disease are also associated with chromosome 17.

REFERENCES

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2. Nusbaum, R., et al. 2006-2007. Susceptibility to breast cancer: hereditary syndromes and low penetrance genes. Breast Dis. 27: 21-50.
3. Al-Dirbashi, O.Y., et al. 2007. Quantification of N-acetylaspartic acid in urine by LC-MS/MS for the diagnosis of Canavan disease. J. Inherit. Metab. Dis. 30: 612.
4. Dann, R.B., et al. 2007. Strategies for ovarian cancer prevention. Obstet. Gynecol. Clin. North Am. 34: 667-686.
5. Farrell, C.J. and Plotkin, S.R. 2007. Genetic causes of brain tumors: neurofibromatosis, tuberous sclerosis, von Hippel-Lindau, and other syndromes. Neurol. Clin. 25: 925-946.
6. Suela, J., et al. 2007. Neurofibromatosis 1, and Not TP53, seems to be the main target of chromosome 17 deletions in *de novo* acute myeloid leukemia. J. Clin. Oncol. 25: 1151-1152.
7. Tai, Y.C., et al. 2007. Breast cancer risk among male BRCA1 and BRCA2 mutation carriers. J. Natl. Cancer Inst. 99: 1811-1814.
8. Yan, J., et al. 2007. BLIMP1 regulates cell growth through repression of p53 transcription. Proc. Natl. Acad. Sci. USA 104: 1841-1846.
9. Murakami, N., et al. 2008. Novel deletion mutation in GFAP gene in an infantile form of Alexander disease. Pediatr. Neurol. 38: 50-52.

CHROMOSOMAL LOCATION

Genetic locus: GLTPD2 (human) mapping to 17p13.2.

STORAGE

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

PROTOCOLS

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

SOURCE

GLTPD2 (V-18) is a purified rabbit polyclonal antibody raised against GLTPD2 of human origin.

PRODUCT

Each vial contains 100 µg IgG in 1.0 ml PBS with < 0.1% sodium azide, 0.1% gelatin and < 0.02% sucrose.

APPLICATIONS

GLTPD2 (V-18) is recommended for detection of GLTPD2 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)] and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for GLTPD2 siRNA (h): sc-93721, GLTPD2 shRNA Plasmid (h): sc-93721-SH and GLTPD2 shRNA (h) Lentiviral Particles: sc-93721-V.

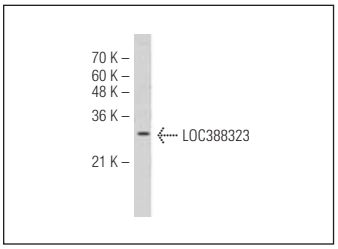
Molecular Weight of GLTPD2: 32 kDa.

Positive Controls: Jurkat whole cell lysate: sc-2204.

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).

DATA



LOC388323 (V-18): sc-102011. Western blot analysis of LOC388323 expression in Jurkat whole cell lysate.

RESEARCH USE

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