

SignalSilence® YB1 siRNA II



✓ 10 µM in 300 µl
(100 transfections)

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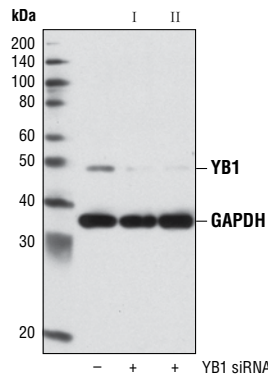
For Research Use Only. Not For Use In Diagnostic Procedures.

Species Cross-Reactivity: H, (M, R)

Description: SignalSilence® YB1 siRNA from Cell Signaling Technology (CST) allows the researcher to specifically inhibit YB1 expression using RNA interference, a method whereby gene expression can be selectively silenced through the delivery of double stranded RNA molecules into the cell. All SignalSilence® siRNA products are rigorously tested in-house and have been shown to reduce target protein expression in specified cell lines.

Background: The Y-box binding protein 1 (YB1) belongs to a family of evolutionarily conserved, multifunctional Y-box proteins that bind single-stranded DNA and RNA and function as regulators of transcription, RNA metabolism, and protein synthesis (1). YB1 binds to Y-box sequences (TAACC) found in multiple gene promoters and can positively or negatively regulate transcription. YB1 activates genes associated with proliferation and cancer, such as cyclin A, cyclin B1, matrix metalloproteinase-2 (MMP-2), and the multi-drug resistance 1 (MDR1) gene (2-4). YB1 represses genes associated with cell death, including the Fas cell death-associated receptor and the p53 tumor suppressor gene (5-7). It also interacts with the RNA-splicing factor SRp30c and stabilizes interleukin 2 mRNA upon induction of T lymphocytes by interleukin 2 (8,9). The majority of YB1 protein localizes to the cytoplasm, with a minor pool found in the nucleus; however, nuclear localization appears to be critical for its role in promoting proliferation. Nuclear translocation is cell cycle-regulated, with YB1 protein accumulating in the nucleus during G1/S phase (2). In addition, nuclear translocation is induced in response to extracellular stimuli such as hyperthermia and UV irradiation, or treatment of cells with thrombin, interferons or insulin-like growth factor (IGF-1) (2,10). Treatment of the MCF-7 breast cancer cell line with IGF-1 results in Akt-mediated phosphorylation of YB1 on Ser102, which is required for nuclear translocation of YB1 and its ability to promote anchorage-independent growth (10). YB1 is over-expressed in many malignant tissues, including breast cancer, non-small cell lung carcinoma, ovarian adenocarcinomas, human osteosarcomas, colorectal carcinomas, and malignant melanomas, and nuclear expression correlates with high levels of proliferation, drug resistance, and poor tumor prognosis (2,7,10).

Directions for Use: CST recommends transfection with 100 nM YB1 siRNA II 48 to 72 hours prior to cell lysis. For transfection procedure, follow protocol provided by the transfection reagent manufacturer. Please feel free to contact CST with any questions on use.



Western blot analysis of extracts from HeLa cells, transfected with 100 nM SignalSilence® Control siRNA (Unconjugated) #6568 (-), SignalSilence® YB1 siRNA I #6206 (+) or SignalSilence® YB1 siRNA II (+), using YB1 Antibody #2749 and GAPDH (14C10) Rabbit mAb #2118. The YB1 antibody confirms silencing of YB1 expression, while the GAPDH (14C10) rabbit mAb was used to control for loading and specificity of YB1 siRNA.

Quality Control: Oligonucleotide synthesis is monitored base by base through trityl analysis to ensure appropriate coupling efficiency. The oligo is subsequently purified by affinity-solid phase extraction. The annealed RNA duplex is further analyzed by mass spectrometry to verify the exact composition of the duplex. Each lot is compared to the previous lot by mass spectrometry to ensure maximum lot-to-lot consistency.

Entrez-Gene ID #3725
Swiss-Prot Acc. #P05412

Storage: YB1 siRNA II is supplied in RNase-free water. Aliquot and store at -20°C.

Please visit www.cellsignal.com for a complete listing of recommended companion products.

Background References:

- (1) Matsumoto, K. and Wolffe, A.P. (1998) *Trends Cell Biol.* 8, 318-23.
- (2) Jurchott, K. et al. (2003) *J. Biol. Chem.* 278, 27988-96.
- (3) Mertens, P.R. et al. (1997) *J. Biol. Chem.* 272, 22905-12.
- (4) Uchiumi, T. et al. (1993) *Cell Growth Differ.* 4, 147-57.
- (5) Lasham, A. et al. (2000) *Gene* 252, 1-13.
- (6) Lasham, A. et al. (2003) *J. Biol. Chem.* 278, 35516-23.
- (7) Homer, C. et al. (2005) *Oncogene* 24, 8314-25.
- (8) Raffetseder, U. et al. (2003) *J. Biol. Chem.* 278, 18241-8.
- (9) Chen, C.Y. et al. (2000) *Genes Dev.* 14, 1236-48.
- (10) Sutherland, B.W. et al. (2005) *Oncogene* 24, 4281-92.