

GID4 (m): 293T Lysate: sc-124887

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-Dube syndrome and Canavan disease are also associated with chromosome 17.

REFERENCES

1. Welsch, M.J., et al. 2005. Birt-Hogg-Dube Syndrome. *Int. J. Dermatol.* 44: 668-673.
2. Nusbaum, R., et al. 2006-2007. Susceptibility to breast cancer: hereditary syndromes and low penetrance genes. *Breast Dis.* 27: 21-50.
3. Al-Dibbashi, 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: *Gid4* (mouse) mapping to 11 B2.

PRODUCT

GID4 (m): 293T Lysate represents a lysate of mouse GID4 transfected 293T cells and is provided as 100 µg protein in 200 µl SDS-PAGE buffer.

STORAGE

Store at -20° C. Repeated freezing and thawing should be minimized. Sample vial should be boiled once prior to use. Non-hazardous. No MSDS required.

APPLICATIONS

GID4 (m): 293T Lysate is suitable as a Western Blotting positive control for mouse reactive CCDC198 antibodies. Recommended use: 10-20 µl per lane.

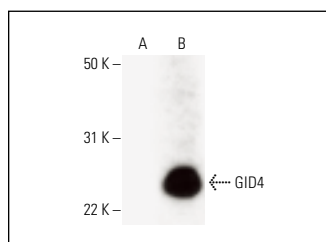
Control 293T Lysate: sc-117752 is available as a Western Blotting negative control lysate derived from non-transfected 293T cells.

GID4 (B-6): sc-390887 is recommended as a positive control antibody for Western Blot analysis of enhanced mouse GID4 expression in GID4 transfected 293T cells (starting dilution 1:100, dilution range 1:100-1:1,000).

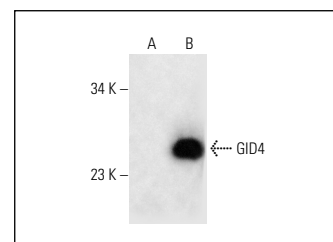
RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended:
1) Western Blotting: use m-IgGκ BP-HRP: sc-516102 or m-IgGκ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz Marker™ Molecular Weight Standards: sc-2035, UltraCruz® Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048.

DATA



GID4 (B-6): sc-390887. Western blot analysis of GID4 expression in non-transfected: sc-117752 (A) and mouse GID4 transfected: sc-124887 (B) 293T whole cell lysates.



GID4 (G-10): sc-390559. Western blot analysis of GID4 expression in non-transfected: sc-117752 (A) and mouse GID4 transfected: sc-124887 (B) 293T whole cell lysates.

RESEARCH USE

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

PROTOCOLS

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