

Svs7 (K-14): sc-246904

BACKGROUND

With approximately 135 million base pairs and 1,400 genes, chromosome 11 makes up around 4% of human genomic DNA and is considered a gene and disease association-dense chromosome. The chromosome 11-encoded *Atm* gene is important for regulation of cell cycle arrest and apoptosis following double strand DNA breaks. *Atm* mutation leads to the disorder known as ataxia-telangiectasia. The blood disorders Sickle cell anemia and β thalassemia are caused by HBB gene mutations. Wilms' tumors, WAGR syndrome and Denys-Drash syndrome are associated with mutations of the *WT1* gene. Jervell and Lange-Nielsen syndrome, Jacobsen syndrome, Niemann-Pick disease, hereditary angioedema and Smith-Lemli-Opitz syndrome are also associated with defects in chromosome 11. The FLJ41047 gene product has been provisionally designated FLJ41047 pending further characterization.

REFERENCES

1. Grossfeld, P.D., et al. 2004. The 11q terminal deletion disorder: a prospective study of 110 cases. *Am. J. Med. Genet. A* 129: 51-61.
2. Lousouarn, G., et al. 2006. KCNQ1 K⁺ channel-mediated cardiac channelopathies. *Methods Mol. Biol.* 337: 167-183.
3. Taylor, T.D., et al. 2006. Human chromosome 11 DNA sequence and analysis including novel gene identification. *Nature* 440: 497-500.
4. Zehelein, J., et al. 2006. Skipping of exon 1 in the KCNQ1 gene causes Jervell and Lange-Nielsen syndrome. *J. Biol. Chem.* 281: 35397-35403.
5. Ataga, K.I., et al. 2007. β -thalassaemia and sickle cell anaemia as paradigms of hypercoagulability. *Br. J. Haematol.* 139: 3-13.
6. Berger, A.C., et al. 2007. The subcellular localization of the Niemann-Pick type C proteins depends on the adaptor complex AP-3. *J. Cell Sci.* 120: 3640-3652.
7. Lee, J.H. and Paull, T.T. 2007. Activation and regulation of ATM kinase activity in response to DNA double-strand breaks. *Oncogene* 26: 7741-7748.
8. O'Connor, M.J., et al. 2007. Targeted cancer therapies based on the inhibition of DNA strand break repair. *Oncogene* 26: 7816-7824.
9. Kaste, S.C., et al. 2008. Wilms' tumour: prognostic factors, staging, therapy and late effects. *Pediatr. Radiol.* 38: 2-17.

CHROMOSOMAL LOCATION

Genetic locus: *Svs7* (rat) mapping to 8q21.

SOURCE

Svs7 (K-14) is an affinity purified goat polyclonal antibody raised against a peptide mapping within an internal region of *Svs7* of rat origin.

PRODUCT

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

Blocking peptide available for competition studies, sc-246904 P, (100 μ g peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

Svs7 (K-14) is recommended for detection of *Svs7* of rat origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), 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).

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use donkey anti-goat IgG-HRP: sc-2020 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible donkey anti-goat IgG-HRP: sc-2033 (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) Immunofluorescence: use donkey anti-goat IgG-FITC: sc-2024 (dilution range: 1:100-1:400) or donkey anti-goat IgG-TR: sc-2783 (dilution range: 1:100-1:400) with UltraCruz™ Mounting Medium: sc-24941.

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.

PROTOCOLS

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