



PGC-1 α siRNA (m): sc-38885

BACKGROUND

Transcription factors exert their effects by associating with co-activator or corepressor proteins. The co-activator complexes are thought to be constitutively active, requiring only proper positioning in the genome to initiate transcription. Co-activators include the steroid receptor coactivator (SRC) and CREB binding protein (CBP) families that contain histone acetyltransferase (HAT) activity, which modifies chromatin structure. PPAR γ co-activator-1 (PGC-1) is a transcriptional cofactor of nuclear respiratory factor-1 (NRF-1), PPAR β , PPAR α and other nuclear receptors that is induced by exposure to cold temperatures and is involved in regulating thermogenic gene expression, protein uncoupling, and mitochondrial biogenesis. PGC-1 has a low inherent transcriptional activity when it is not bound to a transcription factor. Docking of PGC-1 to PPAR γ stimulates an apparent conformational change that then enables PGC-1 to bind to and assemble into complexes, which include the additional cofactors SRC-1 and CBP/p300, and results in a large increase in transcriptional activity.

REFERENCES

1. Onate, S.A., et al. 1995. Sequence and characterization of a co-activator for the steroid hormone receptor superfamily. *Science* 270: 1354-1357.
2. Torchia, J., et al. 1997. The transcriptional co-activator p/CIP binds CBP and mediates nuclear-receptor function. *Nature* 387: 677-684.

CHROMOSOMAL LOCATION

Genetic locus: Ppargc1a (mouse) mapping to 5 C1.

PRODUCT

PGC-1 α siRNA (m) 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 μ M solution once resuspended using protocol below. Suitable for 50-100 transfections. Also see PGC-1 α shRNA Plasmid (m): sc-38885-SH and PGC-1 α shRNA (m) Lentiviral Particles: sc-38885-V as alternate gene silencing products.

For independent verification of PGC-1 α (m) gene silencing results, we also provide the individual siRNA duplex components. Each is available as 3.3 nmol of lyophilized siRNA. These include: sc-38885A, sc-38885B and sc-38885C.

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

APPLICATIONS

PGC-1 α siRNA (m) is recommended for the inhibition of PGC-1 α expression in mouse 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 μ M in 66 μ 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

PGC-1 α (D-5): sc-518025 is recommended as a control antibody for monitoring of PGC-1 α 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 PGC-1 α gene expression knockdown using RT-PCR Primer: PGC-1 α (m)-PR: sc-38885-PR (20 μ l, 511 bp). Annealing temperature for the primers should be 55-60° C and the extension temperature should be 68-72° C.

SELECT PRODUCT CITATIONS

1. Li, L., et al. 2016. PGC-1 α promotes ureagenesis in mouse periportal hepatocytes through SIRT3 and SIRT5 in response to glucagon. *Sci. Rep.* 6: 24156.
2. Hiraike, Y., et al. 2017. NFIA co-localizes with PPAR γ and transcriptionally controls the brown fat gene program. *Nat. Cell Biol.* 19: 1081-1092.
3. Wang, F., et al. 2017. Protein kinase C- α suppresses autophagy and induces neural tube defects via miR-129-2 in diabetic pregnancy. *Nat. Commun.* 8: 15182.
4. Lee, H. and Choi, S.J. 2018. Mild hyperthermia-induced myogenic differentiation in skeletal muscle cells: implications for local hyperthermic therapy for skeletal muscle injury. *Oxid. Med. Cell. Longev.* 2018: 2393570.
5. Hu, L., et al. 2019. Targeting mitochondrial dynamics by regulating Mfn2 for therapeutic intervention in diabetic cardiomyopathy. *Theranostics* 9: 3687-3706.
6. Gao, P., et al. 2020. Inhibition of mitochondrial calcium overload by SIRT3 prevents obesity- or age-related whitening of brown adipose tissue. *Diabetes* 69: 165-180.
7. Han, X., et al. 2023. Placental mesenchymal stem cells alleviate podocyte injury in diabetic kidney disease by modulating mitophagy via the SIRT1-PGC-1 α -TFAM pathway. *Int. J. Mol. Sci.* 24: 4696.
8. Han, J.Y., et al. 2023. CRISPR-Cas9 mediated genome editing of Huntington's disease neurospheres. *Mol. Biol. Rep.* 50: 2127-2136.

RESEARCH USE

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