ProductInformation

MONOCLONAL ANTI-Cdk4, CLONE DCS-31
Mouse Ascites Fluid

Product Number C8218

Product Description

Monoclonal Anti-Cdk4 (mouse IgG2a isotype) is derived from the DCS-31 hybridoma produced by the fusion of mouse myeloma cells and splenocytes from an immunized mouse. A recombinant Cdk4 protein of human origin was used as the immunogen. The isotype is determined using Sigma ImmunoType Kit (Sigma ISO-1) and by a double diffusion immunoassay using Mouse Monoclonal Antibody Isotyping Reagents (Sigma ISO-2). The product is provided as ascites fluid with 15 mM sodium azide (see MSDS)* as a preservative.

Monoclonal Anti-Cdk4 may be used for the localization of Cdk4, using various immunochemical assays such as immunoblotting, immunocytochemistry and immunoprecipitation.

During the cell cycle of most somatic cells, DNA synthesis (S-phase) and mitosis (M-phase) are separated by two gap phases (G1 and G2) of varying duration. Thus, a typical eukaryotic cell sequentially passes through G1, S, G2, and M and back into G1 during a single cycle.[1] Regulation of cell cycle progression in eukaryotic cells depends on the expression of proteins called cyclins.[2] These proteins form complexes with several different cyclin-dependent kinases (CDKs). Within the complexes, the cyclin subunit serves a regulatory role, whereas the CDKs have a catalytic protein kinase activity.[3] Complexes of cyclins and CDKs play a key role in cell cycle control. The eukaryotic cell cycle is regulated by the sequential activation of CDKs. The association of members of the cyclin family with the kinase subunit forms an active kinase, which can initiate M phase of mitosis and meiosis, or function as key regulators of each step of the cell cycle by phosphorylation of several cellular targets. Two general mechanisms, protein phosphorylation and association with regulatory subunits, including the cyclins and the CDK inhibitors (CKIs), regulate the catalytic activity of CDKs. Several mammalian CDK inhibitors have been identified including $p16^{INK4a}$, $p15^{INK4b}$, $p18^{INK4c}$, $p19^{INK4d}$, $p21^{Cip1}$, $p27^{Kip1}$ and $p57^{Kip2}$. Complexes formed by Cdk4 and the D-type cyclins have been strongly implicated in the control of cell proliferation during the G1 phase.[4-6] Cdk4 (previously known as PSK-J3, $p34^{PSK-J3}$)[7] exists, in part, as a multi-protein complex with a D-type cyclin, proliferating cell nuclear antigen (PCNA) and a protein inhibitor, $p21^{Cip1}$. Cdk4 associates separately with p16, particularly in cells lacking a functional retinoblastoma protein.[8-11] The availability of monoclonal antibody reacting specifically with Cdk4 enables the subcellular detection and localization of Cdk4 and the measurement of relative differences in Cdk4 levels as a function of cell cycle phase.

Reagents

Monoclonal Anti-Cdk4 reacts specifically with Cdk4. It does not recognize other Cdk types. The product may be used for immunoblotting (33 kD, and possibly additional weak bands of lower and higher m.w.), immunocytochemistry and immunoprecipitation. Reactivity has been observed with human, rat and mouse Cdk4.

Precautions and Disclaimer

* Due to the sodium azide content a material safety sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazardous and safe handling practices.

Storage/Stability

For continuous use, store at 2-8°C for up to one month. For extended storage freeze in working aliquots. Repeated freezing and thawing is not recommended. Storage in "frost- free" freezers is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use.

Product Profile

A minimum working dilution of 1:1,000 is determined by immunoblotting using a cultured human tumor cell line extract.
Note: In order to obtain best results in different techniques and preparations we recommend determining optimal working dilutions by titration test.

References

1. Freeman, R.S., and Donoghue, D.J., Biochemistry, **30**, 2293 (1991).
2. Pines, J., and Hunter, T., J. Cell Biol., **115**, 1 (1991).
3. Yamashita, M., et al., Dev. Growth Differ., **33**, 617 (1991).
4. Motokura, T., and Arnold, A., Biochim. Biophys. Acta, **1155**, 63 (1991).
5. Sherr, C.J., Cell, **73**, 1059 (1993).
6. Elledge, S.J., Science, **274**, 1664 (1996).
7. Matsushime, H., et al., Cell, **71**, 323 (1992).
8. Xiong, Y., et al., Genes Dev., **7**, 1572 (1993).
9. Lukas, J., et al., Nature, **375**, 503 (1995).
10. Aagaard, L., et al., Int. J. Cancer, **61**, 115 (1995).
11. Lukas, J., et al., Cancer Res., **55**, 4818 (1995).

lpg 4/98