

DB047: p107 (C17)

Background:

The pocket protein family consists of three structurally and functionally related proteins, Rb, p107, and p130 (1). This family of tumor suppressors function to regulate important cellular transcription factors, such as the E2F family (1,2). The E2F proteins regulate the expression of genes whose products are important for cell cycle progression. The inactivation Rb is catalyzed by CDK phosphorylation thereby releasing E2F during the G1-S phase cellular progression (3). Unchecked inactivation of Rb in G1 phase has been indicated as a universal mechanism underlying cellular transformation (4,5). While Rb has been the most studied among the pocket proteins, p107 and p130 have also been shown to be key regulators of E2F (6). Several studies have also provided evidence that p107/p130 provide different functions in E2F regulation than does Rb (6,7). Rb, p107, and p130 each contain a conserved 'A/B pocket', which is the target of several viral oncoproteins, namely SV40 large T-antigen and adenovirus E1A (8).

Origin:

p107 (C17) is provided as an affinity purified rabbit polyclonal antibody, raised against a peptide mapping to the carboxy terminal domain of human p107.

Product Details:

Each vial contains 200 μ g/ml of affinity purified rabbit IgG, p107 *DB047 (C17)*, in 1 ml PBS containing 0.1 % sodium azide and 0.2% gelatin.

Competition Studies:

A blocking peptide is also available, DB047P, 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:

p107 DB047 (C17) reacts with p107 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. Halaban R. 1999. Melanoma cell autonomous growth: the Rb/E2F pathway. Cancer Metastasis Rev 18(3):333-343.
- Sidle A., Palaty C., Dirks P., Wiggan O., Kiess M., Gill R.M., Wong A.K., Hamel P.A. 1996. Activity of the retinoblastoma family proteins, pRB, p107, and p130, during cellular proliferation and differentiation. Crit Rev Biochem Mol Biol 31(3):237-271.
- 3. Classon M., Dyson N. 2001. p107 and p130: versatile proteins with interesting pockets. Exp Cell Res 264(1):135-147.
- 4. Hatakeyama M., Weinberg R.A. 1995. The role of RB in cell cycle control. Prog Cell Cycle Res 1:9-19.
- 5. Nevins J.R. 2001. The Rb/E2F pathway and cancer. Hum Mol Genet 10(7):699-703.
- 6. Hurford R.K., Cobrinik D., Lee M.H., Dyson N. 1997. pRB and p107/p130 are required for the regulated expression of different sets of E2F responsive genes. Genes Dev 11(11):1447-1463.
- Smith E.J., Leone G., Nevins J.R. 1998. Distinct mechanisms control the accumulation of the Rb-related p107 and p130 proteins during cell growth. Cell Growth Differ 9(4):297-303.
- 8. Knudsen E.S., Wang J.Y. 1998. Hyperphosphorylated p107 and p130 bind to T-antigen: identification of a critical regulatory sequence present in RB but not in p107/p130. Oncogene 16(13):1655-1663.