



# Met siRNA (h): sc-29397

## BACKGROUND

The c-Met oncogene was originally isolated from a chemical carcinogen-treated human osteogenic sarcoma cell line by transfection analysis in NIH/3T3 cells. The Met proto-oncogene product was identified as a transmembrane receptor-like protein with tyrosine kinase activity that is expressed in many tissues. A high proportion of spontaneous NIH/3T3 transformants overexpress c-Met and by transfection analysis the c-Met proto-oncogene has been shown to exhibit transforming activity. Tyrosine phosphorylation of apparently normal Met protein has also been observed in certain human gastric carcinoma cell lines. Tyrosine phosphorylation enhances the receptor kinase activity, while serine phosphorylation of Met on residue 985 has an inhibitory effect. The c-Met gene product has been identified as the cell surface receptor for hepa-tocyte growth factor, a plasminogen-like protein thought to be a humoral mediator of liver regeneration.

## REFERENCES

1. Cooper, C.S., et al. 1984. Molecular cloning of a new transforming gene from a chemically transformed human cell line. *Nature* 311: 29-33.
2. Park, M., et al. 1986. Mechanism of Met oncogene activation. *Cell* 45: 895-904.
3. Cooper, C.S., et al. 1986. Amplification and overexpression of the Met gene in spontaneously transformed NIH/3T3 mouse fibroblasts. *EMBO J.* 5: 2623-2628.

## CHROMOSOMAL LOCATION

Genetic locus: MET (human) mapping to 7q31.2.

## PRODUCT

Met siRNA (h) is a pool of 3 target-specific 19-25 nt siRNAs designed to knock down gene expression. Each vial contains 3.3 nmol of lyophilized siRNA, sufficient for a 10  $\mu$ M solution once resuspended using protocol below. Suitable for 50-100 transfections. Also see Met shRNA Plasmid (h): sc-29397-SH and Met shRNA (h) Lentiviral Particles: sc-29397-V as alternate gene silencing products.

For independent verification of Met (h) gene silencing results, we also provide the individual siRNA duplex components. Each is available as 3.3 nmol of lyophilized siRNA. These include: sc-29397A, sc-29397B and sc-29397C.

## STORAGE AND RESUSPENSION

Store lyophilized siRNA duplex at -20° C with desiccant. Stable for at least one year from the date of shipment. Once resuspended, store at -20° C, avoid contact with RNases and repeated freeze thaw cycles.

Resuspend lyophilized siRNA duplex in 330  $\mu$ l of the RNase-free water provided. Resuspension of the siRNA duplex in 330  $\mu$ l of RNase-free water makes a 10  $\mu$ M solution in a 10  $\mu$ M Tris-HCl, pH 8.0, 20 mM NaCl, 1 mM EDTA buffered solution.

## APPLICATIONS

Met siRNA (h) is recommended for the inhibition of Met expression in human cells.

## SUPPORT REAGENTS

For optimal siRNA transfection efficiency, Santa Cruz Biotechnology's siRNA Transfection Reagent: sc-29528 (0.3 ml), siRNA Transfection Medium: sc-36868 (20 ml) and siRNA Dilution Buffer: sc-29527 (1.5 ml) are recommended. Control siRNAs or Fluorescein Conjugated Control siRNAs are available as 10  $\mu$ M in 66  $\mu$ l. Each contain a scrambled sequence that will not lead to the specific degradation of any known cellular mRNA. Fluorescein Conjugated Control siRNAs include: sc-36869, sc-44239, sc-44240 and sc-44241. Control siRNAs include: sc-37007, sc-44230, sc-44231, sc-44232, sc-44233, sc-44234, sc-44235, sc-44236, sc-44237 and sc-44238.

## GENE EXPRESSION MONITORING

Met (D-4): sc-514148 is recommended as a control antibody for monitoring of Met gene expression knockdown by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) or immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500).

## RT-PCR REAGENTS

Semi-quantitative RT-PCR may be performed to monitor Met gene expression knockdown using RT-PCR Primer: Met (h)-PR: sc-29397-PR (20  $\mu$ l, 566 bp). Annealing temperature for the primers should be 55-60° C and the extension temperature should be 68-72° C.

## SELECT PRODUCT CITATIONS

1. Mukhopadhyay, I., et al. 2006. Molecular mechanism of adaphostin-mediated G<sub>1</sub> arrest in prostate cancer (PC-3) cells: signaling events mediated by hepatocyte growth factor receptor, c-Met, and p38 MAPK pathways. *J. Biol. Chem.* 281: 37330-37344.
2. Buraschi, S., et al. 2010. Decorin antagonizes Met receptor activity and down-regulates  $\beta$ -catenin and Myc levels. *J. Biol. Chem.* 285: 42075-42085.
3. Bu, R., et al. 2011. HGF/c-Met pathway has a prominent role in mediating antiapoptotic signals through AKT in epithelial ovarian carcinoma. *Lab. Invest.* 91: 124-137.
4. Neill, T., et al. 2012. Decorin antagonizes the angiogenic network: concurrent inhibition of Met, hypoxia inducible factor 1 $\alpha$ , vascular endothelial growth factor A, and induction of thrombospondin-1 and TIMP3. *J. Biol. Chem.* 287: 5492-5506.
5. Tsou, H.K., et al. 2013. HGF and c-Met interaction promotes migration in human chondrosarcoma cells. *PLoS ONE* 8: e53974.
6. Neill, T., et al. 2014. Decorin induces mitophagy in breast carcinoma cells via peroxisome proliferator-activated receptor  $\gamma$  coactivator-1 $\alpha$  (PGC-1 $\alpha$ ) and mitostatin. *J. Biol. Chem.* 289: 4952-4968.
7. Yao, Y., et al. 2015. MACC1 suppresses cell apoptosis in hepatocellular carcinoma by targeting the HGF/c-Met/AKT pathway. *Cell. Physiol. Biochem.* 35: 983-996.

## RESEARCH USE

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