



DB052: Sp1 (I18)

Background:

The Sp (specificity protein) family is a family of transcription factors that bind to cis-elements in the promoter regions of various genes. Members of the family bind with varying affinities to sequences designated as 'Sp1 sites' (e.g., GC-boxes, CACCC-boxes, and basic transcription elements) (4,8). In human cells, telomerase activity is tightly regulated by the expression of human telomerase reverse transcriptase (hTERT). Sp1 and Sp3 have been shown to associate with the hTERT promoter, recruiting histone deacetylase for the localized deacetylation of nucleosomal histones and transcriptional silencing of the hTERT gene in normal human somatic cells (3). The role of the Sp family members in many diseases is being investigated. Sp-1 has been discussed in association with diabetic microvasculopathy and Huntington's disease (2,5). It has been suggested that transcription of the Sp3 gene is blocked in immune cells from most patients with multiple sclerosis and that this contributes to the development of central nervous system inflammation in the disease (6). Sp3 has also been shown to be required for proper skeletal ossification (7). Down-regulation of the transcription factor Sp-1 is thought to be involved in the inhibition of strain-induced mitogenesis in human vascular smooth muscle cells by estrogen via an estrogen receptor mediated process (1).

Origin:

Sp1 (I18) is provided as an affinity purified rabbit polyclonal antibody, raised against a peptide mapping to an internal domain of human Sp1.

Product Details:

Each vial contains 200 µg/ml of affinity purified rabbit IgG *DB052* Sp1 (I18), in 1 ml PBS containing 0.1 % sodium azide and 0.2% gelatin.

Competition Studies:

A blocking peptide is also available, *DB052P*, for use in competition studies. Each vial contains 100 µg of peptide in 0.5 ml PBS with 0.1% sodium azide and 100 µg BSA.

Specificity:

DB052 Sp1 (I18) reacts with Sp1 of mouse, rat and human origin by western blotting. Western blotting starting dilution: 1:200.

Immunoprecipitation not yet tested. Immunohistochemistry not yet tested.

Storage:

Store this product at 4° C, do not freeze. The product is stable for one year from the date of shipment.

References:

1. Ling S; Deng G; Ives HE; Chatterjee K; Rubanyi GM; Komesaroff PA; Sudhir K. Estrogen inhibits mechanical strain-induced mitogenesis in human vascular smooth muscle cells via down-regulation of Sp-1. *Cardiovasc Res* - 01-Apr-2001; 50(1): 108-14
2. Tanaka N; Yonekura H; Yamagishi S; Fujimori H; Yamamoto Y; Yamamoto H. The receptor for advanced glycation end products is induced by the glycation products themselves and tumor necrosis factor-alpha through nuclear factor-kappa B, and by 17beta-estradiol through Sp-1 in human vascular endothelial cells. *J Biol Chem* - 18-Aug-2000; 275(33): 25781-90
3. Won J, Yim J, Kim TK. Sp1 and Sp3 recruit histone deacetylase to repress transcription of human telomerase reverse transcriptase (hTERT) promoter in normal human somatic cells. *J Biol Chem* 2002 Jul 31
4. Kishikawa S, Murata T, Kimura H, Shiota K, Yokoyama KK. Regulation of transcription of the Dnmt1 gene by Sp1 and Sp3 zinc finger proteins. *Eur J Biochem* 2002 Jun;269(12):2961-70
5. Dunah AW, Jeong H, Griffin A, Kim YM, Standaert DG, Hersch SM, Mouradian MM, Young AB, Tanese N, Krainc D. Sp1 and TAFII130 transcriptional activity disrupted in early Huntington's disease. *Science*. 2002 Jun 21;296(5576):2149-50
6. Grekova MC, Salerno K, Mikkilineni R, Richert JR. Sp3 expression in immune cells: a quantitative study. *Lab Invest*. 2002 Sep;82(9):1131-8
7. Gollner H, Dani C, Phillips B, Philipsen S, Suske G. Impaired ossification in mice lacking the transcription factor Sp3. *Mech Dev* 2001 Aug;106(1-2):77-83
8. Black AR, Black JD, Azizkhan-Clifford J. Sp1 and kruppel-like factor family of transcription factors in cell growth regulation and cancer. *J Cell Physiol* 2001 Aug;188(2):143-60

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