Diva siRNA (m): sc-37302



The Power to Question

BACKGROUND

Diva (also designated Boo, BCL2L10, Bcl-B, Bcl-2-like 10 apoptosis facilitator) is a pro-apoptotic member of the Bcl-2 protein family. Diva contains conserved BH4, BH1 and BH2 domains and can interact with other members of the Bcl-2 protein family, including Bcl-2, BCL2L1/Bcl-x $_{\rm L}$ and Bax. Bcl-2 family members form hetero- or homodimers and act as anti- or pro-apoptotic regulators that influence a variety of cellular activities. Overexpression of Diva may suppress apoptosis through the prevention of cytochrome C release from the mitochondria. The mouse homolog of Diva interacts with apoptosis activating factor-1 (Apaf-1) and forms a protein complex with caspase-9. In glioma cells, Diva interferes with apoptotic signaling downstream of cytochrome c release, but upstream of caspase activation, consistent with an inhibitory effect on the mitochondrial amplification step involving the apoptosome and Apaf-1.

REFERENCES

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- 2. Xu, Q., et al. 1998. Bax inhibitor-1, a mammalian apoptosis suppressor identified by functional screening in yeast. Mol. Cell 1: 337-346.
- Inohara, N., et al. 1998. Diva, a Bcl-2 homologue that binds directly to Apaf-1 and induces BH3-independent cell death. J. Biol. Chem. 273: 32479-32486.
- 4. Naumann, U., et al. 2001. Diva/Boo is a negative regulator of cell death in human glioma cells. FEBS Lett. 505: 23-26.
- Lee, R., et al. 2001. Characterization of NR13-related human cell death regulator, Boo/Diva, in normal and cancer tissues. Biochim. Biophys. Acta 1520: 187-194.
- Russell, H.R., et al. 2002. Murine ovarian development is not affected by inactivation of the Bcl-2 family member Diva. Mol. Cell. Biol. 22: 6866-6870.

CHROMOSOMAL LOCATION

Genetic locus: Bcl2l10 (mouse) mapping to 9 D

PRODUCT

Diva 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 μM solution once resuspended using protocol below. Suitable for 50-100 transfections. Also see Diva shRNA Plasmid (m): sc-37302-SH and Diva shRNA (m) Lentiviral Particles: sc-37302-V as alternate gene silencing products.

For independent verification of Diva (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-37302A, sc-37302B and sc-37302C.

PROTOCOLS

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

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

Diva siRNA (m) is recommended for the inhibition of Diva 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 µM in 66 µl. 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.

RT-PCR REAGENTS

Semi-quantitative RT-PCR may be performed to monitor Diva gene expression knockdown using RT-PCR Primer: Diva (m)-PR: sc-37302-PR (20 μ I). Annealing temperature for the primers should be 55-60° C and the extension temperature should be 68-72° C.

RESEARCH USE

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

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