



DB016: p21 (C19)

Background:

The progression through the cell cycle is regulated by cyclins and their cognate Cdk by promoting cell cycle transitions (1,2,3). This orderly progression can be inhibited by a family of proteins known as CDK inhibitors (CDIs) that bind to cyclin/Cdk complexes and halt cell division (3). p21 (also designated WAF1/Cip1) is one has been shown to inhibit the activity of each member of the cyclin/Cdk family and over expression of this protein inhibits the proliferation of mammalian cells (5). The expression of p21 is inducible by a wide range of stress stimuli and its transcription can be enhanced by p53 (6). Another member of the CDIs is p27 (also known as Kip1), which also sees up regulation in response to antimitogenic stimuli (7). The increased protein expression of p27 results in cellular arrest by binding to cyclin/Cdk complexes, like cyclin D1/Cdk4 (4,8). An additional CDI has been found to bind Cdk4 and Cdk6, p16 (INK4A), and when such complexes are formed, the progression of the cell cycle is halted (9). It has become increasingly clear that p16 is a very important tumor suppressor gene whose frequent loss occurs early in many human cancers. p16 is a major target in carcinogenesis that is rivaled in frequency only by p53 (10).

Origin:

p21 (C19) is provided as an affinity purified rabbit polyclonal antibody, raised against a peptide mapping to the carboxy terminus of human p21.

Product Details:

Each vial contains 200 µg/ml of affinity purified rabbit IgG, p21 (C19) DB016, in 1 ml PBS containing 0.1 % sodium azide and 0.2% gelatin.

Competition Studies:

A blocking peptide is also available, *DB016P*, 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:

p21 (C19) DB016 reacts with p21(also designated WAF1/Cip1) of mouse, rat, and human origin by western blotting, immunoprecipitation, and immunohistochemistry. Western blotting starting dilution: 1:200.

Storage:

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

References:

1. Harper JW, Adami GR, Wei N, Keyomarsi K, Elledge SJ. 1993. The p21 Cdk-interacting protein Cip1 is a potent inhibitor of G1 cyclin-dependent kinases. *Cell* 75(4):805-816.
2. Gartel AL, Serfas MS, Tyner AL. 1996. p21 - -negative regulator of the cell cycle. *Proc Soc Exp Biol Med* 213(2):138-149.
3. Moller MB. 2000. p27 in cell cycle control and cancer. *Leuk Lymphoma* 39(1-2):19-27.
4. Sgambato A, Cittadini A, Faraglia B, Weinstein IB. 2000. Multiple functions of p27(Kip1) and its alterations in tumor cells: a review. *J Cell Physiol* 183(1):18-27.
5. Xiong Y, Hannon GJ, Zhang H, Casso D, Kobayashi R, Beach D. 1993. p21 is a universal inhibitor of cyclin kinases. *Nature* 366(6456):634.
6. Gorospe M, Wang X, Holbrook NJ. 1999. Functional role of p21 during the cellular response to stress. *Gene Expr* 7(4-6):377-385.
7. Slingerland J, Pagano M. 2000. Regulation of the cdk inhibitor p27 and its deregulation in cancer. *J Cell Physiol* 183(1):10-17.
8. Toyoshima H, Hunter T. 1994. p27, a novel inhibitor of G1 cyclin-Cdk protein kinase activity, is related to p21. *Cell* 78(1):67-74.
9. Shapiro GI, Edwards CD, Rollins BJ. 2000. The physiology of p16(INK4A)-mediated G1 proliferative arrest. *Cell Biochem Biophys* 33(2):189-197.
10. Liggett WH Jr, Sidransky D. 1998. Role of the p16 tumor suppressor gene in cancer. *J Clin Oncol* 16(3):1197-1206.

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