

# FUS/TLS (H-76): sc-25540

## BACKGROUND

EWS and FUS/TLS are nuclear RNA-binding proteins. As a result of chromosome translocation, the EWS gene is fused to a variety of transcription factors, including ATF-1, in human neoplasias. In the Ewing family of tumors, the N-terminal domain of EWS is fused to the DNA-binding domain of various Ets transcription factors, including Fli-1, ETV1 and FEV. The EWS/Fli-1 chimeric protein acts as a more potent transcriptional activator than Fli-1 and can promote cell transformation. In human myxoid liposarcomas and myeloid leukemias, chromosomal translocation results in the fusion of the N-terminal region of FUS/TLS with the open reading frame of CHOP. In normal cells, FUS/TLS binds to the DNA-binding domains of nuclear steroid receptors and is also present in subpopulations of TFIIID complexes, indicating a potential role for FUS/TLS in the processing of primary transcripts that are generated in response to hormone-induced transcription.

## REFERENCES

1. Delattre, O., et al. 1992. Gene fusion with an ETS DNA-binding domain caused by chromosome translocation in human tumours. *Nature* 359: 162-165.
2. May, W.A., et al. 1993. The Ewing's sarcoma EWS/FLI-1 fusion gene encodes a more potent transcriptional activator and is a more powerful transforming gene than FLI-1. *Mol. Cell. Biol.* 13: 7393-7398.
3. Crozat, A., et al. 1993. Fusion of CHOP to a novel RNA-binding protein in human myxoid liposarcoma. *Nature* 363: 640-644.
4. Jeon, I.S., et al. 1995. A variant Ewing's sarcoma translocation (7;22) fuses the EWS gene to the ETS gene ETV1. *Oncogene* 10: 1229-1234.
5. Fujimura, Y., et al. 1996. The EWS-ATF-1 gene involved in malignant melanoma of soft parts with t(12;22) chromosome translocation, encodes a constitutive transcriptional activator. *Oncogene* 12: 159-167.
6. Peter, M., et al. 1997. A new member of the ETS family fused to EWS in Ewing tumors. *Oncogene* 14: 1159-1164.
7. Powers, C.A., et al. 1998. TLS (translocated-in-liposarcoma) is a high-affinity interactor for steroid, thyroid hormone, and retinoid receptors. *Mol. Endocrinol.* 12: 4-18.

## CHROMOSOMAL LOCATION

Genetic locus: FUS (human) mapping to 16p11.2; Fus (mouse) mapping to 7 F3.

## SOURCE

FUS/TLS (H-76) is a rabbit polyclonal antibody raised against amino acids 451-526 of FUS/TLS 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.

## STORAGE

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

## APPLICATIONS

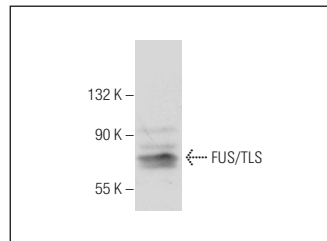
FUS/TLS (H-76) is recommended for detection of FUS/TLS of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for FUS/TLS siRNA (h): sc-40563, FUS/TLS siRNA (m): sc-40564, FUS/TLS shRNA Plasmid (h): sc-40563-SH, FUS/TLS shRNA Plasmid (m): sc-40564-SH, FUS/TLS shRNA (h) Lentiviral Particles: sc-40563-V and FUS/TLS shRNA (m) Lentiviral Particles: sc-40564-V.

Molecular Weight of FUS/TLS: 75 kDa.

Positive Controls: THP-1 cell lysate: sc-2238, NIH/3T3 whole cell lysate: sc-2210 or Jurkat whole cell lysate: sc-2204.

## DATA



FUS/TLS (H-76): sc-25540. Western blot analysis of FUS/TLS expression in THP-1 whole cell lysate.

## SELECT PRODUCT CITATIONS

1. Oikawa, K., et al. 2006. Generation of the novel monoclonal antibody against TLS/EWS-CHOP chimeric oncoproteins that is applicable to one of the most sensitive assays for myxoid and round cell liposarcomas. *Am. J. Surg. Pathol.* 30: 351-356.
2. Göransson, M., et al. 2009. The myxoid liposarcoma FUS-DDIT3 fusion oncoprotein deregulates NFκB target genes by interaction with NFKBIZ. *Oncogene* 28: 270-278.
3. Edelmann, M.J., et al. 2009. Structural basis and specificity of human otubain 1-mediated deubiquitination. *Biochem. J.* 418: 379-390.
4. Deng, H.X., et al. 2010. FUS-immunoreactive inclusions are a common feature in sporadic and non-SOD1 familial amyotrophic lateral sclerosis. *Ann. Neurol.* 67: 739-748.
5. Nishimura, A.L., et al. 2010. Nuclear import impairment causes cytoplasmic trans-activation response DNA-binding protein accumulation and is associated with frontotemporal lobar degeneration. *Brain* 133: 1763-1771.

## RESEARCH USE

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