Species Cross-Reactivity: H

Description: SignalSilence® CTCF siRNA I from Cell Signaling Technology (CST) allows the researcher to specifically inhibit CTCF 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 from CST are rigorously tested in-house and have been shown to reduce target protein expression by western analysis.

Background: CCCTC-binding factor (CTCF) and its paralog, the Brother of the Regulator of Imprinted Sites (BORIS), are highly conserved transcription factors that regulate transcriptional activation and repression, insulator function, and imprinting control regions (ICRs) (1-4). Although they have divergent amino and carboxy termini, both proteins contain 11 conserved zinc finger domains that work in combination to bind the same DNA elements (1). CTCF is ubiquitously expressed and contributes to transcriptional regulation of cell-growth regulated genes, including c-myc, p19/ARF, p16/INK4A, BRC1A, p53, p27, E2F1, and TERT (1). CTCF also binds to and is required for the enhancer-blocking activity of all known insulator elements and ICRs, including the H19/pg2; Prader-Willi/Angelman syndrome, and inactive X-Specific Transcript (XIST) antisense loci (5-7). CTCF DNA-binding is sensitive to DNA methylation, a mark that determines selection of the imprinted allele (maternal vs. paternal) (1). The various functions of CTCF are regulated by at least two different post-translational modifications. Poly(ADP-ribosyl)ation of CTCF is required for insulator function (8). Phosphorylation of Ser612 by protein kinase CK2 facilitates a switch of CTCF from a transcriptional repressor to an activator at the c-myc promoter (9). CTCF is ubiquitously expressed and contributes to transcriptional regulation of cell-growth regulated genes, including c-myc, p19/ARF, p16/INK4A, BRC1A, p53, p27, E2F1, and TERT (1). CTCF also binds to and is required for the enhancer-blocking activity of all known insulator elements and ICRs, including the H19/pg2; Prader-Willi/Angelman syndrome, and inactive X-Specific Transcript (XIST) antisense loci (5-7). CTCF DNA-binding is sensitive to DNA methylation, a mark that determines selection of the imprinted allele (maternal vs. paternal) (1). The various functions of CTCF are regulated by at least two different post-translational modifications. Poly(ADP-ribosyl)ation of CTCF is required for insulator function (8). Phosphorylation of Ser612 by protein kinase CK2 facilitates a switch of CTCF from a transcriptional repressor to an activator at the c-myc promoter (9). CTCF mutations or deletions have been found in many breast, prostate, and Wilms tumors (10,11).

Expression of BORIS is restricted to spermatocytes and is mutually exclusive of CTCF (3). In cells expressing BORIS, promoters of X-linked cancer-testis antigens like MAGE-1A are demethylated and activated, but methylated and inactive in CTCF-expressing somatic cells (12). Like other testis specific proteins, BORIS is abnormally expressed in different cancers, such as breast cancer, and has a greater affinity than CTCF for DNA binding sites, detracting from CTCF’s potential tumor suppressing activity (1,3,13,14).

Directions for Use: CST recommends transfection with 100 nM CTCF siRNA I 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.

Quality Control: Oligonucleotide synthesis is monitored base by base through trityl analysis to ensure appropriate composition of the duplex. Each lot is compared to the recommended companion products.

Storage: CTCF siRNA I is supplied in RNAase-free water. Aliquot and store at -20°C.

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

Background References: